soho2023
ELEVENTH ANNUAL MEETING

INSIGHT INTO HYPEREOSINOPHILIA FROM THE HEMATOLOGY CLINIC; A SINGLE-CENTER EXPERIENCE

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INTRODUCTION

The objective of this study is to present the experience with hypereosinophilia cases where no underlying secondary cause was identified. The focus is on the management and treatment decisions for patients presenting with elevated absolute eosinophil count (AEC) and the potential complications associated with eosinophil-mediated organ damage.

AIM

Here, we summarize our experience in hypereosinophilia referred after no underlying secondary cause has been found.

METHOD

This retrospective study involved the evaluation of cases of hypereosinophilias recorded in the hematology clinical electronic system. Patient data were reviewed to identify cases where no underlying secondary cause was found. The organs affected at presentation and the final diagnosis were documented. All patients were screened for myeloid neoplasms associated with imatinib-sensitive mutations, and empiric antiparasitic therapy was administered. Genomic analyses were not performed due to financial limitations. Steroids were the most commonly used treatment in primary care, followed by hydroxyurea.

METHODOLOGY-RESULTS

The study included 31 patients with a mean age of 48 years and a slight male-to-female predominance. The mean eosinophil level at diagnosis was 8600/µL. Antiparasitic treatment led to improvement in 13% of patients, while 16% had underlying autoimmune pathology. No cases of myeloproliferative or lymphocytic variant hypereosinophilic syndrome (HES) were identified. Lymphoproliferative diseases accounted for 13% of cases, and idiopathic HES was diagnosed in 45% of patients. Cardiac involvement was the most common symptom, observed in 39% of patients, with manifestations such as endomyocardial fibrosis and myocardial thickening. Pulmonary symptoms resembling asthma were reported in 19% of patients, and thromboembolic events occurred in 6 patients.

Results: Eosinophil counts were not found to be significantly associated with cardiac involvement or thrombosis in this patient group.

	N	Min	Max	Mean
Age	31	19	89	49.1
Wbc x108/µL	31	6.1	70	18.6
Eozsinophil x108/µL	31	1.9	46.1	8.5
Hb g/dl	30	8,5	18,0	13,1
LDH U/L	13	172	993	338,1
IgM mg/dL	20	44	233,0	100,7
Total IgE IU/mL	18	6	7333	1319,4
Tryptase ug/L	29	2,5	16,0	7,5
Vitamin B12 pg/mL	31	145	1869	563,9

CONCLUSIONS

Hypereosinophilia can have serious consequences regardless of the underlying cause. A multidisciplinary approach involving various specialists is crucial for accurate diagnosis, treatment monitoring, and identification of myeloproliferative or lymphocytic variants of HES or idiopathic HES.

i-HES	17
Parasitosis	4
Lymphoproliferative	4
M-HES	1
Vasculitis	3
Autoimmune	2
Total	31

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