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REVIEW ARTICLE

WHEN EOSINOPHILS OVERWHELM: A CASE OF FULMINANT ARDS, HIGH IGE, AND RAPID DEMISE

[1]Dr. Shweta Awasthi and [2]Dr. Indranil Das

[1]Postgraduate Trainee(DNB Emergency Medicine), Medica Supers peciality Hospital Kolkata (MSHK); [2]MBBS, FEM(RCGP-UK), MEM(GWU-USA), MRCEM-UK, Sr. Consultant and HOD, Dept. Emergency Medicine, MSHK

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Corresponding author: Dr. Shweta Awasthi

ABSTRACT

A 68-year-old male with no significant past medical history presented with acute severe respiratory failure. Investigations on admission revealed ARDS, profound peripheral eosinophilia (Absolute Eosinophil Count >30,000/µL) with a markedly elevated serum IgE (4595 IU/mL), hyperleukocytosis, severe thrombocytopenia, coagulopathy, and multiorgan dysfunction. Multiple ecchymotic patches were noted. He rapidly developed refractory shock, succumbing within 12 hours of ICU admission despite maximal supportive care. This report documents a catastrophic presentation driven by extreme eosinophilia, highlighting the devastating clinical course and the importance of considering eosinophilic emergencies, potentially with IgE-mediated or T-cell driven mechanisms, in unexplained critical illness.

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INTRODUCTION

Acute Respiratory Distress Syndrome (ARDS) is a life-threatening form of hypoxemic respiratory failure (1), affecting approximately 10% of intensive care unit (ICU) patients and with mortality rates ranging from 30% to over 50% (1). While ARDS can result from various insults, its presentation with extreme peripheral eosinophilia signals a distinct and challenging clinical scenario. Eosinophils, when excessively numerous or activated, can inflict significant tissue damage (3). Hypereosinophilia (AEC >1,500/µL) is a hallmark of disorders such as Hypereosinophilic Syndromes (HES)—with an estimated US incidence of 0.3–6.3 per 100,000 individuals (2)—Acute Eosinophilic Pneumonia (AEP)—a rarer condition with approximately 400 cases reported globally (7)—and Eosinophilic Granulomatosis with Polyangiitis (EGPA). These conditions can precipitate life-threatening multiorgan failure. A markedly elevated serum IgE level can further refine the diagnostic considerations within this spectrum. This report details an exceptionally fulminant case of ARDS in a patient with previously unrecognized extreme eosinophilia and significantly elevated IgE, underscoring its catastrophic potential.

CASE PRESENTATION

History: A 68-year-old male, with no known chronic diseases, presented to the Emergency Department (ED) with acute severe dyspnea. He communicated that approximately one day prior, he had attended an outpatient clinic for right shoulder pain, during which routine blood tests were performed. He also self-reported a history of progressive orthopnea and generalized weakness for about four weeks, abdominal pain for one week, melena for five days, and obstipation for two days prior to this acute presentation. No history of asthma, new medications, recent travel, or known significant allergies was elicited in the acute setting.

Initial Examination and Management: Upon ED arrival, he was in severe respiratory distress (respiratory rate 41/min), tachycardic (109/min), with SpO2 at 86% on room air. Blood pressure was 150/80 mmHg. Bilateral widespread lung crepitations were audible. Notably, multiple ecchymotic patches were present over his trunk and lower limbs. Initial arterial blood gas (ABG) analysis revealed severe hypoxemia (PaO2/FiO2 ratio ~38) and lactic acidosis (pH 7.35, lactate 5.3 mmol/L). Emergent intubation and mechanical ventilation were initiated.

Investigations

Performed Approximately One Day Prior to Acute Admission (Outpatient Basis):

- Hematology revealed: Hemoglobin 11.7 g/dL, Total Leukocyte Count (TLC) 73,760/mm³ (corrected), Platelets 16,000/mm³, Eosinophils 46.1% (AEC approximately 34,000/µL).

- Biochemistry showed: Urea 132 mg/dL, Creatinine 1.33 mg/dL, AST 174 U/L, ALT 187 U/L, Total Bilirubin 3.5 mg/dL (conjugated 2.2 mg/dL), Albumin 2.4 g/dL. C-Reactive Protein (CRP) was 72.68 mg/L.

Performed on Emergency Department Arrival and During Acute Admission:

- Repeat Hematology: Hemoglobin 10.6 g/dL, TLC 80,610/mm³ (corrected for 5 NRBCs/100 WBCs), with Eosinophils 38.4% (AEC 30,950/µL) and Metamyelocytes 2.0%. Platelet count was 43,000/mm³. Peripheral blood smear confirmed eosinophilia, thrombocytopenia, and a leukoerythroblastic picture.
- Coagulation studies: D-Dimer >10,000 ng/mL; Fibrinogen 543.5 mg/dL (INR from day prior: 1.57).
- Inflammatory, Cardiac, and Specific Markers: Procalcitonin 2.110 ng/mL; LDH 729 U/L; Ferritin 1116.6 ng/mL; hs-Troponin-I 151.60 ng/L; NT-proBNP 15275 pg/mL. Serum IgE level was 4595 IU/mL (Typical Reference: 0.0-100.0 IU/mL).
- Infectious Workup: Malaria Antigen tests were negative. Blood and urine cultures showed no growth. Serologies for Scrub Typhus, Leptospira, Dengue, Chikungunya, HIV, HBsAg, and HCV Ab were negative. A broader screen for other parasites was not documented.

Imaging: Imaging on admission included a chest X-ray (bilateral interstitial opacities) and HRCT chest, which revealed severe ARDS. Bedside echocardiogram showed concentric left ventricular hypertrophy, adequate EF (55%), and Grade II diastolic dysfunction.

Clinical Course and Management in ICU: In the ICU, the patient received lung-protective ventilation; settings were later adjusted (RR 25/min, I:E 1:3, TV 450 mL) in an attempt to manage severe acidosis (pH 6.9, pCO2 79 mmHg). Empiric broad-spectrum antibiotics and intravenous corticosteroids were administered promptly. Despite this, he developed refractory shock requiring high-dose norepinephrine and vasopressin. Platelet and FFP transfusions were given for severe thrombocytopenia and coagulopathy. His extremely rapid deterioration precluded planned advanced diagnostics such as bone marrow biopsy, flow cytometry, and ANCA serology. Approximately 12 hours after ICU admission, he suffered a cardiac arrest and could not be resuscitated. The documented cause of death included septic shock, multiorgan dysfunction, ARDS, severe thrombocytopenia, and UGI bleed.

DISCUSSION

This case illustrates a catastrophic trajectory driven by extreme eosinophilia (AEC >30,000/µL) and markedly elevated IgE (4595 IU/mL), leading to fulminant ARDS and multiorgan failure within hours of acute presentation. The central challenge is identifying the underlying driver from the constellation of findings—massive eosinophilia, hyper-IgE, severe thrombocytopenia, coagulopathy, a leukoerythroblastic picture, and widespread organ damage—in the absence of definitive diagnostic tests precluded by the patient's rapid demise. The combination of extreme eosinophilia and very high IgE strongly points towards a Th2-polarized immune process, narrowing the differential primarily to Hypereosinophilic Syndrome (HES), Eosinophilic Granulomatosis with Polyangiitis (EGPA), and certain Hematologic Malignancies. HES, particularly the Lymphocytic Variant (L-HES), aligns well with the potential for aberrant T-cell cytokine production (e.g., IL-5, IL-4, IL-13) driving both the profound eosinophilia and the hyper-IgE state, leading to multiorgan damage (4). Cardiac involvement (suggested by biomarkers, possibly eosinophilic myocarditis) and involvement of liver, kidneys, and the hematologic system are well-described in HES. EGPA is also a strong possibility given the eosinophilia, high IgE, ARDS, and potential vasculitic components (possibly contributing to ecchymoses beyond coagulopathy) (5, 8). While the lack of documented asthma is atypical, it's not exclusionary, but confirmation would require ANCA results or biopsy evidence of eosinophilic vasculitis. An underlying Hematologic Malignancy remains a critical consideration, especially given the leukoerythroblastic picture and severe thrombocytopenia alongside the hyperleukocytosis (2). Certain T-cell lymphomas, capable of driving IgE and eosinophilia, or eosinophilic leukemias could present aggressively. Differentiating these neoplastic processes from reactive HES subtypes requires bone marrow analysis, flow cytometry, and cytogenetics, which were unobtainable.

Other diagnoses seem less likely to explain the entire picture. Classic Acute Eosinophilic Pneumonia (AEP) typically lacks such extreme systemic involvement and marked IgE elevation (7). A Severe Allergic Reaction is inconsistent with the prodrome and complex hematologic derangements. While invasive Parasitic Infections can cause high eosinophilia/IgE, specific screening was limited. The final picture of "septic shock" likely represented the end stage of uncontrolled systemic inflammation triggered by the primary eosinophilic disorder, rather than a primary bacterial infection. Pathophysiologically, the fulminant course suggests massive, uncontrolled degranulation of eosinophils, releasing cytotoxic proteins that cause direct endothelial damage (leading to ARDS via capillary leak), myocardial injury, and contributing to the broader inflammatory state and organ dysfunction (3). The extreme rapidity likely overwhelmed compensatory mechanisms. Management in such scenarios is fraught with difficulty. Although cornerstones like early high-dose corticosteroids and aggressive organ support were implemented, the trajectory was unalterable. This case underscores the critical need for immediate recognition and multidisciplinary input (including hematology/immunology) in suspected eosinophilic catastrophes, acknowledging that even theoretical interventions like emergency leukapheresis face immense logistical and clinical challenges in the context of such precipitous decline.

Limitations: The foremost limitation is the inability to establish a definitive etiological diagnosis due to the patient's extremely rapid death. This precluded comprehensive investigations crucial for differentiating between severe HES, EGPA, an aggressive hematologic malignancy with eosinophilia, or other potential causes. Specifically, bone marrow aspiration and biopsy, flow cytometry, ANCA serologies, broader parasitic screening, and specific genetic testing were not performed. An autopsy, which could have provided invaluable histopathological information, was also not conducted.

CONCLUSION AND LEARNING POINTS

This report documents a devastating case of ARDS and multiorgan failure, driven by extreme eosinophilia and markedly elevated IgE, resulting in death within hours of acute presentation. It underscores the catastrophic potential of disorders leading to massive eosinophil proliferation and activation.

Key learning points include:

- The combination of extreme eosinophilia and high IgE in a critically ill patient with ARDS and multiorgan dysfunction should prompt urgent consideration of aggressive eosinophilic syndromes (like L-HES), vasculitides (like EGPA), and underlying hematologic malignancies.
- The fulminant nature of such conditions can severely limit diagnostic and therapeutic interventions, often leaving clinicians with only supportive measures and empiric corticosteroids.
- Prognosis in such hyperacute presentations is exceptionally grave, emphasizing the need for immediate recognition and escalation of care.
- Heightened awareness and education regarding the rapid recognition pathways and multidisciplinary emergency management (including early hematology input) for severe eosinophilic disorders are crucial, although even optimal care may be insufficient in the most aggressive cases.

Conflict of Interest: The authors declare no conflict of interest.

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