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EOSINOPHILIA: A COMPREHENSIVE REVIEW FOR PHARMACY PRACTICE

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ABSTRACT

Eosinophilia is defined as an elevated eosinophil count in the peripheral blood and is often associated with a wide range of clinical conditions. This review discusses the aetiology, classification, and pathophysiological mechanisms of eosinophilia, with special attention to diseases such as eosinophilic granulomatosis with polyangiitis (EGPA), eosinophilic esophagitis (EoE), and paediatric eosinophilia. Diagnostic approaches, treatment strategies, and implications for pharmacy practice are also explored. A comprehensive understanding of eosinophilia is vital for accurate diagnosis, targeted therapy, and effective patient counselling.

Keywords: *Eosinophilia, EGPA, Eosinophilic Esophagitis, Paediatrics, Pharmacy Practice*.

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INTRODUCTION

Eosinophils are a type of granulocyte that leaves the bone marrow to circulate in the blood and migrate into tissues. They normally account for about 1–5% of circulating white blood cells, with counts usually under 0.5 × 10^9/L [1]. Their growth and tissue distribution are influenced by cytokines such as IL-3, IL-5, GM-CSF, and a range of chemokines [2]. An eosinophil count above 0.5 × 10^9/L is defined as peripheral eosinophilia, while higher and persistent levels≥1.5 × 10^9/L recorded at least twice a month apart or associated with tissue infiltrationindicate hypereosinophilia (HE). This raises concern for hypereosinophilic syndrome (HES) or related disorders [3,4]. Eosinophilia can arise from many different processes, ranging from common allergic or parasitic reactions to primary hematologic neoplasms driven by PDGFRA/B or FGFR1 rearrangements. Some cases involve organ-limited disorders, whereas others manifest as systemic syndromes such as eosinophilic granulomatosis with polyangiitis (EGPA). Identifying the underlying cause is essential, since unchecked eosinophil activity can lead to complications like cardiac fibrosis, neuropathy, and chronic lung disease [5,6]. The last decade has seen major advances in both diagnostics and treatment. Molecular testing helps identify genetic drivers, while newer targeted therapiessuch as IL-5/IL-5R blockers (mepolizumab, benralizumab, reslizumab), tyrosine kinase inhibitors (imatinib, Pemigatinib), and biologics like dupilumab-have transformed management options [7–10].

METHODS

A structured literature search of articles on eosinophilia was conducted from January 2023 to June 2025. Keywords included "eosinophilia review," "hypereosinophilic syndrome treatment," "EGPA mepolizumab benralizumab," "eosinophilic esophagitis biologics," and "pediatric eosinophilia epidemiology." Eligible publications were human studies in English, including RCTs, systematic reviews, clinical guidelines, and robust observational data. Where available, full-text PMC sources and updates from the WHO and I-COG classification systems were reviewed.

Information was extracted across several themes:

- Terminology and classification (etiologic and molecular frameworks).
- Epidemiology and subtypes.
- Pathways underlying reactive, clonal, and organ-specific disease.
- Diagnostic approaches (laboratory, histology, imaging, and molecular tests).

- Treatment strategies including corticosteroids, biologics, kinase inhibitors, and supportive care.
- Special disease categories: EGPA, eosinophilic esophagitis (EoE), and pediatric eosinophilia.
- Pharmacy-related issues: medicationcauses, counselling, monitoring, and public awareness.

CLASSIFICATION AND EPIDEMIOLOGY

Eosinophilia is broadly grouped into:

- Reactive (secondary): due to allergy, parasitic infection, medications, autoimmune conditions, or malignancy [11,12].
- Primary (clonal): hematologic neoplasms with PDGFRA/B or FGFR1 rearrangements, chronic eosinophilic leukaemia, or idiopathic HES [13].
- Organ-restricted/idiopathic forms: where eosinophilia is localised, or no clear cause is identified [14].

While mild eosinophilia is common (1–2% of the population), hypereosinophilia is rare, affecting about 0.3–6 per 100,000 people per year in Western countries [16].

PATHOPHYSIOLOGY

- Cytokines (IL-5, IL-3, GM-CSF) and epithelial signals such as IL-25, IL-33, and TSLP that promote survival and activation are the main drivers of reactive eosinophilia [17].
- Clonal illness arises from mutations or gene fusions (e.g., FIP1L1-PDGFRA, FGFR1, JAK2) that lead to unregulated proliferation and organ invasion.
- Tissue injury occurs when eosinophils release toxic proteins (MBP, ECP) and reactive oxygen species, leading to inflammation and fibrosis. Importantly, severity is not always proportional to the blood eosinophil count [18].

DIAGNOSTIC APPROACH

Evaluation begins with a CBC and differential, along with a careful history (allergies, travel, medications) and physical exam. If eosinophilia persists ≥1.5 × 10^9/L or organ damage is suspected, next steps include:

- Bone marrow studies with morphology.
- Cytogenetic/FISH testing for PDGFRA/B, FGFR1.
- Molecular assays such as RT-PCR.
- Flow cytometry and imaging (e.g., echocardiography, CT) [19].
- The WHO/I-COG 2022 update emphasises molecular categorisation to guide therapy

TREATMENT APPROACHES

- Observation: mild, asymptomatic eosinophilia may simply be monitored.
- Corticosteroids: remain first-line therapy for most HE/HES cases; Strongyloides testing is essential before use in endemic regions [20].
- TKIs: Imatinib is highly effective in PDGFRA/B-positive cases; pemigatinib may be used for FGFR1 fusions.

Biologics:

- Mepolizumab is FDA-approved for HES, reducing flares [21].
- Benralizumab depletes eosinophils through antibody-dependent cytotoxicity and benefits both HES and eosinophilic asthma [22].
- Dupilumab blocks IL-4/13 pathways and is effective in eosinophilic asthma and EoE [23].
- Reslizumab has shown steroid-sparing effects in small trials [24].
- Real-world studies confirm reduced steroid dependence with these agents [25].

EGPA

EGPA is an ANCA-associated small-vessel vasculitis characterised by asthma, sinus disease, pulmonary infiltrates, eosinophilia, neuropathy, and other organ involvement. Around one-third of patients are ANCA-positive, influencing presentation [26]. Both mepolizumab and benralizumab have shown success in inducing remission and lowering steroid exposure [27]. Adjunctive therapies such as IVIG, plasma exchange, and avacopan are being explored [28].

Eosinophilic Esophagitis (EoE)

EoE is a chronic, allergen-driven disease presenting with dysphagia, food impaction, vomiting, or reflux-like symptoms [29]. Without treatment, it can progress to fibrostenotic disease. The inflammatory pathway involves epithelial barrier dysfunction and type 2 cytokines (IL-25, IL-33, TSLP, IL-5, IL-13) [30].

Current management includes dietary elimination, topical steroids, PPIs, and oesophageal dilation. Dupilumab gained FDA approval in 2022 and has shown strong efficacy; other biologics targeting IL-5/13 are under study [31,32]. Meta-analyses suggest IL-5 agents have a modest benefit, while IL-4/13 blockade achieves stronger histologic responses [32]. AI-based histology assessment tools are an emerging adjunct [33].

Pediatric Eosinophilia

A review of 771 children found that mild eosinophilia (500–1,500/μL) was usually linked to allergy/atopy, whereas severe eosinophilia (>4,500/μL) often signalled immunodeficiency or genetic syndromes [34]. Causes vary geographically: in wealthier regions, ~78% are allergic, while in lower-income settings, parasitic infections dominate (17–88%) [35].

DISCUSSION

Clinical and pharmacy considerations

- Medications (antibiotics, anticonvulsants, NSAIDs) can trigger eosinophilia, including DRESS; early recognition is vital [36].
- Molecular testing directs use of TKIs [13].
- With biologics, pharmacists play a role in counselling on safety, monitoring response, and cost issues [25].

- In EGPA, avacopan may be helpful in ANCA-positive cases [28].

Public health perspective

- While mild eosinophilia is often benign, persistence ≥1.5 × 10^9/L should prompt evaluation [11].
- Some dietary supplements have been linked to eosinophilia-myalgia syndrome and should be avoided [37].
- Pediatric evaluation must consider local epidemiology-parasites in some regions, atopy in others [35].

Future challenges:

- The optimal duration of biologic therapy remains unknown [38].
- Biomarkers and AI tools show promise in diagnostics [33].
- Cost and access remain barriers, particularly in low-resource settings [13].
- Non-oesophagal eosinophilic GI diseases remain under-studied [39].

CONCLUSION

Eosinophilia encompasses a wide spectrum of disorders—reactive, clonal, organ-restricted, and systemic. While corticosteroids are still foundational, biologic agents and targeted therapies are redefining management by reducing steroid dependence and improving quality of life. For pharmacy students and clinicians alike, understanding molecular drivers, treatment strategies, and patient counselling is essential. Continued research in diagnostics, therapy optimization, and equitable access will shape the next phase of eosinophilia care.

ABBREVIATIONS

AI – Artificial Intelligence
ANCA – Anti-Neutrophil Cytoplasmic Antibodies
CBC – Complete Blood Count
CT – Computed Tomography
DRESS – Drug Reaction with Eosinophilia and Systemic Symptoms
ECP – Eosinophil Cationic Protein
EGPA – Eosinophilic Granulomatosis with Polyangiitis
EoE – Eosinophilic Esophagitis
FGFR1 – Fibroblast Growth Factor Receptor 1
FIP1L1-PDGFRA – FIP1-Like 1–Platelet-Derived Growth Factor Receptor Alpha (fusion gene)
FISH – Fluorescence In Situ Hybridization
GM-CSF – Granulocyte-Macrophage Colony-Stimulating Factor
HE – Hypereosinophilia
HES – Hypereosinophilic Syndrome
I-COG – International Consensus on (Myeloid) Neoplasms and Related Disorders Classification
IL – Interleukin
IVIG – Intravenous Immunoglobulin
JAK2 – Janus Kinase 2
MBP – Major Basic Protein
NSAIDs – Nonsteroidal Anti-Inflammatory Drugs
PCR/RT-PCR – Polymerase Chain Reaction / Reverse Transcriptase–Polymerase Chain Reaction
PDGFRA/B – Platelet-Derived Growth Factor Receptor Alpha/Beta
PPI – Proton Pump Inhibitor
RCT – Randomised Controlled Trial
TKI – Tyrosine Kinase Inhibitor
TSLP – Thymic Stromal Lymphopoietin
WHO – World Health Organisation

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