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CLINICAL IMMUNOLOGICAL ALTERATIONS IN ENTEROBIASIS: A COMPREHENSIVE REVIEW

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Enterobiasis, caused by the nematode *Enterobius vermicularis*, is a common parasitic infection worldwide, particularly among children. Though often considered mild, the infection triggers distinct changes in both innate and adaptive immunity. This review explores the clinical immunological responses associated with enterobiasis, including cellular activation, antibody production, cytokine shifts, and systemic immune regulation. Recognizing these alterations is crucial for improving diagnostic accuracy, guiding therapeutic approaches, and informing potential immunomodulatory strategies.

Introduction.

Enterobiasis, commonly referred to as pinworm infection, is one of the most frequent helminth infections globally, affecting millions of children and adults. The parasite, *Enterobius vermicularis*, inhabits the human large intestine, laying eggs primarily around the perianal region. Transmission occurs predominantly through the fecal-oral route, often facilitated by contaminated hands, clothing, bedding, or household surfaces. Reinfection is frequent in communal settings such as schools, making the infection persistent in endemic areas (Hotez et al., 2020).While many cases remain asymptomatic, some individuals experience perianal itching, sleep disruption, irritability, and gastrointestinal discomfort. These symptoms are not purely mechanical; host immune responses are increasingly recognized as central to symptom development and parasite clearance (Nutman, 2018).

The immune system mounts a multi-layered response to *E. vermicularis*. Innate immunity provides the initial defense via eosinophils, neutrophils, macrophages, mucosal barriers, and dendritic cells. These mechanisms help limit parasite colonization and initiate inflammatory signaling. Adaptive immunity, predominantly Th2-oriented, promotes IgE synthesis, eosinophil recruitment, and memory responses that facilitate worm expulsion. Chronic infections can additionally modulate regulatory pathways, including Treg activation, which may influence the host's response to other infections or allergens (Maizels & McSorley, 2016).This review summarizes current knowledge on clinical immunological changes in enterobiasis, detailing both systemic and mucosal responses, innate and adaptive mechanisms, cytokine modulation, and their implications for clinical management.

Innate immunity acts as the first line of defense against *E. vermicularis*:

- **Eosinophils:** These cells are central to anti-helminth defense, releasing cytotoxic proteins and reactive oxygen species to damage parasites. Elevated eosinophil counts are frequently observed in infected patients.
- **Neutrophils and Macrophages:** Neutrophils contribute through phagocytosis and inflammatory mediator release. Macrophages process parasitic antigens and secrete cytokines such as IL-1β and TNF-α, facilitating immune cell recruitment.

- **Mucosal barriers:** The intestinal epithelium and mucus layers impede parasite adherence. Goblet cells secrete mucins that trap eggs, enhancing expulsion.
- **Dendritic cells:** Capture parasite antigens and prime T cells, bridging innate and adaptive immunity.

Table 1. Key innate immune components in enterobiasis

Component	Role	Clinical Relevance
Eosinophils	Cytotoxic degranulation	Symptom correlation
Neutrophils	Phagocytosis, inflammatory signaling	Tissue inflammation
Macrophages	Antigen presentation	T-cell activation
Goblet cells	Mucus secretion	Physical barrier
Dendritic cells	Antigen presentation	Initiates adaptive immunity

Adaptive immunity provides targeted, long-lasting protection against enterobiasis:

- **Humoral Immunity:** IgE production is a hallmark, while IgG and IgA antibodies target parasite antigens, aiding parasite clearance.
- **Cell-mediated Immunity:** Th2-skewed responses dominate, with IL-4, IL-5, and IL-13 facilitating eosinophil recruitment, IgE synthesis, and mucus secretion. Th1 responses, such as IFN-γ production, are typically reduced in chronic cases.
- **Cytokine Dynamics:** Elevated IL-10 and TGF-β promote regulatory T-cell activity, limiting excessive inflammation but potentially enabling parasite persistence.

Patients with enterobiasis often exhibit measurable immune changes:

1. **Peripheral eosinophilia:** Correlates with symptom severity, particularly pruritus.
2. **Elevated IgE:** Reflects Th2-mediated immune activity.
3. **Parasite-specific IgG/IgA:** Supports humoral recognition and immune memory.
4. **Cytokine shifts:** Increased IL-4, IL-5, IL-10, IL-13; Th1 cytokines remain low.
5. **Regulatory T-cell activity:** Modulates inflammatory responses, promoting transient immune tolerance.

Table 2. Immunological markers in enterobiasis

Marker	Change	Significance
Eosinophils	Increased	Infection indicator
IgE	Elevated	Mediates pruritus
IgG/IgA	Detectable	Parasite-specific immunity
IL-4, IL-5, IL-13	Upregulated	Th2 polarization
Tregs	Enhanced activity	Limits inflammation

5. Implications for Diagnosis and Management

Recognizing immunological changes has practical significance:

- **Diagnosis:** Laboratory findings such as eosinophilia, elevated IgE, and detection of parasite-specific antibodies complement stool tests and perianal tape tests.
- **Treatment:** Anthelmintics (e.g., mebendazole, albendazole) eradicate the parasite and often normalize immune parameters.

- **Immunomodulatory approaches:** Understanding host immune modulation may inform vaccine development or adjunctive therapies for recurrent infections.

Conclusion

Enterobiasis induces notable clinical immunological alterations, including Th2-skewed responses, peripheral eosinophilia, elevated IgE, and enhanced regulatory T-cell activity. These changes contribute to symptom development, influence parasite clearance, and may modulate responses to other infections or allergens. Recognizing and understanding these immune dynamics is essential for accurate diagnosis, effective treatment, and future immunotherapeutic interventions. Further research is needed to clarify long-term immune impacts, especially in pediatric populations.

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