Head lice presenting with isolated severe eosinophilia in a child

Panagopoulou P1, Ioannidou G1, Beropouli S2, Fotoulaki M1

14th Department of Pediatrics
21st Department of Pediatrics
School of Medicine, Aristotle University of Thessaloniki, Greece

Abstract

Background: Eosinophilia is frequent in pediatrics and concerns regarding its etiology are often raised. Pediculosis capitis is a common ectoparasitic disease in children but is not traditionally associated with changes in laboratory parameters.

Case description: We present the case of a healthy, 9-year-old girl who presented with abdominal pain and a history of loose stools (2-3) in the preceding week. Laboratory investigations showed leukocytosis with significant eosinophilia. A thorough investigation concerning the most common causes of eosinophilia was negative. Detailed physical examination and history revealed a persistent scalp infestation with head lice. Successful pediculosis management coincided with the gradual resolution of eosinophilia without recurrence until the most recent follow-up visit twelve months later.

Conclusions: The presented case indicates a possible association between eosinophilia and pediculosis capitis, which was not previously displayed, but also highlights the importance of detailed history and clinical examination. Research focusing on the systematic effects of pediculosis capitis in children could clarify whether it can represent a frank cause of eosinophilia. HIPPOKRATIA 2023, 27 (2):112-114.

Keywords: Eosinophilia, pediculosis capitis, head lice, children

Corresponding author: Panagopoulou Paraskevi, MD, MPH, PhD, Associate Professor of Pediatrics-Pediatric Hematology & Oncology, 4th Department of Paediatrics, AUTH, Papageorgiou General Hospital, Ring Road, Nea Efkarpa, Thessaloniki, 56403, Greece, tel.: +302313323313, fax: +302313323918, e-mail: vivianpa@icloud.com, ppanagopoulou@auth.gr

Introduction

Eosinophilia is defined as an absolute eosinophil count (AEC) >500 /µL, is frequent in children, and can be mild (500-1500 /µL), moderate (1500-5000 /µL), or severe (>5000 /µL). Hypereosinophilia (HE) is the persistence of AEC >1500 /µL on two measurements at least four weeks apart or marked tissue eosinophilia1,2. “Hypereosinophilic syndrome” is the combination of HE and end-organ involvement [heart, lungs, nervous system, gastrointestinal (GI) tract, skin] that is caused by the deposition of eosinophil granule products in these tissues. The main causes of eosinophilia include atopic diseases in industrialized and parasitic infections in developing countries. Pediculosis capitis is the most common ectoparasitic disease in children 3-11 years old in both developing and affluent countries, affecting all socioeconomic groups3. Other ectoparasites like scabies are a recognized cause of eosinophilia, and a possible association between head lice and eosinophilia has rarely been described in homeless adults; such an association has rarely been described in children4,5. We report the case of a child with severe eosinophilia and pediculosis capitis and discuss a possible association.

Case description

A previously healthy, 9-year-old girl presented to the Emergency Department of a tertiary hospital with recurrent episodes of abdominal pain and soft stool (2-3/day) without mucus or blood for eight days. Pain duration was short (<5 min) and occurred 3-10 times per day but did not affect her appetite, activity, or sleep. No exposure to domestic/farm animals, gastroenteritis contact, or travel were reported. She had a history of cow’s milk allergy that resolved at the age of four years and occasional asthma episodes managed with inhaled salbutamol when needed until she was seven years old.

At presentation, her vital signs were normal (blood pressure: 100/50 mmHg, pulse: 82 beats/min, temperature: 36.7 °C, respirations: 25/min). Physical examination only revealed small cervical lymphadenopathy bilaterally. Laboratory investigations showed leukocytosis with eosinophilia [white blood cells (WBC): 25,600 /µL, AEC: 14,848 /µL] but normal hemoglobin and platelet count. Inflammation markers were negative [erythrocyte sedimentation rate: 8 mm/h, C-reactive protein: 0.31 mg/dL]. Renal and liver function tests, electrolytes, uric acid, lactate dehydrogenase, and ferritin levels were normal.
Stool culture and polymerase chain reaction were negative. Interestingly, a full blood count (FBC) performed upon symptom onset seven days earlier had also shown leukocytosis with eosinophilia on two consecutive days (WBC: 17,800 /μL, AEC: 5300 /μL, and WBC: 19,400 /μL, AEC: 7760 /μL). Because of worsening severe eosinophilia, our investigation was two-fold: i) to identify its cause and ii) to diagnose possible end-organ involvement.

Total immunoglobulin E (IgE) was high but within normal range [351 IU/ml (range: 0.5-393 IU/ml)]. The radioallergosorbent testing (RAST) for specific immunoglobulins for common allergens was negative, ruling out food and environmental allergies. Microscopic examination of the stool (x 3) did not show ova of parasites, blood or eosinophils.

Although the blood film showed no other abnormality except increased but normal-looking eosinophils, a bone marrow aspiration (BMA) was planned to exclude hematological malignancies. Normal serum immunoglobulin levels (IgG, IgA, IgM), lymphocyte subsets, and no history of infections ruled out severe immunodeficiency. Normal thyroxine and cortisol levels, auto-antibodies (Anti-Nuclear, Anti-Neutrophil Cytoplasmic Antigen, Anti-ds-DNA Antibodies), and complement components (C3, C4) excluded endocrinological and autoimmune causes.

A chest radiograph (CXR) and abdominal ultrasound revealed no masses (i.e., hilar/abdominal lymphadenopathy or solid tumors). The normal CXR and electrocardiogram excluded pulmonary and cardiac involvement. Despite normal stool microscopy, serum total protein, and albumin, an upper/lower gastrointestinal (GI) tract endoscopy was scheduled to exclude infiltration of the gut mucosa with eosinophils (eosinophilic gastroenteritis). Finally, both the patient and her parents had normal eosinophil counts in earlier FBCs, excluding familial causes.

Interestingly, a repeated thorough physical examination and review of medical history performed while waiting for planned procedures (endoscopy/BMA) revealed a persistent scalp infestation with head lice managed successfully with two applications of an anti-lice lotion based on essential oils. Next day the AEC, albeit still abnormal, showed a remarkable decrease (WBC: 17,200 /μL, AEC: 9740 /μL) (Figure 1). Given the decreasing AEC and symptom resolution (soft stool and abdominal pain), the planned GI endoscopy and BMA were withheld. The AEC continued to gradually drop (Figure 1) to 950 /μL (eight days following her discharge and 23 days from the initial abnormal FBC) and to a minimum of 490 cells/μL five months from diagnosis and has remained within normal limits since. Similarly, IgE dropped from 351 to 81 IU/ml.

As no cause was identified for the increased eosinophils, pediculosis capitis emerged as a possible association because eosinophilia coincided with the diagnosis, resolved with treatment, and neither of the two recurred during follow-up up to twelve months thereafter.

Discussion

We describe a school-aged female with an episode of severe eosinophilia possibly associated with pediculosis capitis. The patient presented with a history of abdominal pain and soft stool for one week. Repeated blood tests confirmed severe eosinophilia. A thorough clinical

**Figure 1:** Diagram showing the trend of white cell count and absolute eosinophil count of our patient during the disease course and follow-up period.

WCC: white cell count, AEC: absolute eosinophil count.
examination and history revealed infestation with head lice, whereas targeted laboratory investigations excluded common causes of eosinophilia, suggesting that the infestation was a possible cause. Pediculosis treatment coincided with correction of the eosinophilia. The case highlights the importance of history and clinical examination and indicates head lice as a possible cause of eosinophilia in this age-group.

Eosinophilia can be a manifestation of various conditions, including atopic diseases (allergic rhinitis, eczema, asthma), infections (parasitic, viral, fungal), malignancies (leukemia/lymphoma, solid tumors), immunodeficiencies/autoimmune/connective tissue diseases, eosinophilic gastrointestinal diseases, adrenal insufficiency, or medications. Primary eosinophilia due to myeloproliferative disease is extremely rare in children. Severe eosinophilia may be due to benign or severe causes, i.e., myeloproliferative diseases/malignancies, hypereosinophilic syndrome or eosinophilic granulomatosis with polyangitis (EGPA), formerly known as Churg-Strauss angitis.

Common parasites possibly associated with mild/moderate eosinophilia in toddlers include gastrointestinal pinworms and *Toxocara* spp. Lice and scabies are common ectoparasites, but scabies that causes intense pruritus, visible skin burrows, and excoriations is the only reported associated with eosinophilia.

Patients with head lice do not routinely undergo blood testing, as systemic manifestations are uncommon; therefore an association with eosinophilia had rarely emerged before. Pediculosis capitis causes pruritus and scratching that can lead to skin infection and cervical lymphadenopathy. There are reports of moderate eosinophilia in adults with body lice. Brouqui et al, found a significant association between severe body lice infestation and peripheral blood eosinophilia (eosinophils >450 /μl) in homeless people. Such an association has much less commonly been reported in children.

Another possible cause of eosinophilia could be its association with iron deficiency anemia (IDA), which is occasionally observed in adults or children with severe, chronic infestations. Our patient case is unique because eosinophilia was isolated, severe, persisted for approximately three weeks, and was finally attributed to pediculosis capitis after exclusion of common causes.

In our patient, the GI symptoms were initially considered to be gastroenteritis; however, no infectious agent was identified. The finding of severe eosinophilia prompted a thorough investigation, including a scheduled GI endoscopy and biopsy that were, however, not performed because the symptoms and eosinophilia gradually resolved and did not recur after pediculosis treatment. Therefore, it was impossible to clarify our hypothesis that the initial GI symptoms were related to the migration of eosinophils to the gut mucosa, which is a limitation of our study.

In conclusion, our case highlights that clinicians should maintain medical history-taking and physical examination skills at the core of their clinical practice to avoid costly and uncomfortable investigations. In addition, further research could explore whether pediculosis can represent a rare but underrecognized cause of eosinophilia in children.

**Conflict of interest**

All authors declare no financial interests or potential conflicts of interest.

**Acknowledgments**

Ioannidou G and Panagopoulou P have contributed equally to the study.

The patient’s parents have provided informed consent to this publication.

This case was presented in the 2nd Conference of the Hellenic Society of Pediatric Gastroenterology, Hepatology and Nutrition, 18-20 October 2019, Thessaloniki, Greece.

**References**