



A Common but an Unusual Disease in a Child with Eosinophilia

Gökcan Öztürk^{1*}, Tutku Parlar¹, Şule Haskoloğlu², Kübra Baskın², Nazlı Deveci², Ergin Çiftçi³, Figen Doğu² and Aydan İkinciogulları²

¹Department of Pediatric Health and Diseases, Ankara University School of Medicine, Turkey

²Department of Pediatric Immunology and Allergy, Ankara University School of Medicine, Turkey

³Department of Pediatric Infectious Diseases, Ankara University School of Medicine, Turkey

Abstract

Hypereosinophilia is generally described as having an absolute eosinophil count of more than 500/mm³ in peripheral blood. 500-1500/mm³ is classified as mild, 1500-5000/mm³ as moderate, 5000/mm³ and above as severe eosinophilia. The causes of eosinophilia in children are similar to those seen in adults and it is separated into two groups as primary and secondary eosinophilia. Primary eosinophilia includes some genetic conditions such as hereditary eosinophilic syndrome. Secondary eosinophilia develops as a reaction to a certain cause. Among possible causes of secondary eosinophilia, we can list infections, atopy, medications, gastrointestinal diseases, lymphocytic diseases, and immunodeficiencies. In this paper, we discuss the differential diagnosis of hypereosinophilia in a 3-month-old girl presenting with generalized seborrheic dermatitis, hypereosinophilia, CMV viremia and pneumonia.

Case Presentation

As the patient initially experienced crusted lesions on a red and puffy skin when she was 1.5 months old, she was taken to a secondary healthcare facility. The patient's lesions were considered to be atopic dermatitis and recommendations were provided accordingly. However, a month later, her lesions increased and she had irritability; so, there was a second visit to the healthcare facility. Her laboratory tests showed a total eosinophil count of 800/mm³, immunoglobulin G, A, M levels were normal for her age, and IgE was 2.500 IU/ml (0-64). In addition to skin lesions, she had hypereosinophilia and high levels of IgE. Food allergy and primary immunodeficiency were considered initially. The patient was referred to our pediatric immunology department with these preliminary diagnoses (Figure 1, 2). At the time of her first admission to our center, the patient was 3 months old. She had generalized erythematous crusted lesions on the skin and seborrheic dermatitis of the hair. On her physical examination, her body weight was at 3rd to 10th percentile, her height was at 10th to 25th percentile, and her head circumference was below 3rd percentile. Other system examinations were normal. In her personal history, she had been hospitalized at newborn intensive care unit for congenital pneumonia for 10 days. In her family history, we found that her parents were second degree relatives. Two daughters of her paternal uncle have died because of unknown reasons before 1 year of age. The patient was referred to our Dermatology department and was diagnosed as atopic dermatitis. She was given topical steroids and recommendations were provided for moistening. Her laboratory tests were performed at our center revealing a white cell count of 43.100/mm³ and an eosinophil count of 16.030/mm³. Other series were within normal limits. There were no atypical cells on blood smear examination. Total IgE was 2.038 IU/ml and CMV PCR was reported as 1.323 copies. Foscarnet treatment was given for CMV viremia. Thymus shadow was observed on her lung X-rays. In the blood culture obtained at an outside center, there was growth of *S. Aureus* and she was given ampicillin sulbactam; however, as there were consolidation areas on the upper lobe and lower lobe superior segment of the right lung together with atelectasis and as dependent ground glass opacities were identified on posterior sides of both lungs in thorax tomography, this treatment regimen was changed to piperacillin tazobactam. Primary immunodeficiency was the first diagnosis in our minds, but her lymphocyte subgroup panel results were reported as normal. Hyperimmunoglobulin E syndrome score calculation was 28 and DOCK-8 expression was normal. Milk specific IgE was reported as 2+ and she was fed with hypoallergenic formula. Stool microscopy and parasite examinations were normal. Skin biopsy did not show any findings correlating with Langerhans cell histiocytosis. Although the patient was referred to our center with the preliminary

OPEN ACCESS

*Correspondence:

Gökcan Öztürk, Department of Pediatrics, Ankara University School of Medicine, Ankara, Turkey, Tel: +905358526250; E-mail: gokcan_ozturk@hotmail.com

Received Date: 29 Mar 2022

Accepted Date: 06 Apr 2022

Published Date: 16 May 2022

Citation:

Öztürk G, Parlar T, Haskoloğlu Ş, Baskın K, Deveci N, Çiftçi E, et al. A Common but an Unusual Disease in a Child with Eosinophilia. *Ann Clin Case Rep.* 2022; 7: 2196.

ISSN: 2474-1655

Copyright © 2022 Gökcan Öztürk. This is an open access article distributed under the Creative Commons Attribution License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original work is properly cited.



Figure 1: a, b) The skin lesions of the patient at the time of admission to our center (Verbal and written consent was obtained from patient's family for the use of photographs).



Figure 3: The patient's condition during her last follow-up visit (Verbal and written consent was obtained from patient's family for the use of photographs).

Positive Pathological Findings that make us consider Primary Immunodeficiency in our patient

- 1) Generalized atopic dermatitis like skin rash
- 2) Leukocytosis, eosinophilia and high levels of IgE
- 3) Consanguineous marriages and family history of infant deaths
- 4) Oral moniliasis as a newborn
- 5) Growth of *S. aureus* in blood culture
- 6) CMV viremia
- 7) Pneumonia

Figure 2: Positive pathological findings.

diagnosis of primary immunodeficiency (Figure 2), we started investigating the causes of secondary hypereosinophilia after these initial test results. Bone marrow aspiration was performed and there was not any infiltrative pathology. In cardiac examination, there was no pathology other than an increase in trabeculation in the apical part of the right ventricle. Abdominal ultrasound examination was reported as normal. After the topical steroids that were prescribed for the skin rash at the time of her admission were stopped, her rash recurred. She was reevaluated by the Dermatology department; scabies were identified in her palms and on her back. Sulphur containing creams were given to the patient and her family with a preliminary diagnosis of scabies. After a month-long treatment, her skin lesions regressed. In the complete blood count, her total eosinophil count was seen to decrease to $1.030/\text{mm}^3$ and IgE level to 74.2 IU/ml (Figure 3).

Discussion

In the differential diagnosis of eosinophilia during childhood, possible causes for secondary eosinophilia should be considered before primary eosinophilia. Because these are more frequently encountered [1,2]. Among the causes of secondary hypereosinophilia, we can list parasitic, fungal, bacterial and viral infections; hyper IgE syndrome and primary immunodeficiencies like T cell immunodeficiency as well as pulmonary, dermatological, oncological, gastrointestinal diseases [3,4]. In our patient, in addition to skin lesions, there was a pathologically elevated eosinophil and IgE level. After diagnostic tests were performed, she was found to have pneumonia and *S. aureus* bacteremia, CMV viremia and scabies skin infection. As the patient experienced bacteremia, pneumonia and viremia during the first three months of her life and there was a consanguineous marriage

of her parents together with a family history of deaths during infancy, she was considered to have primary immunodeficiency; however, her lymphocyte subgroup panel results were reported as normal. Hypereosinophilic syndrome score was calculated as 28 and her DOCK-8 expression was normal. Before examinations were performed for allergic diseases, the patient also had atopic dermatitis. Adding to eosinophilia and elevated IgE levels, her milk specific IgE was reported as 2+ and she was fed with hypoallergenic formula. Yet, this nutritional regimen did not improve her clinical findings. As her findings exacerbated after the completion and cessation of treatment, a dermatology consultation was requested. As a result, her skin rash was found to be correlating with scabies infection and after proper treatment her findings resolved completely. Following the treatment of her infection, blood eosinophilia and IgE levels returned to normal as well. Parasitic infections are one of the best-known causes of eosinophilia [5]. As was the case in our patient, when investigating the causes of secondary hypereosinophilia in patients having atypical skin rashes, scabies infections caused by *Sarcoptes scabiei* should be kept in mind. In a case report presented by Sanchez-Flores, a 3-month-old baby with hypereosinophilia and generalized papule/pustule was identified to have a scabies infection after performing necessary tests and the rash improved following treatment with sulphur [6]. In conclusion, in cases identified to have hypereosinophilia and elevated IgE levels, scabies infection should definitely be kept in mind despite the fact that such cases might have atypical clinical findings.

References

1. Burris D, Rosenberg CE, Schwartz JT. Pediatric hypereosinophilia: Characteristics, clinical manifestations, and diagnoses. *J Allergy Clin Immunol Pract.* 2019;7(8):2750-2758.e2.
2. Schwartz JT, Fulkerson PC. An approach to the evaluation of persistent hypereosinophilia in pediatric patients. *Front Immunol.* 2018;9:1944.
3. Wright BL, Vickery BP. Eosinophils, *Nelson Textbook of Pediatrics*, Elsevier. 2020;1131-3.
4. Nacaroglu HT. Allerjide Kullanilan Temel Testler, *Pediatri*, Ankara Nobel Tıp Yayınları, 2021;386-90.
5. Soussi Gounni A, Lamkhioued B, Ochiai K, Tanaka Y, Delaporte E, Capron A, et al. High-affinity IgE receptor on eosinophils is involved in defence against parasites. *Nature.* 1994;367(6459):183-6.
6. Sánchez-Flores X, Cancel-Artau KJ, Figueroa L. Eosinophilia with leukemoid reaction secondary to *Sarcoptes scabiei*. *JAAD Case Rep.* 2020;8:13-5.