A patient who presented with abdominal pain and eosinophilia

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Case

A 16-year old male patient who presented to emergency department with a complaint of abdominal pain was referred to pediatric surgery for operation stating that he had an abdominal mass. On abdominal ultrasonography performed in pediatric surgery clinic, enlargement in the mesenteric lymph nodes, intestinal malrotation and invagination in an approximately 3 cm segment in the jejunal bowel loops were found. Upper gastrointestinal tract radiography was obtained and the findings were found to be compatible with malrotation, invagination. It was decided to monitor the patient with supportive treatment, because volvulus was not found and there was no problem in the gastrointestinal passage. When complete blood count performed during the follow-up in the ward revealed the following findings: white blood cells: 75 400/mm³, hemoglobin: 13.7 g/dL

and platelets: 293 000/mm³, the patient was referred to the Division of Pediatric Hematology/Oncology with a prediagnosis of leukemia. In the history of the patient, it was learned that he was studying in the Eastern Anatolian region, he presented to the regional public hospital when his complaints started and then presented to a university hospital. He was referred to Cerrahpaşa Medical Faculty, Department of Pediatric Surgery because of his state of emergency. On physical examination, his general status was good, he had no organomegaly and cervical lymphadenopathy the largest one having a size of 1.5x1 cm and left axillary lymphadenopathy (1x1 cm) were present. Peripheral blood smear revealed 78% eosinophils (64 000/mm³) and no atypical cell. His erythrocyte sedimentation rate was found to be 3 mm/h and biochemical measurements were found to be normal.

Diagnosis: Toxocariasis

Echocardiography which was performed to evaluate organ involvement for hypereosinophilic syndrome which was included in the differential diagnosis was found to be normal during the investigations performed to elucidate the etiology of hypereosinophilia. Lung graphy was also found to be normal. ANA, anti-ds DNA, p-ANCA and c-ANCA tests performed in terms of connective tissue diseases and inflammatory bowel diseases were found to be negative. Immunglobuline (Ig) G was found to be 915 mg/dL (normal), Ig M was found to be 289 mg/dL (normal), Ig A was found to be 156 mg/dL (normal) and Ig E was found to be 210 mg/dL (increased). Bone marrow aspiration and biopsy was performed to investigate eosinophilic leukemia. No blasts were detected in the bone marrow excluding increased mature eosinophils and flow cytometry and cytogenetic examination of the bone marrow were found to be normal.

Serological tests for salmonella, brucella, leptospira, echinococcus and *Toxocara canis* were sent. Brucella was found to be 1/1 280 positive, salmonella was found to be 1/40 positive and *Toxocara canis* which was studied simultaneously by ELISA method was found to be 32 NTU positive. When the department of microbiology was consulted, it was learned that brucella and salmonella antibody positivities could be related with cross reaction with toxocara infection and the values found did not show active infection. A diagnosis of toxocariasis was made with these findings. Ophthalmologic examination performed to investigate eye involvement was found to be normal. Eosinophilic leukocytosis regressed spontaneously in the patient who was followed up without treatment. The leukocyte count was found to be 7 600/mm³ and the eosinophil count was found to be 640/mm³ in the final complete blood count performed one month later.

Discussion

Eosinophilia is defined as an eosinophil count higher than 500/mm³ in the peripheral blood. The degree of eosinophilia is classified as mild (500-1 500/mm³), moderate (1 500-5 000/mm³) and severe (>5 000/mm³) (1). Eosinophilia in the peripheral blood may occur in relation with many causes including allergic, infectious and malign diseases (Table 1). The etiology should be elucidated, because treatments for the underlying causes are different and eosinophilia is a sign of serious pictures. It is important to carefully evaluate the patient's history, physical examination, clinical and laboratory findings and specific laboratory tests (1, 2).

Toxocariasis which is among the causes of hypereosinophilia is also named "visceral larva migrans". Toxocariasis occurs as a result of intake of infective eggs of *T. canis* (less frequently *T. cati*) by the oral route. The larvae which

become free with opening of infective eggs in the intestines are transferred to the circulation by entering in the mucosa (3). *Toxocara canis* larvae release large amounts of immunogenic glycolized proteins. These antigens lead to an immune response which causes to production of eosinophils and polyclonal and antigen specific IgE. The typical histopathological lesion includes eosinophilia, multinuclear giant cells and granulomas containing collagen (4). The clinical signs and symptoms may vary depending on the intensity and time of infection, the anatomic location of the larva and the host's immune response. Although it is an infectious disease which can be observed worldwide, it occurs more frequently in tropical regions and rural areas (5).

Toxocariasis may occur in conditions where visceral larva migrans, ocular larva migrans or both clinical pictures are observed in association (6, 7). Visceral larva migrans mostly occurs in children and leads to hepatitis or pneumoniae depending on the movement of the larvae in tissues. In more severe infections, fever, mal-

Table 1. Diseases related with eosinophilia

Allergic diseases

Atopic conditions (urticaria, medication)

Drug-induced eosinophilia

Hematologic/neoplastic diseases

Hypereosinophilic syndrome

Lymphoma

Leukemia

Mastocytosis

Infections

Parasitoses

Fungus infections

Endocrine

Hypoadrenalism

Diseases characterized with specific organ involvement

Skin diseases (eczema, scabies, pemphigus)

Lung diseases (asthma, Loeffler syndrome)

Gastrointestinal diseases (ulcerative colitis, protein losing enteropathy, etc.)

Rheumatoid diseases (sarcoidosis)

Renal diseases (Good Pasture syndrome, peritoneal dialysis, etc.)

Cardiac diseases (endomyocardial disease)

Immunological reactions

Specific immune deficiencies (Omenn syndrome)

Transplant rejection

aise, restlessness, respiratory distress and skin lesions may be observed. Ocular larva migrans mostly occurs in the adolescence and leads to granulomatous reaction around the larvae located in the eye. Rarely uveitis, papillitis or endophthalmitis may also be observed. Toxocariasis may also be manifested only with eosinophilia besides visceral larva migrans or ocular larva migrans (7). Our patient was diagnosed with toxocariasis only with eosinophilia.

Marked leukocytosis is observed as a normal response of the body in many infectious diseases. In the literature, leukocytosis and associated marked eosinophilia have been reported in toxocara infections (8). Leukocytosis and eosinophilia were prominent in our patient. Decreased hemoglobin (Hb) level has been shown in patients infected with toxocara in comparison with uninfected individuals (9), but anemia was not observed in our patient.

Toxocara infection should be considered in presence of leukocytosis, eosinophilia and hypergammaglobulinemia (IgE, IgG) in association with clinical findings. Although there is no definite approach in treatment of toxocara, patients with mild symptoms are followed up without treatment and the disease resolves spontaneously. However, eosinophilia may persist for a long time because of the antigenic stimulus of the dead larvae. Albendazole is used in severe cases. In addition, prednisolone may be added to treatment in presence of respiratory distress, myocardial involvement and neurological symptoms.

Our patient presented because of abdominal pain and simultaneous leukocytosis/eosinophilia was found in association with volvulus and invagination during investigations. In the literature, no human case of volvulus/ invagination accompanying toxocara infection has been reported, but one animal case has been reported (10). The etiology of volvulus/ invagination in our patient was not clearly elucidated and it was thought to be related with mesenteric lymphadenitis. It was decided that the patient be followed up without treatment, because he had mild complaints. The patient's leukocyte count regressed to 20 000/mm³ in ten days and to 7 600/mm³ after one month and eosinophilia regressed up to 640/mm³. The level of toxocara antigen was found to be 12 NTU (low) at the end of the first month. The patient is still being followed up by us.

The etiology of hypereosinophilia should be urgently elucidated, because treatments of the underlying causes are different and it is a sign of severe pictures including hypereosinophilia syndrome, inflammatory bowel disease, leukemia and lymphoma. Parasitic infections should primarily be considered in the etiology especially in the rural areas in our country. In this article, we aimed to review the approach to hypereosinophilia,

remind the diseases included in the differential diagnosis and emphasize the role of parasitic infections in the etiology.

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