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Case Report

Per Orbital Edema in a Child with Serologically Confirmed Toxocariasis: A Case Report

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Abstract

Toxocariasis is a globally distributed zoonotic helminthic infection caused by *Toxocara* species. Humans are accidental hosts in whom the larvae cannot complete their life cycle; instead, they migrate through various tissues, resulting in a broad spectrum of clinical manifestations. We report the case of a 4-year-old boy from western Iran with a history of close contact with cats and dogs who presented with per orbital edema and swelling of the hands and feet. He was referred to the Diagnostic Laboratory of Helminths, School of Public Health, Tehran University of Medical Sciences, Tehran, Iran in March 2024. Laboratory investigations revealed marked hypereosinophilia, raising suspicion of a parasitic etiology. Serological testing was positive for toxocariasis, while computed tomography (CT) revealed subtle hepatic abnormalities. The patient's clinical manifestations improved following concomitant treatment with albendazole and prednisolone, and the eosinophil count returned to the normal range during follow-up. This case highlights the importance of considering toxocariasis in children presenting with edema, marked hypereosinophilia, and a history of close contact with cats or dogs. However, although the patient was serologically positive for toxocariasis, not all clinical manifestations could be definitively attributed to the infection.

Introduction

Toxocariasis is a zoonotic helminthic disease caused by the larval stages of *Toxocara canis* and *T. cati*. Humans are accidental (paratenic) and dead-end hosts in whom the parasite cannot complete its life cycle. After ingestion of embryonated eggs, the larvae migrate through various tissues without developing into adult worms, resulting in different clinical manifestations, including visceral larva migrans (VLM), ocular larva migrans (OLM), neurotoxocariasis, and covert toxocariasis (1).

Although toxocariasis occurs worldwide, it is more prevalent in tropical and subtropical re-

gions and in areas where close contact with dogs and cats increases the risk of infection. The global seroprevalence of toxocariasis is estimated at approximately 19%, with higher rates in lows and middle-income countries (2). In Iran, the pooled seroprevalence is about 6.6%, and children are among the most affected due to frequent exposure to contaminated soil and close contact with dogs and cats (3, 4).

The diagnosis of human toxocariasis remains challenging because parasite eggs are not excreted in human feces, making stool examination ineffective. Tissue biopsy can confirm the presence of larvae but is invasive and rarely performed, while molecular techniques have limited sensitivity because of the low parasite burden and the deep tissue localization of larvae. Imaging modalities may help identify organ involvement but are generally non-specific and cannot establish the diagnosis independently. Therefore, serological methods remain the most important and widely used diagnostic tools for human toxocariasis. ELISA is widely used as the initial screening assay because of its high sensitivity, whereas Western blot serves as the confirmatory test owing to its higher specificity and reduced cross-reactivity with other helminth infections (5). Because toxocariasis often presents with non-specific clinical manifestations, it may be overlooked, particularly in patients with unexplained eosinophilia.

We present a pediatric case of toxocariasis with initial per orbital edema followed by edema of the extremities. Laboratory evaluation revealed significant eosinophilia as a key finding, and serological testing supported the diagnosis.

Case presentation

A 4-year-old boy with a history of close contact with stray dogs and cats presented in March 2024 with right-sided per orbital edema, followed by edema of the hands and feet. Initially, the patient was examined by an ophthalmologist, and no ocular abnormalities were detected. As the edema persisted without improvement, he was admitted to the hospital for further evaluation (Fig. 1). Following hospitalization, a comprehensive diagnostic workup was initiated to determine the underlying cause of the recurrent edema. Laboratory investigations revealed leukocytosis, thrombocytopenia, elevated liver enzyme levels with ultrasonography evidence of hepatic inflammation, marked eosinophilia, and increased serum IgE, ESR, and CRP level (Table 1).

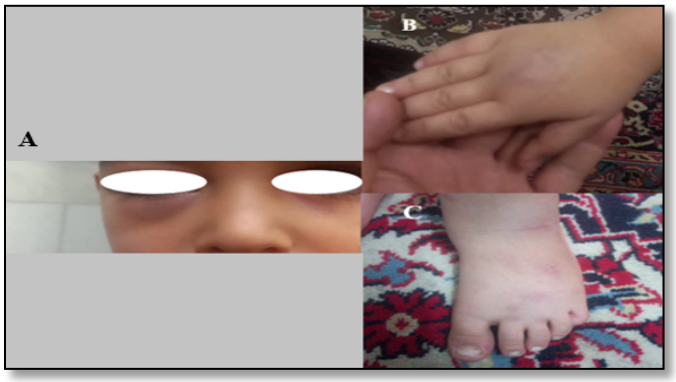

Fig. 1: Clinical photographs showing swelling of the right side of the body: (A) right per orbital swelling, (B) swelling of the dorsum of the right hand, and (C) swelling of the dorsum of the right foot

Cardiopulmonary evaluation, including chest CT and echocardiographic assessment showed no evidence of cardiac or pulmonary disease contributing to the patient's edema. Abdominal CT revealed mild hepatic inflammatory changes, including per portal edema and heterogeneous liver enhancement. A hematology consultation was performed for the patient. Peripheral blood smear examination, serum protein electrophoresis, and BMA (bone marrow aspiration) and BMB (bone marrow biopsy) revealed no evidence of hematologic malignancy or other primary hematologic disease.

Table 1: Laboratory test results of the child before and after Albendazole & Prednisolone administration

Indicator	Before treatment	One month after treatment	Three month after treatment	Reference Range
White blood cell count (×1000/µl)	34.2	25.90	10.38	4-11
Eosinophil (%)	55	15	1	<5
Hemoglobin concentration (g/dl)	12.4	12.8	12.1	12-16.5
Platelet count (×1000/µl)	60	72	154	150-450
SGOT (IU/L)	-	29	26	Child <60
SGPT (IU/L)	-	61	68	Child <50
LDH (U/L)	579	548	510	4-6 Years <615
CRP Quantitative (mg/L)	33.4	14.4	7	0-10
Anti CCP (U/mL)	3.1	-	-	<10
ESR	40	17	1	Up to 10
IgE	156.7	-	-	1-5 Years: up to 60
Titer of specific *Toxocara* spp., IgG	33.4	-	5.5	11 NTU: seropositive result 9–11 NTU: borderline (doubtful) <9 NTU: seronegative result

The patient was also evaluated for rheumatologic diseases, including systemic lupus erythematosus and systemic vasculitis, were also excluded based on negative immunological findings, as well as for Renal disorders, including nephrotic syndrome and other protein-losing nephropathies, were excluded based on normal renal function, the absence of proteinuria, and normal serum albumin levels. Therefore, the clinical findings, along with the marked hypereosinophilia and positive serology, were highly suggestive of active visceral toxocariasis. Following an eight-month clinical course and several diagnostic investigations, due to persistent significant eosinophilia, the patient was eventually referred by the attending physician to the "diagnostic Laboratory of Helminths at the School of Public Health, Tehran University of Medical Sciences" for assessment of possible toxocariasis.

Serological test of anti-*Toxocara* IgG was performed using a commercial ELISA kit (Novalisa, NovaTec, Germany), which has a sensitivity of 98.63% and a specificity of 96.92% (According to the manufacturer's protocol, cross-reactivity with antibodies against *Ascaris lumbricoides* and *Schistosoma* species cannot be completely excluded) testing procedures followed the manufacturer's protocol. Absorbance values were recorded at 450 nm, and IgG concentrations were derived based on the kit's guidelines. The patient tested positive for anti-*Toxocara* IgG antibodies by ELISA (Table 1). To minimize the possibility of cross-reactivity with other helminth infections, three consecutive stool samples were examined by direct microscopy, formalin–ether concentration, and nutrient agar culture, with no intestinal parasites detected. Furthermore, because of the patient's travel history to northern regions, where *Strongyloides stercoralis* infection is endemic, serological testing for *S. stercoralis* was also performed and was negative.

Finally, considering the patient's history of close contact with stray dogs and cats, compatible clinical manifestations, persistent, elevated serum IgE levels, mild hepatic involvement, exclusion of alternative diagnoses, and

supportive positive anti-*Toxocara* IgG serology, visceral toxocariasis was considered the most likely diagnosis, and combination therapy with albendazole and prednisolone was initiated. Albendazole was prescribed to eradicate the underlying *Toxocara* infection, whereas prednisolone was administered to control the excessive eosinophilic inflammatory response and reduce immune-mediated tissue inflammation. Prednisolone 5 mg tablets were prescribed three times daily for one week, followed by once daily for three months until re-evaluation. Albendazole was administered at 400 mg twice daily for a 14-day course. Three months after treatment, the patient returned for follow-up as requested by the treating physician. Repeat anti-*Toxocara* IgG ELISA testing was performed to evaluate the therapeutic response. A decline in antibody titers to below the cut-off value, together with clinical improvement and normalization of eosinophil counts, was considered indicative of a favorable therapeutic response (Table 1).

Discussion

Human toxocariasis is a neglected zoonotic helminthic infection with a wide spectrum of clinical manifestations, ranging from asymptomatic infection to severe visceral or ocular disease. Clinical presentation depends on parasite burden, host immune response, and the organs affected, often making diagnosis challenging, particularly in children with atypical manifestations that mimic allergic, autoimmune, hematologic, or infectious diseases (1).

Our patient had a history of close contact with stray dogs and cats, an established risk factor for *Toxocara* infection. However, toxocariasis was not initially suspected because the child presented with unilateral per orbital edema followed by recurrent edema of the hand and foot, an uncommon manifestation of visceral toxocariasis. Consequently, extensive investigations were performed to exclude more common causes of recurrent edema. Renal disease was ruled out based on normal renal function, absence of proteinuria, and normal serum albumin levels. Autoimmune and hematologic disorders were also excluded by negative immunological findings, hematologic evaluation, and bone marrow aspiration. Such atypical inflammatory features may mimic autoimmune, hematologic, or infectious disorders and contribute to diagnostic delays in toxocariasis. Per orbital and peripheral edema can also occur in various infectious diseases, including Epstein–Barr virus infection (6), bacterial orbital cellulitis (7), and parasitic infections such as *trichinellosis* and *toxocariasis* (8). In our patient, the absence of renal, rheumatologic, hematologic, and other infectious causes, together with marked eosinophilia and positive *Toxocara* serology, supported that recurrent edema was a manifestation of active toxocariasis. Ocular involvement is a recognized manifestation of toxocariasis and can complicate diagnosis. Jayawardene et al reported a child with unilateral per orbital swelling who was initially treated for orbital cellulitis, but persistence of symptoms and further evaluation led to the diagnosis of ocular toxocariasis. In contrast, our patient had a similar initial presentation but no ocular involvement, and final diagnosis was consistent with visceral toxocariasis (9).

The key laboratory findings in our patient were persistent hypereosinophilia and noticeably elevated total serum IgE level. Ha et al also reported that persistent eosinophilia and elevated total IgE are important laboratory findings in pediatric toxocariasis (10). Our patient also showed mild hepatic involvement, with elevated liver enzymes and imaging abnormalities, consistent with visceral toxocariasis. In contrast, Huynh et al reported severe hepatic involvement mimicking a liver abscess, highlighting the broad spectrum of hepatic manifestations (11).

An additional diagnostic challenge in this case was the coexistence of documented sensitization to grass pollen and fungal spores, and

the patient's presentation was initially attributed to an allergic disorder. Recent evidence from Iran also highlights the overlap between toxocariasis and allergic diseases. Similarly, Pouryousef et al reported a higher seroprevalence of *Toxocara* infection among children with allergic asthma, however persistent eosinophilia, positive *Toxocara* serology, and clinical response to treatment supported toxocariasis as the underlying diagnosis (12). Although pulmonary manifestations are among the most frequently reported features of visceral toxocariasis, our patient had no respiratory symptoms. This finding highlights the broad clinical spectrum of the disease and emphasizes that the absence of pulmonary involvement does not exclude the diagnosis (13).

In endemic settings, positive serological findings should be interpreted cautiously because anti-*Toxocara* antibodies may persist after previous exposure and do not necessarily indicate active infection. Therefore, the diagnosis of toxocariasis should be based on the integration of epidemiological history, compatible clinical manifestations, laboratory findings—particularly marked eosinophilia and elevated serum IgE—and serological results rather than serology alone (5). Similarly, Monti et al. emphasized that while ELISA is an appropriate screening tool, confirmatory Western blot improves diagnostic specificity, and serological findings should always be interpreted within the overall clinical context (14). Likewise, El-Sayed and Ramadan highlighted that an integrated clinico-epidemiological and laboratory approach is essential for the diagnosis of pediatric toxocariasis and recommended albendazole as the treatment of choice, with corticosteroids reserved for selected severe cases (13). Our case further supports this diagnostic and therapeutic strategy. A recent pediatric case reported visceral toxocariasis with marked eosinophilia, elevated serum IgE levels and pulmonary involvement that responded well to combination therapy with albendazole and prednisolone (15). Likewise, our patient also showed a favorable clinical and laboratory response to the same therapeutic regimen, despite the absence of respiratory manifestations. These observations suggest that combined anti parasitic and corticosteroid therapy may be beneficial in carefully selected patients with prominent eosinophilia and inflammatory manifestations, even in the absence of severe organ involvement. Finally, this case highlights the importance of considering toxocariasis in the differential diagnosis of unexplained recurrent per orbital or peripheral edema accompanied by persistent eosinophilia, even in children with evidence of allergic sensitization. Early serological testing and a systematic diagnostic approach may prevent unnecessary invasive investigations and reduce diagnostic delays.

Conclusion

This case demonstrates the remarkable clinical variability of pediatric visceral toxocariasis with recurrent unilateral per orbital and peripheral edema representing an uncommon presentation. Careful integration of clinical, epidemiological, and serological findings is essential for recognizing atypical cases and ensuring timely diagnosis and management. Awareness of these unusual presentations may contribute to earlier diagnosis, appropriate treatment, and improved clinical outcomes in pediatric patients.

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Conflict of interest

The authors report that they have no conflicts of interest.

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