Eosinophilic Ascites in a Patient with Toxocara Canis Infection. A Case Report

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Abstract

We report the case of a young female patient, admitted for a recent ascites of unknown origin. The acute onset was with colicky abdominal pain and peritoneal effusion, which led to the suspicion of perforated ulcer. A diagnostic laparoscopy was performed which showed free peritoneal fluid and normal abdominal viscera. At upper gastrointestinal endoscopy, performed a few days later, patchy erythema in the antral region and duodenal edema were revealed. Duodenal biopsies showed marked eosinophilic infiltration. The ascitic fluid was straw coloured, sterile with 90% eosinophils. Eosinophilic gastroenteritis was suspected, but differential diagnosis required the exclusion of migrant parasites. The stool exams were negative but serology for Toxocara antibodies was positive. The treatment with albendazole (Zentel 400 mg twice a day for 5 days) led to the disappearance of signs and symptoms. The eosinophilic infiltrate of the gut was absent in duodenal biopsies taken two months later.

The final diagnosis was consistent with Toxocara canis infection while the clinical, sonographic and histological findings suggested an eosinophilic gastroenteritis. We emphasize the need to exclude parasitic infection in all patients with eosinophilic gastroenteritis symptoms.

Key words

Eosinophilic ascites - eosinophilic gastroenteritis - human toxocariasis

Rezumat

Prezentăm cazul unei tinere femei, internată pentru o ascită de cauză neprecizată, recent instalată. Debutul a fost acut, cu durere abdominală colicativă și colecție peritoneală, ceea ce a condus la suspiciunea unui ulcer perforat. Laparoscopia diagnostică a evidențiat prezenta lichidului liber în peritoneu și organe abdominale normale. La endoscopia digestivă superioară, efectuată câteva zile mai târziu, s-a evidențiat eritem parțial în regiunea antrală și edem al mucoasei duodenale. Biopsiile duodenale au relevat infiltrat eosinofil abundant. Lichidul ascitic, serocitric a fost steril, cu un conținut de 90% eosinofile. S-a susținut o gastroenterită eosinofilică, dar diagnosticul diferențial necesită exclusiunea unei infestații cu paraziți cu migre re vestală. Examele coproparazitologice au fost negative, dar anticorpi anti Toxocara au fost pozitivi. Tratamentul cu albendazole (Zentel 2 x 400 mg/zi- 5 zile) a determinat dispariția semnelor și simptomelor. Infiltratul eosinofil din mucoasa duodenală a fost absent în biopsiile de control, recoltate 2 luni mai târziu.

Diagnosticul final a fost de toxocaroză, în timp ce trăsăturile clinice, ecografice și histologice au sugerat o gastroenterită eosinofilică. Subliniem necesitatea excluzionii infestației parazitare la toți pacienții cu simptome de gastroenteritară eosinofilică.

Introduction

Eosinophilic ascites is rarely observed in clinical practice. It may be associated with abdominal lymphomas, peritoneal dialysis, eosinophilic gastroenteritis, hypereosinophilic syndrome and some migrant parasites (1-4).

Eosinophilic gastroenteritis is characterized by eosinophilic infiltration into one or more layers of the gastrointestinal tract (1-4). The pathogenesis is so far poorly understood. Up to 40% of cases were reported to have underlying allergy (1-4). It affects adults as well as children and involves any area of the gastrointestinal tract, although the stomach and proximal small bowel seem to be the most commonly affected (1-4). Eosinophilic ascites is common in suberosal type of eosinophilic gastroenteritis (1-4).

Human toxocariasis is usually acquired by ingestion of embryonated eggs of Toxocara, found in areas contaminated
with dog feces. It is common in small children with geophagia (5-9). In adults, the infection is possible by eating raw vegetable or raw meat or liver from potential paratenic hosts such chickens, lambs or calves (5-9). In the small intestine, the larvae hatch from the eggs, then penetrate the intestinal wall reaching the blood vessels. Via portal vein they pass through the liver and lungs to the left heart from where they are disseminated by systemic circulation. These larvae may penetrate through the capillary vessels to the surrounding tissues where they may become encapsulated within granulomas. Previous studies showed that the larvae have been found in the liver, lungs, heart, eyes and brain (5-9). The clinical picture of the disease called “visceral larva migrans” varies depending on the organ involved. The following clinical forms have been described: general, dermatologic, pulmonary, hepatic, rheumatologic, cardiac, neurologic, ocular (5-9). The eosinophilic ascites and pleural effusion have been rarely reported (10,11).

The case of a young female infected with Toxocara canis who developed symptoms which mimicked the eosinophilic gastroenteritis is presented.

**Case report**

A 17 year old female was admitted for recent ascites of unknown origin. The onset of the disease was 7 days before admission, when the patient complained of acute abdominal pain, mostly in the right iliac fossa. Nausea, vomiting and diarrhea were associated. After a first surgical examination, there was a suspicion of appendicitis. The presence of free fluid in the peritoneal cavity, evidenced by the abdominal sonography, suggested the diagnosis of perforating ulcer. A diagnostic laparoscopy was performed which did not reveal lesions of abdominal and genital organs. It confirmed the presence of a free fluid, straw coloured (0.5%). This was sterile. The upper gastrointestinal endoscopy showed reflux, patchy erythema in the antral region, and duodenal edema. Biopsies were taken from the second portion of the duodenum. Due to the fact that the origin of the ascites remained unknown, the patient was referred to our gastroenterological department.

The general examination was unremarkable, no presence of lymphadenopathy or skin lesions were noted. Lung was clear on auscultation. The patient had a blood pressure of 110/60 mmHg; the heart sounds were normal on auscultation. The abdomen was tender, painless. No hepatomegaly, splenomegaly or masses noted on palpation.

At presentation, a complete blood count showed hemoglobin of 13.2 g/dl, hematocrit 38.7%, 10,500 leukocytes/mm³, with 44% eosinophils on peripheral smear. Erythrocyte sedimentation rate was 7 mm /hour. Electrolytes, urine analysis and renal function were all normal. The serum and urine amylase of blood and urine showed normal values. Liver enzymes were mildly increased: ASAT 78 U/L (normal 31), ALAT 60 U/L (normal 32). The serum total protein (5.9 g/dl) and albumin were slightly diminished.

The abdominal sonography revealed moderate amount of free fluid. The liver, spleen, pancreas and genital organs were normal. The gallbladder contained small stones. Small mesenteric lymph nodes were visible. The walls of the stomach, duodenum and small bowel was thickened (Fig.1). The ascitic fluid prelated under sonographic guidance was clear and straw coloured. The cell count showed 4500-cells/ mm³ with 90% eosinophils (Fig.2). Glucose, amylase and LDH had normal values. Based on these results, eosinophilic ascites was diagnosed.

The clinical features, eosinophilic ascites, thickening of the stomach and intestinal walls, and hypereosinophilia were consistent with the diagnosis of eosinophilic gastroenteritis. The diagnosis requires histological evidence of eosinophilic infiltration into the digestive tract (1-4). The biopsy of duodenum showed eosinophilic infiltrate in mucosa and lamina propria of the duodenal wall (> 20 eosinophils /high power field) (Fig.3).

Eosinophilia and accumulation of the eosinophils in the gut is a common feature in food allergy (12). Our patient had a history of urticaria, but no evidence of the food specific IgE antibodies. The skin prick testing diagnosed atopy. Total Ig E was increased: 412 UI/ml (normal range 5 to 200). Total Ig E increase may be seen also in infection with visceral parasites and in eosinophilic gastroenteritis (1, 7,9).

We initiated the treatment with Montelukast 10 mg/day (Singulair, MSD) which was stopped after two weeks when serology for parasitic disease was available. Thereafter no decrease of eosinophilia was recorded. Montelukast is a selective leukotriene receptor antagonist, which actively and selectively blocks the D₄ receptor of cysteinyl leukotrienes present in eosinophils (13,14). Eosinophils are not removed but their activity is negated. The use of Montelukast in eosinophilic esophagitis showed improvement of symptoms but the infiltration with eosinophils did not change (14).

The differential diagnosis also included the possibility of parasitic infestation. Some intestinal parasites such as Toxocara species, Strongyloides stercoralis, Trichinella spiralis may penetrate the gut and disseminate, causing visceral symptoms. The stool tests for ova and parasites were negative. Serology for Trichinella spiralis was negative but positive for Toxocara canis antibodies (Ig G). The diagnosis of active toxocariasis was based on the association of seropositivity for Toxocara antibodies, high peripheral eosinophilia and increased serum total Ig E concentration. We initiated Albenzazole (Zentel - SmithKline Beecham) treatment, 400 mg twice a day for 5 days. One month later, ascites was no longer apparent at sonographic evaluation and the eosinophil count decreased to 8%. The thickness of gastric and duodenal walls, sonographic evaluated, became normal only after two months. At this moment, eosinophilic infiltration was absent in the biopsies of the stomach and the duodenum.

We concluded that the clinical picture, laboratory findings and therapeutic results made the diagnosis of active toxocariasis pertinent regarding our patient.
liver parasite), Anisakis (acquired by eating raw seafood) (5-9). Human toxocariasis is caused by Toxocara canis (a canine roundworm) or rarely by Toxocara cati. The adult forms of both ascarids live in the small intestine of their definitive hosts (dogs or cats). The female worms may excrete up to 200,000 eggs per day. Toxocara eggs, passed in the feces, need several weeks of optimal environmental conditions (10-35°C, high soil humidity) to develop to infective, embryonated eggs (5-9). Infection can occur by ingesting embryonated eggs or by transmission in utero from the infected mother (7). Older dogs tend to have dormant infections, but pregnancy reactivates Toxocara, ensuring transmission to the next generation (6). The main source of eggs is the feces of puppies younger than 3 months (because they are infected in utero) and of lactating female dogs (5-9).

Human toxocariasis is a soil-transmitted zoonosis, Geophagia and poor personal hygiene are the main risk factors (5-9). Outdoor parks in urban and suburban settings are highly contaminated. The proportion of soil samples positive for Toxocara eggs range from 15 to 78% (9). Toxocara eggs have been also recovered from soil samples from gardens and from salads or other raw vegetables (9). Therefore the consumption of raw vegetables grown in contaminated gardens may result in low dose infection (5-9).

Less commonly, the infection is associated with consumption of raw meat from potential paratenic hosts such as chickens, lambs or calves. The infective larvae can be released from animal tissues during digestion and subsequently cause human toxocariasis (9). In our patient the source of contamination remains unclear (the patient admitted that, occasionally, she ate raw vegetables).

The human host is aberrant with respect to the completion of the life cycle. The larvae hatch from infective eggs, penetrate the walls of the small intestine, but fail to develop to adult worms. They wander throughout the body for months causing tissue damage (5,7,9). The presence of the larvae in the tissues heralds the onset of marked immediate-type and delayed-type of hypersensitivity responses. The immediate hypersensitivity responses to larvae produce symptoms characteristic of visceral larva migrans (5-9).

In 1950, Wilder described the first patient with ocular larva migrans. In 1952, Beaver and colleagues reported a similar series of children with multisystem disease - visceral larva migrans (7-9). For many years this helminthiasis was regarded as an uncommon pediatric disease. The clinical signs and symptoms are variable depending on which tissue has been invaded, the number of migrating larvae and the age of the host. Symptoms resembling asthma, enlargement of the liver and the spleen, myocarditis, nephritis, seizure, neuropsychiatric symptoms, allergic manifestations like urticaria and edema have been reported (5,7-9). Pleural and peritoneal effusions were rarely described as manifestation of visceral larva migrans (10,11,15). Eosinophilia up to 70% and hypergammaglobulinemia of Ig M, Ig G and Ig E classes are commonly present (5-9).

Discussion

The “Visceral larva migrans” may be caused by several nematodes of animals like Toxocara canis, Toxocara cati, Trichinella spiralis, Ascaris suum (a common intestinal roundworm of domestic swine), Capillaria hepatica (a rat
Examination of stools has no role in the evaluation of toxocariasis. The diagnostic is based on serologic findings. The enzyme–linked immunoabsorbent assay (ELISA) which employs antigen secreted by the second stage larvae has sufficient specificity to be the best indirect test for diagnosing of infection (5,7,8). The positive reaction for Toxocara antibody is not capable of distinguishing between current and past infection. The diagnosis of active toxocariasis is based on the association of positive serological tests for toxocara antibody and peripheral eosinophilia. The increase of serum total IgE concentration > 500 UI is further evidence of recent infection (9).

Sometimes the manifestations of human toxocariasis resemble eosinophilic gastroenteritis (10,11). Experimentally induced canine toxocariasis in mice was associated with moderate ascites, hepatomegaly, lymphadenopathy and focal lesions in liver, lung, kidney, intestine. Microscopically, focal eosinophilic gastroenteritis was found (16). In our patient besides ascites and eosinophilic gastroenteritis, the liver (transient increased aminotransferases) and abdominal lymph nodes were involved. One should look for a potential parasitic infection in all patients with symptoms of eosinophilic gastroenteritis (10,11).

Most cases of visceral larva migrans are self-limited and not life threatening (5,7-9). Treatment is reserved for cases with overt manifestation, such as our patient. The treatment of choice for toxocariasis is albendazole (5,17). The anthelmintic therapy is efficient in most cases; some of them, however, require steroid therapy (5,17). Regarding our patient, the therapy with albendazole was efficient; the symptoms ceased, the eosinophile count normalized, and the microscopy of the duodenal wall showed no eosinophilic infiltration after two months.

Visceral larva migrans is very rarely observed in our clinical practice. In Romania only a few cases have been so far reported (18,19). Nevertheless, we must be aware of this disease, when we find high eosinophilia without an apparent cause.

References