CEREBRAL TOXOCARIASIS AFTER CONSUMPTION OF RAW DUCK LIVER

BODO HOFFMEISTER,* SVEN GLAESER, HOLGER FICK, SEBASTIAN PORNSCHLEGL, NORBERT SUTTORP, AND FRANK BERGMANN
Department of Infectious Diseases and Pulmonology, Charité University Hospital, Berlin, Germany; Department of Neurology, Schloßpark-Klinik, Berlin, Germany; Department of Infectious Diseases and Pulmonology, Charité University Hospital, Berlin, Germany

Abstract. Human toxocariasis is usually contracted by exposure to contaminated soil. This disease is rarely transmitted by raw meat or giblets of paratenic animals, such as chickens, lambs, or cows. We present a case of isolated cerebral toxocariasis presumably caused by the consumption of raw duck liver. This 55-year-old woman had sudden-onset hemiparesis of the right leg, eosinophilia of 30%, and markedly elevated total serum IgE levels. Magnetic resonance imaging demonstrated multiple cerebral hyperintense lesions on T2-weighed images. Tests for antibodies to *Toxocara* in serum and cerebrospinal fluid yielded highly positive results. Repeated courses of albendazole and corticosteroids led to significant clinical improvement.

INTRODUCTION

Human toxocariasis is a common helminthozoonosis in industrialized nations. The disease is caused by infestation with larvae of the nematode worms *Toxocara canis* or *T. cati*. The eggs of the worms are excreted with the feces of their definite hosts, dogs and cats. Transmission to humans occurs when infective embryonated eggs are ingested. This usually occurs after exposure to contaminated soil, for instance by consumption of contaminated raw vegetables, poor personal hygiene, or soil eating (geophagia).

Most commonly children are affected, e.g., while playing in sandboxes or playgrounds. However, the modes of transmission in adults are less clear. Many species other than definite hosts and humans can also become infested by *Toxocara* species. Consumption of raw or undercooked meat and giblets of these animals can also result in transmission of toxocariasis. Raw liver appears to be particularly infective: several cases of human toxocariasis have been observed after consumption of raw chicken, cow, or lamb liver.

We report a case of cerebral toxocariasis presumably caused by the consumption of raw duck liver. We review the transmission of human toxocariasis by paratenic hosts, with emphasis on the role of raw liver as the source of infestation.

CASE REPORT

In January 2006, a 55-year-old previously healthy woman had sudden-onset weakness of the right leg, gait disturbance, a sensory loss of the right arm, and a frontal headache. These symptoms had occurred suddenly the day before presentation. Two weeks earlier, the patient and her husband had eaten a baked duck. The patient but not her husband had also consumed the raw liver of the duck. A day later she developed an acute gastroenteritis with a temperature of 38°C. After a few days, these symptoms subsided spontaneously. Shortly thereafter, fits of coughing and sneezing ensued. The husband exhibited no symptoms.

Upon admission, neurologic examination showed a spastic hemiparesis of the right leg with a positive Babinski reflex, hypoesthesia of the right arm, and vertigo. Laboratory findings showed a leukocytosis of 20.1/μL (normal value < 9.8/μL) with marked eosinophilia of 30.0% (< 1%), a total serum IgE level of 16.986 IE/mL (< 87 IE/mL), a lactate dehydrogenase level of 493 U/L (< 279 U/L), and a C-reactive protein level of 7.15 mg/dL (< 0.5 mg/dL). Cerebrospinal fluid was clear and colorless with 0 cells/μL (< 5 cells/μL). Magnetic resonance imaging (MRI) performed the following day showed several cortical and subcortical lesions in both hemispheres. On the T1-weighed images, these lesions were hyperintense, whereas in the T2-weighed and fluid-attenuated inversion recovery images the lesions appeared hypointense (Figure 1A). Treatment with prednisone (250 mg intravenously for three days followed by 75 mg orally once a day) was initiated and the patient was transferred to our department for further examination. An enzyme-linked immunosorbent assay for antibodies to *T. canis* yielded highly positive results in serum: values surpassed the upper limit of the test (approximate titer 1:4096). These findings together with peripheral blood eosinophilia, the markedly elevated total serum IgE level, and the neuroimaging results, suggested visceral larva migrans syndrome with involvement of the central nervous system. A lumbar puncture was repeated. Titers for *T. canis* were also markedly elevated in cerebrospinal fluid (approximate titer > 1:2048), which confirmed the diagnosis. Results of serologic examinations for toxoplasmosis, echinococcosis, trichinosis, and cysticercosis, and multiple stool examinations were negative.

Further examinations were carried out to investigate for manifestations in other organs. Ophthalmic examination, motor-evoked potentials, abdominal ultrasonography, echocardiography, and a computed tomography scan of the thorax, abdomen, and pelvis, as well as bone marrow examination all yielded normal results. A treatment scheme combining dexamethasone, 8 mg orally three times a day, and albendazole, 400 mg orally twice a day, was initiated. Albendazole was discontinued after five days and the dexamethasone dosages were reduced progressively. Under this therapy, the neurologic status of the patient steadily improved: the patient began to walk again, and hypoesthesia of the right arm and headache subsided. In addition, eosinophilia and total serum IgE levels decreased (Figure 1B). Another MRI scan after discontinuation of the therapy showed significant reduction in number and size of lesions (Figure 1A). The patient was discharged five weeks after admission and began a neurologic rehabilitation program.

* Address correspondence to Bodo Hoffmeister, Department of Infectious Diseases and Pulmonology, Charité University Hospital, Augustenburger Platz 1, 13353 Berlin, Germany, E-mail: b.hoffmeister-infekt@charite.de
Toxocara larvae. A. MRI performed on day 2 after presentation (upper left panel) shows multiple cortical and subcortical hyperintense lesions in T2-weighted images in both hemispheres. Three weeks later, i.e., after discontinuation of antihelminthic therapy with albendazole, there was partial regression of the lesions (upper right panel). In three-month (lower left panel) and six-month (lower right panel) follow-up examinations, no new lesions were detected. The lesions underwent cystic transformation. B. Clinical course according to percentage of eosinophils (Eos%), absolute counts of eosinophils (Eos/nL) and total serum-IgE levels in peripheral blood. Alb = albendazole; Pre = Prednisone.

**Figure 1.** Clinical course of the patient indicated by serial magnetic resonance imaging (MRI) and laboratory parameters. A, MRI performed on day 2 after presentation (upper left panel) shows multiple cortical and subcortical hyperintense lesions in T2-weighted images in both hemispheres. Three weeks later, i.e., after discontinuation of antihelminthic therapy with albendazole, there was partial regression of the lesions (upper right panel). In three-month (lower left panel) and six-month (lower right panel) follow-up examinations, no new lesions were detected. The lesions underwent cystic transformation. B, Clinical course according to percentage of eosinophils (Eos%), absolute counts of eosinophils (Eos/nL) and total serum-IgE levels in peripheral blood. Alb = albendazole; Pre = Prednisone.

At a three-month follow-up visit, laboratory results again showed markedly elevated eosinophil counts of 52.7%, leukocytosis (21.05 cells/nL), a total serum IgE level of 10.614 IE/mL, and a CRP level of 2.81 mg/dL. These findings were interpreted as ongoing disease activity, although no new lesions on MRI were detected (Figure 1A). To prevent a disease relapse, another treatment course with albendazole and prednisone was initiated. In further follow-up examinations after six and nine months, no new lesions appeared on MRI, and eosinophilia and total serum IgE levels decreased. Currently, a mild right-sided spastic hemiparesis remains; otherwise, the patient is well.

**DISCUSSION**

Toxocariasis is a worldwide distributed helminthozoosnosis caused by infestation with *T. canis* and *T. cati*, the dog and cat roundworms, respectively. Definite hosts excrete unembryonated eggs with their feces. Eggs become embryonated, i.e., infectious, under appropriate conditions after an incubation period of 1–2 weeks in the environment. Thus, the disease is usually contracted by exposure to contaminated soil containing the infective eggs. The eggs hatch after uptake in the small intestine and release immature larvae. These penetrate the intestinal wall and are carried by the bloodstream to the lungs. Here, the larvae enter the alveoli, migrate up the Airways, and are swallowed again. The larvae then develop into adult parasites in the intestines, where the female worms become capable of producing eggs after sexual mating. Immunologic control by the host is mainly based on activation of eosinophil leukocytes by CD4+ T cells by interleukin-5. In an effort to contain their spread, eosinophils form eosinophil granulomas around the parasites and produce IgE. Migration of larvae can nevertheless continue for long periods.

A broad spectrum of species other than the definite hosts can also become infested: invertebrates such as earthworms, snails, and flies, birds, and most mammals including humans. In these species, however, the larvae are unable to complete their replication cycle. After uptake and penetration of the intestinal wall, they are carried by blood vessels to the liver. Dissemination then occurs through blood or lymph to a variety of tissues, including lungs, heart, muscles, eyes, and the central nervous system. The larvae migrate through the tissues, thus the term visceral larva migrans.

Consumption of raw or undercooked meat and giblets of these so-called paratenic animals may also result in infestation with *Toxocara larvae*. A number of cases of human toxocariasis were attributed to this mode of transmission. Raw liver seems to be particularly infective in this setting. This assumption is based on several observations. In most cases reported after consumption of paratenic animals, raw liver was the suspected source of infestation. Liver is usually one of the most heavily affected organs in toxocariasis because this organ drains portal venous blood containing large amounts of larvae after oral uptake. In a recent animal model using chickens, up to 94% of total larvae recovered were found in the liver. In several studies examining the seroprevalence of toxocariasis, the habit of eating raw liver was associated with significantly higher seroprevalence rates. This was demonstrated for such different populations as Korean citizens, French veterinary students, mountain aborigines in Taiwan, and Korean patients with eosinophilia of unknown origin. These observations led to more detailed examinations using animal models. In several studies, the infectivity of raw giblets, including raw chicken liver, was demonstrated. Taaira and others concluded that eating raw chicken, especially the liver, poses a potential risk for transmission of toxocariasis to humans.

In our case, infestation was also most likely caused by the consumption of raw liver. None of the known risk factors, such as ingestion of soil, poor personal hygiene, or close contacts with young dogs or cats, were apparent. Soon after consumption of the raw duck liver, typical symptoms such as an acute gastroenteritis and low-grade fever occurred. The husband, who had eaten only the baked duck, also tested positive for antibodies to *T. canis*. However, he did not show any symptoms of disease.

We conclude that infestation with *Toxocara* species through paratenic animals is most likely a pervasive and vola-
tile mode of transmission to humans, especially to adults. The small number of sporadic reports may not adequately represent the prevalence of such transmission. There is a broad spectrum of potential vectors, most common among them pigs, cows, and poultry. Consumption of the raw liver of these animals seems to be particularly infective. This appears especially relevant in societies that frequently consume raw liver, e.g., persons in the Mediterranean or eastern Asia. In Europe, however, such an incident is rare. Our case illustrates the severity of the potential consequences.

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Authors’ addresses: Bodo Hoffmeister, Sven Glaeser, Holger Flick, Norbert Suttrop, and Frank Bergmann, Department of Infectious Diseases and Pulmonology, Charité University Hospital, Augustenburger Platz 1, 13353 Berlin, Germany, E-mails: b.hoffmeister-infekt@charite.de, sven.glaeser@charite.de, holger.flick@charite.de, norbert.suttrop@charite.de, and frank.bergmann@charite.de. Sebastian Porschlegel, Schlosspark-Klinik, Heubnerweg 2, 14059 Berlin, Germany, E-mail: Sebastian.Porschlegel@schlosspark-klinik.de.

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