CASE REPORTS: A FAMILIAL CASE OF VISCERAL LARVA MIGRANS AFTER INGESTION OF RAW CHICKEN LIVERS: APPEARANCE OF SPECIFIC ANTIBODY IN BRONCHOALVEOLAR LAVAGE FLUID OF THE PATIENTS

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Abstract. We report a familial case of visceral larva migrans (VLM) caused by *Toxocara canis* larvae. Patient 1 was a 45-year-old man who presented to our university hospital complaining of mild fever, general fatigue, and headache. Patient 2 was a 71-year-old man and was the father of Patient 1; he presented complaining of cough and hyper-viscous white sputum. Laboratory data from both patients showed extensive eosinophilia, their chest X-ray findings revealed multiple pulmonary infiltrates, and their bronchoalveolar lavage fluid (BALF) showed an elevated eosinophil count. The diagnosis of VLM was made based on a positive result in a serological test using *T. canis* larval excretory–secretory both in the serum and BALF. *T. canis* larvae were identified in meat that was prepared from chicken taken from the same source as that ingested. This is the first report to identify antibodies in BALF in patients with VLM.

INTRODUCTION

Tissue invasion to the liver by *Toxocara* larvae was first described by Beaver and others. Since then, the migrating larvae of *Toxocara canis* have been found in various organs, commonly in the liver. Pulmonary involvement, which is characterized by the infiltration of eosinophils, is often observed in clinical cases. The diagnosis of toxocariasis is usually based on serological tests, because it is very difficult to detect a larval segment histologically in a tissue specimen. Aragane and others showed a *Toxocara* larva directly from a patient’s skin in a case that showed severe pulmonary inflammation and small nodules in the lung. To our knowledge, this is the first study to identify *T. canis* larvae in contaminated meat eaten by a patient and relate this with a positive antibody test result from an analysis of his bronchoalveolar lavage fluid (BALF). Here, we report a familial case of visceral larva migrans (VLM) caused by *T. canis* larvae caused by the southern Japanese custom of eating raw chicken livers without a past history of pica. We also suggest the usefulness of antibody detection in BALF in patients with pulmonary invasion by these larvae.

CASE 1

The patient is a 45-year-old man whose father raised a dozen floor-reared chickens for his personal consumption. The patient says that they also bred a few dogs around his garden, where they often met the chickens. He visited a neighborhood medical practitioner on May 18, 2004, complaining of general fatigue, low-grade fever, posterior headache, and neurologic abnormality of the front of the right arm of 3-week duration. Medical examination of the peripheral blood showed leucocytosis (15,000/µL) and eosinophilia (36%). His chest radiograph showed multiple nodules in both lung fields, and computed tomography (CT) scans showed slight pleural effusion in the right lung field (Figure 1A). The laboratory findings did not respond to intravenous steroids (hydrocortisone sodium succinate, 200 mg/d for the first 2 days and then 100 mg/d for 3 days) and intravenous antibiotics (clindamycin). The abnormal chest shadows gradually disappeared, but the peripheral eosinophil count increased to 51%. He was admitted to our university hospital on June 3 for further examination, and further tests revealed elevated concentrations of non-specific immunoglobulin E (22,000 IU/mL) and abnormal liver findings by abdominal ultrasonography. A detailed interview revealed that he had eaten raw chicken liver several weeks earlier, suggesting some sort of zoonotic parasite infection. A serological test using a dot-enzyme-linked immunosorbent assay (ELISA) was weakly positive for the *Ascaris suum* adult antigen and negative for the *T. canis* adult antigen. His BALF revealed an increasing number of eosinophils (11%), but a transbronchial lung biopsy (TBLB) could not identify the origin of the pulmonary multiple infiltrations. Finally, the diagnosis of VLM caused by *T. canis* larvae was made based on the positive results of a rapid diagnostic test for toxocariasis, ToxocaraCHEK (E. Y. Laboritories Inc., San Mateo, CA), from both his serum sample and BALF. After the initial positive result, we carried out further serological tests. Using the larval excretory–secretory (ES) antigen and a plate ELISA, we obtained a strongly positive result for the antigen, but not for the larval ES of *A. suum*. In addition, Ouchterlony double diffusion test showed several strong precipitin bands against the larval ES antigen of *T. canis* (Figure 2A). Moreover, from a liver taken from a chicken from the same breeding group as that of the liver ingested by the patient, we were able to recover 202 larvae that closely resembled *Toxocara* sp. (Figure 3). Furthermore, histologic examination of liver specimens obtained from the patient by ultrasonographic-guided biopsies revealed multiple focal necrosis with extensive eosinophilic infiltrations (Figure 4). After treatment with diethylcarbamazine (300 mg/d for 12 days), his symptoms gradually improved, and his blood eosinophil count, plasma aspartate
aminotransferase, alkaline phosphatase, and γ-gamma-glutamyl transpeptidase levels reverted to normal within 2 weeks of cessation of treatment; however, his non-specific IgE level remained high.

CASE 2

A 71-year-old man, who was the father of Patient 1 and an avid hunter of wild boars using dogs, ingested raw chicken liver derived from his poultry and boar farm. His son also ate a piece of the same liver. His chief complaints were cough and increasingly hyper-viscous white sputum. He visited our clinic on May 17, 2005, and examination of the peripheral blood showed hyper-leucocytosis (25,900/μL) with eosinophilia (63%). His chest radiograph showed an infiltration shadow in the right middle lung field, and CT scans showed multiple infiltrations in both lung fields (Figure 1B). No abnormalities were found in the liver by abdominal ultrasonography on the day of admission. His hyper-eosinophilia responded well to oral corticosteroid therapy (prednisolone 40 mg on the first day, 60 mg/d for 3 days, 30 mg/d for 10 days, 20 mg/d for 8 days), and his platelet count increased. His abnormal chest shadows gradually disappeared, and bronchoscopy was performed on the day after cessation of prednisolone treatment. An analysis of his BALF revealed an increase in eosinophils (30%), and a dot-ELISA against the A. suum adult antigen

![Figure 1](image1.png)

**Figure 1.** A, Chest CT scan of Patient 1 showing multiple nodules in both lung fields. B, Chest CT scan of Patient 2 showing an infiltration shadow in the right middle lung field.

![Figure 2](image2.png)

**Figure 2.** A, Ouchterlony double diffusion test of Patient 1 showing a strong precipitin band against the larval ES antigen of *T. canis*. B, Ouchterlony double diffusion test of Patient 2 showing a strong precipitin band against the larval ES antigen of *T. canis*.

![Figure 3](image3.png)

**Figure 3.** *Toxocara* sp. larvae closely resembling those found in the patients were recovered from a chicken liver from the same group as that of the liver eaten by the patients.

![Figure 4](image4.png)

**Figure 4.** Liver specimens obtained with ultrasonographic-guided biopsies revealing multiple focal necrosis with extensive eosinophilic infiltrations (H&E stain ×50).
was strongly positive but was only weakly positive for *T. canis* adult antigens. We abandoned TBLB because of his tendency to bleed from the bronchial mucosa when the fiberscope made contact. However, we suspected VLM because this patient had the same dietary history and similar symptoms as his son (Case 1). Therefore, we performed further serological tests. An Ouchterlony test against ES products of *T. canis* larvae was strongly positive (Figure 2B), whereas a Toxocara CHEK and plate ELISA of the serum sample and BALF were positive for larval ES antigens. He was started on albendazole treatment (600 mg/d for 35 days). By the end of this treatment, the cough and sputum production had disappeared, the serum eosinophil count had decreased, and the platelet count had increased. However, the non-specific IgE level remained high as in Case 1. Unfortunately, we stopped the drug treatment because of signs of impaired liver function in the form of elevated plasma alanine aminotransferase of up to 330 IU/L. Thereafter, this patient was lost to follow-up. No abnormality was found in the optic fundi or head on CT in either of these two cases.

**DISCUSSION**

Parasitic infections remain relatively common even in developed countries, caused in part by an increasing number of overseas travelers and pet owners. In Japan, the diversification of dietary habits is thought to be another reason for the persistence of food-borne parasitic diseases. It had been believed that toxocariasis was a disease of children who accidentally ingested infective eggs of *T. canis*. In Japan, however, adult cases of toxocariasis have been reported after the ingestion of meat from quails, cows, and chickens, suggesting that toxocariasis in Japan must be considered a food-borne parasitic disease of adulthood. After invasion of the host, these larvae hatch in the digestive track and migrate to the liver through the host portal system. Thereafter, some larvae continue to migrate to the lungs and systemic organs through the circulation, resulting in the symptoms characteristic of the clinical disease. Recently, several cases of VLM by *A. suum* were reported in the southern part of Japan. The origin of such infections involves swine husbandry and the inappropriate disposal of waste materials. In such cases, the antibodies against larval ES of *A. suum* should be detectable, although this was not possible with our patients.

Glickman and others reported that hunting or living in a household with hunting dogs was a high risk factor for VLM in people, especially those with signs of allergies. In our cases, there was no history of allergies previously, although they have had such a risk factor. It is obvious that VLM symptoms were brought on by the ingestion of raw chicken livers because the event was the first onset. Therefore, we must consider the way of infection not only from hunting dogs directly but also from the raw meat of domestic fowl contaminated with *T. canis* eggs. Incidentally, this is the first report that *Toxocara* sp. was proven to be recovered from raw materials of a meal directly.

While a definitive diagnosis in parasitic diseases is best made after the detection of worms or eggs from patients, the direct detection of *Toxocara* larvae is quite rare. Generally, food-borne toxocariasis can be diagnosed through a combination of a patient’s admission with a history of ingesting raw meat, laboratory findings such as hypereosinophilic syndrome, and the presence of specific antibodies against larval ES antigens of *T. canis* in the serum. Our findings further suggest that specific antibodies in BALF should be measured in patients with respiratory symptoms.

We found that the ToxocaraCHEK, a rapid diagnostic test kit for toxocariasis, was inexpensive, easy to use, and accurate in detecting antibodies to larval ES products of *T. canis*. This test kit was known to be useful for the detection of antibodies in vitreous fluid, but we successfully applied it to the identification of antibodies in BALF in these cases. We believe that this is the first report to show the specific antibodies to the larval ES antigen of *T. canis* in BALF, although previous investigators showed an elevated eosinophil count in BALF. These observations suggest that clinicians should consider food-borne toxocariasis in cases of severe respiratory disease with eosinophilia and a patient history of raw meat ingestion.

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