

Retrospective Study

Clinical characteristics and progression of liver abscess caused by toxocara

Kyung Ho Ha, Jung Eun Song, Byung Seok Kim, Chang Hyeong Lee

Kyung Ho Ha, Department of Internal Medicine, Daegu Medical Center, Daegu 41845, South Korea

Jung Eun Song, Byung Seok Kim, Chang Hyeong Lee, Department of Internal Medicine, Division of Gastroenterology and Hepatology, Daegu Catholic University, Daegu 42472, South Korea

Author contributions: Ha KH searched literature, drafted the manuscript, incorporated corrections by coauthors into final manuscript and organized details for submission of manuscript; Song JE and Kim BS supervised the manuscript; Lee CH contributed to writing the manuscript, reviews and corrections, final approval and submissions.

Institutional review board statement: The study was reviewed and approved by the Institutional Review Board of Daegu Catholic University.

Informed consent statement: Because of retrospective and anonymous character of this study, the need for informed consent was waived by the institutional review board.

Conflict-of-interest statement: The authors have no conflict of interest related to this publication.

Data sharing statement: No additional data are available.

Open-Access: This article is an open-access article which was selected by an in-house editor and fully peer-reviewed by external reviewers. It is distributed in accordance with the Creative Commons Attribution Non Commercial (CC BY-NC 4.0) license, which permits others to distribute, remix, adapt, build upon this work non-commercially, and license their derivative works on different terms, provided the original work is properly cited and the use is non-commercial. See: <http://creativecommons.org/licenses/by-nc/4.0/>

Manuscript source: Unsolicited manuscript

Correspondence to: Chang Hyeong Lee, MD, Professor, Department of Internal Medicine, Division of Gastroenterology and Hepatology, Daegu Catholic University, 33, Duryugongwon-ro 17-gil, Nam-gu, Daegu 42472, South Korea. chlee1@cu.ac.kr

Telephone: +82-53-6503067
 Fax: +82-53-6284050

Received: February 15, 2016
 Peer-review started: February 16, 2016
 First decision: March 24, 2016
 Revised: April 7, 2016
 Accepted: June 1, 2016
 Article in press: June 3, 2016
 Published online: June 28, 2016

Abstract

AIM: To evaluate the clinical characteristics and progression of liver abscess caused by toxocara.

METHODS: We retrospectively reviewed the medical records of patients with serum IgG antibody to *Toxocara canis* and liver abscess diagnosed using abdominal computed tomography between February 2010 and February 2015. Among 84 patients exhibiting serum IgG antibody to *Toxocara canis*, 34 patients were diagnosed with liver abscess and treated with albendazole. A follow-up period of 1 year was conducted.

RESULTS: Mean patient age was 53 (34-79) years, with 26 (76.5%) patients being male. Twenty-one (61.7%) patients were moderate or heavy drinkers, 23 (67.6%) patients had a history of eating raw meat or liver and 6 (17.6%) patients owned pet dogs or cats. Main patient symptoms consisted of right upper quadrant pain, fever, and fatigue; 18 (52.9%) patients, however, presented with no symptoms. Lung involvement was detected in 44 (11.7%) patients. The eosinophil count increased in 29 (85.3%) patients at initial diagnosis, and decreased in most patients after albendazole treatment. The initial serum IgE level increased in 25 (73.5%) patients, but exhibited various response levels after albendazole treatment. Liver abscess formation improved in all patients.

CONCLUSION: The liver abscess was improved with albendazole treatment.

Key words: Toxocariasis; Liver abscess; Eosinophilia

© **The Author(s) 2016.** Published by Baishideng Publishing Group Inc. All rights reserved.

Core tip: This is a retrospective study to evaluate the clinical characteristics and progression of liver abscess caused by toxocara. Eating uncooked food was a more common route of infection than contact with pet animals. Alcohol consumption, sex (male), and ingestion of raw meat or liver were considered to be significant risk factors for toxocariasis. Patients can present with no specific symptoms, eosinophilia, and/or increased levels of serum IgE. Liver abscess caused by toxocara has characteristic radiologic findings. Even if a few patients experience relapse or migration of abscess posttreatment, a good prognosis exists for the overall clinical course.

Ha KH, Song JE, Kim BS, Lee CH. Clinical characteristics and progression of liver abscess caused by toxocara. *World J Hepatol* 2016; 8(18): 757-761 Available from: URL: <http://www.wjgnet.com/1948-5182/full/v8/i18/757.htm> DOI: <http://dx.doi.org/10.4254/wjch.v8.i18.757>

INTRODUCTION

Toxocariasis is a parasitic infection caused by *Toxocara canis* or *Toxocara cati*. It is known as a main cause of eosinophilia^[1]. Clinical manifestations of toxocariasis range from asymptomatic infection to involvement of various organs. Visceral larva migrans (VLM) means toxocara infection associated with various internal organs of the body^[2]. Liver abscess represent one type of VLM, which differs from pyogenic liver abscess in displaying specific histologic and radiologic findings^[3,4]. Hepatic VLM or liver abscess caused by toxocara can occasionally be detected as an abnormal finding at ultrasonography screening and therefore be misdiagnosed as a malignancy in patients with chronic liver disease or a history of other cancer(s)^[5]. A thorough understanding of the clinical characteristics and progression of hepatic VLM or liver abscess caused by toxocara is necessary in order to determine potential factors that may help improve diagnosis, as well as avoid unnecessary testing and improper disease treatment.

MATERIALS AND METHODS

We retrospectively reviewed the medical records of patients with serum IgG antibody to *Toxocara canis* and liver abscess diagnosed by abdominal computed tomography (CT) at Daegu Catholic University Hospital between February 23, 2010 and February 24, 2015. We investigated patients about a history of moderate

or heavy alcohol consumption, raw meat or cow's liver ingestion, and owning pet dogs or cats. We obtained liver transaminase levels, peripheral blood eosinophil counts, serum IgE levels, hepatitis B surface antigen and antibody and anti-hepatitis C virus antibody results. In addition, we obtained any history of underlying disease and other organ(s) involvement. Eosinophilia was defined as an absolute peripheral blood eosinophil count $\geq 500/\mu\text{L}$. Elevated serum levels of IgE were defined as IgE levels ≥ 100 IU/mL. We treated patients with liver abscess with 400 mg orally twice daily for 5 d. The follow-up protocol consisted of obtaining repeat eosinophil counts and serum IgE levels, as well as performing abdominal CT scans at various intervals for 1 year.

RESULTS

Among a total of 84 patients exhibiting serum IgG antibody to *Toxocara canis*, 34 patients were diagnosed with liver abscess. Mean patient age was 53 years, with serum IgG antibody to *Toxocara canis* being three times more prevalent in men than in women (Table 1). Twenty-three (67.6%) patients had a history of eating raw meat or liver and 6 (17.6%) patients owned pet dogs or cats (Table 1). Four patients had no specific history of eating uncooked food or owning pet animals. Main patient symptoms consisted of right upper quadrant pain, fever, and fatigue. Eighteen (52.9%) patients were asymptomatic (Table 1). Five patients revealed involvement of other organs including the lung, a leg muscle, and the brain in addition to liver involvement (Table 1). One of four patients with lung involvement demonstrated concomitant brain involvement. Aspartate transaminase and alanine transaminase levels were normal in all patients except one who had alcoholic hepatitis. Twenty-nine (85.3%) patients initially presented with eosinophilia. Among these 29 patients, 17 had mild eosinophilia, 7 had moderate eosinophilia, and 5 had severe eosinophilia (Table 1). Twenty-five of 26 patients who had repeat serum IgE levels had initially increased IgE levels (Table 1). The remaining patient demonstrated an upper normal serum IgE level of 99 IU/mL. Liver abscess on dynamic CT included multiple lesions in 19 patients and a single lesion in 15 patients (Table 1). The lesions were seen as ill-defined, low-attenuating, oval nodules. They were faintly seen on arterial and equilibrium phase images and best seen on the portal venous phase. All of these 34 patients were treated with albendazole. After treatment, the eosinophil count was normal in 16 patients, decreased in 8, and remained the same in one (Table 2). The eosinophilic response pattern was divided into two groups: Continuously decreasing (15 patients, 62.5%) and fluctuating (9 patients, 37.5%). We were unable to evaluate the eosinophilic response in 9 patients. Among those patients, 5 had normal eosinophil counts at initial diagnosis and 4 did not participate in the follow-up protocol posttreatment. Fifteen of 24 patients who showed an eosinophilic response did so within 1 mo posttreatment. Six months after treatment, the serum

Table 1 Baseline characteristics of patients with liver abscess caused by toxocara

Patients, <i>n</i>	34
Mean age, yr	53
Male, <i>n</i> (%)	26 (76.5)
Underlying disease, <i>n</i> (%)	
Hypertension	9 (26.5)
Diabetes	4 (11.8)
Tuberculosis	3 (8.8)
Liver cirrhosis	2 (5.9)
Chronic viral hepatitis	2 (5.9)
Cancer history	2 (5.9)
None	9 (26.5)
Alcohol drinking, <i>n</i> (%)	
Heavy drinking	14 (41.2)
Moderate drinking	7 (20.6)
No drinking	9 (26.5)
Unknown	4 (11.8)
Transmission, <i>n</i> (%)	
Eating raw meat or liver	23 (67.6)
Keeping pet dogs or cats	6 (17.6)
No specific history	4 (11.8)
Unknown	7 (20.6)
Symptoms, <i>n</i> (%)	
Asymptomatic	18 (52.9)
RUQ pain	6 (17.6)
Fever	4 (11.8)
Fatigue	4 (11.8)
Anorexia	2 (5.9)
Cough	2 (5.9)
Weakness of legs	2 (5.9)
Involvement of other organs, <i>n</i> (%)	
Lung	4 (11.8)
Muscle of legs	1 (2.9)
CNS	1 (2.9)
Mean AST/ALT, IU/L	31/31
Eosinophilia, <i>n</i> (%)	
Normal (< 500/ μ L)	5 (14.7)
Mild (500-1500/ μ L)	17 (50.0)
Moderate (1500-5000/ μ L)	7 (20.6)
Severe (> 5000/ μ L)	5 (14.7)
Serum IgE, <i>n</i> (%)	
Normal (< 100 IU/mL)	1 (2.9)
Mild elevated (100-500 IU/mL)	10 (29.4)
Severe elevated (> 500 IU/mL)	15 (44.1)
Unknown	8 (23.5)
Liver abscess, <i>n</i> (%)	
Single	15 (44.1)
Multiple	19 (55.9)

Liver abscess caused by toxocara was related to sex (male), alcohol drinking, eating raw meat or liver. Laboratory characteristics showed normal liver enzymes, peripheral blood eosinophilia, and elevated level of serum IgE. RUQ: Right upper quadrant; CNS: Central nervous system; AST: Alanine aminotransferase; ALT: Aspartate aminotransferase.

IgE level increased in 7 patients, decreased in 8, and remained the same in 3 (Table 2). We were unable to evaluate the serum IgE response in 16 patients. Among these patients, 8 patients did not undergo check serum IgE level tests initially and the other 8 patients did not participate in the follow-up protocol. A follow-up CT was performed for 22 patients. Among these patients, 15 demonstrated disappearance of liver abscess within 3 mo and 21 within 6 mo. Relapse or migration of liver abscess was observed in 3 patients.

Table 2 Therapeutic response after treatment with albendazole *n* (%)

Eosinophilia (<i>n</i> = 25)	
Normalized	16 (64)
Decreased	8 (32)
No change	1 (4)
Serum IgE (<i>n</i> = 18)	
Increased	7 (38.9)
Decreased	8 (44.4)
No change	3 (16.7)
Liver abscess (<i>n</i> = 22)	
Improved	22 (100)
Not improved	0

Most eosinophil counts were normalized or decreased and all of the abscesses were improved on computed tomography after 1 year, but the levels of serum IgE showed variable response after 6 mo.

DISCUSSION

Toxocariasis is a worldwide disease. The overall seroprevalence of toxocariasis has been reported as 13.9% in United States^[6], 2%-5% and 14%-37% respectively in the urban and rural areas of France^[7], 18% in China, 20% in Malaysia, 68% in Indonesia, and 81% in Nepal^[8]. In South Korea the seroprevalence has been reported as 5% in Gangwon-do^[9], 6% in Seoul, and 11% in Gyeongsangnam-do^[10]. The seroprevalence of toxocariasis in patients with eosinophilia has been reported as 64.9%-86.7% in Seoul^[8,11], 50.5% in Chungcheongnam-do^[12], and 62% in Pohang^[13]. These reports confirm that toxocariasis is known to be a main cause of eosinophilia. Toxocara infection is caused by ingestion of embryonated eggs from the soil and pet animals or by ingestion of encapsulated larva while eating uncooked paratenic hosts^[14]. In this study, eating uncooked food was a more common route of infection than contact with pet animals (67.6% vs 17.6%). Choi *et al*^[8] suggested that ingestion of raw cow liver was related to an increased risk of toxocariasis, but not ingestion of raw meat or animal blood and owning dogs. Based on the results of our epidemiologic study, demonstrating that men are three times more susceptible to toxocariasis than women, and revealing that approximately 60% of the patients consumed alcohol, we consider sex (male) and alcohol consumption as risk factors for toxocariasis.

The most commonly utilized serologic test for toxocariasis is the detection of the serum IgG antibody to the toxocara excretory/secretory antigen (TES Ag) with a toxocara ELISA kit (Bordier Affinity Products, Crissier, Switzerland)^[15]. Eosinophilia, elevated serum IgE levels or increased eosinophil cationic protein is helpful for the diagnosis of active toxocariasis^[16-18]. Although the toxocara ELISA test possesses high sensitivity and specificity^[15], the test cannot differentiate present from past infections^[16] and may produce cross-reactivity with other parasites such as *Clonorchis sinensis*, *Sparganum*, *Fasciola hepatica*, and *Paragonimus westermani*^[2]. Infected larva can penetrate the intestinal wall through vessels and invade various organs such as the liver, lung, muscle,

eye, heart and central nervous system, etc^[19,20]. We investigated other organ(s) involvement in 84 patients who had the IgG antibody for *Toxocara canis*, and found the organs involved, displayed in order of frequency, were the liver (40.5%), lung (27.4%), eye (8.3%), skin (3.6%), muscle (1.2%), and brain (1.2%). Lung involvement occurred in 11.8% of patients with liver abscess (Table 1). This occurrence, therefore, creates the necessity of assessing the possibility of lung involvement in patients with liver abscess caused by toxocariasis. TES Ag secreted from the epicuticle of the moving larva causes an immune reaction, which produces increased serum IgE and eosinophilia^[21]. Liver abscess, histologically described as eosinophilic abscess or granuloma, results from eosinophilic inflammation which develops when larva remain in the liver^[22].

Approximately 50% of patients were asymptomatic. A small number of patients had right upper quadrant pain, fever, and fatigue (Table 1). It remains impossible to rule out liver abscess in patients with toxocariasis using only symptom information. In contrast to patients with hepatic visceral larva migrans or liver abscess caused by toxocara, approximately 90% of patients with pyogenic liver abscess have fever and approximately 70% have abdominal pain^[23]. The possibility of abscess caused by toxocara must be considered if liver abscess is inadvertently detected upon abdominal ultrasonography during routine medical exams. CT findings of liver abscess caused by toxocara usually include lesions that measure approximately 1-1.5 cm in diameter; possess an oval shape, obscure margin, multiplicity, and hypodensity^[3]. In contrast to hepatocellular carcinoma, CT findings for liver abscess include lesions that are regular, not round, and striking at portal venous phase^[24]. We can confirm the CT findings cited in this study; however, one finding that differs from the previous study^[24] is that data from this study reveal that a relatively high number of patients with single abscess existed.

Toxocariasis is a self-limiting disease; therefore, patients with mild symptoms do not necessarily require medication^[2]. However, if patients have moderate or severe symptoms due to visceral larva migrans, they should be treated with albendazole^[24]. Previous literature has recommended treating liver abscess regardless of symptom status because, compared with the control group, the albendazole group demonstrated accelerated liver abscess healing^[5]. Twenty-four of 25 patients who presented with eosinophilia and had been treated with albendazole displayed decreased eosinophil counts. Fifteen of these patients (62.5%) had checked their eosinophil count within 1 mo posttreatment. All patients experienced decreased eosinophil counts. Previous literature has reported an eosinophilic response occurred 1 mo posttreatment^[16]; therefore, we concluded that the eosinophilic response could be evaluated 1 mo posttreatment. In addition, if the eosinophilic count initially decreases, but continually increases during the posttreatment period, relapse or migration of lesions should be considered. Transient eosinophil count fluctuations, which can occur among eosinophilic response patients

as observed in this study, must also be considered. Abdominal CT and repeat eosinophil counts at follow-up can help distinguish relapse or migration from eosinophilic fluctuation. Repeat serum IgE levels at follow-up provides an inadequate evaluation measurement of treatment response because serum IgE responds unpredictably^[16]. CT follow-up was performed within 3 mo for 68.2% of patients and liver abscess disappeared in all of them. We therefore concluded that CT scan results could be evaluated 3 mo posttreatment. We encountered relapse or migration of lesions in three patients at 4, 6 and 8 mo posttreatment. Eosinophilia developed in only one of these patients, while the other two experienced continuously decreasing eosinophil levels despite relapse or migration of lesions. Two of these three patients were retreated with albendazole, while the other was only observed. Subsequently, all lesions of all three patients disappeared. If this phenomenon is observed posttreatment, the possibility of reinfection also needs to be considered. No evidence exists confirming that patients who are experiencing relapse or migration of liver abscess, regardless of clinical symptoms or eosinophilia, should be retreated with albendazole. It is reasonable, however, to retreat relapsed or migrated lesions with albendazole because albendazole is inexpensive, easily available over the counter and has no significant side effects. Existence of toxocara-specific IgG antibody can persist for years after the disappearance of liver abscess. One study reported that the mean duration of IgG antibody existence in the body was 2.7 years^[25]. IgG antibody detection was conducted at 3, 9, 18, 24 mo and 5 years each in five patients from this study. All patients persistently displayed IgG antibody during the follow-up period, therefore, excluding detection of serum IgG antibody testing from the follow-up protocol^[16].

The study has limitations as a retrospective study, so our recommendations about the evaluation measurement of treatment response are based on the literature data and not on the results of this study.

Liver abscess caused by toxocara is a disease resulting from the ingestion of uncooked food, which causes an immune reaction in the liver. Patients can present with no specific symptoms, eosinophilia, and/or increased levels of serum IgE. Toxocariasis has characteristic radiologic findings and may involve other organs such as the lung. Treatment of toxocariasis consists of taking albendazole for 5 d. After treatment, the eosinophil count starts to decrease within 1 mo and the abscess begins to disappear within 3 mo as displayed on CT scan. Complete disappearance of liver abscess can occur after 1 year. Even if a few patients experience relapse or migration of abscess posttreatment, a good prognosis exists for the overall clinical course of this disease.

COMMENTS

Background

Toxocariasis is a parasitic infection caused by *Toxocara canis* or *Toxocara cati*. It is known as a main cause of eosinophilia. Clinical manifestations of

toxocarasis range from asymptomatic infection to involvement of various organs. Liver abscess caused by toxocara can occasionally be detected as an abnormal finding at ultrasonography screening and therefore be misdiagnosed as a malignancy in patients with chronic liver disease or a history of other cancer(s). The authors evaluated the clinical characteristics and progression of liver abscess caused by toxocara.

Research frontiers

This study contributes to determining potential factors that may help improve diagnosis of liver abscess caused by toxocara, as well as avoid unnecessary testing and improper treatment.

Innovations and breakthroughs

In this study, all patients (62.5%) who had checked their eosinophil count within 1 mo posttreatment experienced decreased eosinophil counts. And all patients (68.2%) who had checked computed tomography (CT) follow-up within 3 mo posttreatment experienced disappearance of liver abscess. Therefore, the authors concluded that the eosinophilic response could be evaluated 1 mo posttreatment and CT scan could be evaluated 3 mo posttreatment.

Applications

Human toxocarasis can clinically present as liver abscess. If a patient with a history of eating raw meat or liver presents peripheral eosinophilia and abnormal liver imaging, liver abscess caused by toxocara should be considered for diagnosis.

Terminology

Toxocarasis: An infection transmitted from animals to humans caused by the parasitic roundworms commonly found in the intestine of dogs (*Toxocara canis*) and cats (*Toxocara cati*).

Peer-review

Studies exploring toxocarasis in liver abscess have been infrequent. The author of this paper evaluated the clinical characteristics and progression of liver abscess caused by toxocara. This study is useful for diagnosing and monitoring the disease in the clinical practice.

REFERENCES

- 1 **Kwon NH**, Oh MJ, Lee SP, Lee BJ, Choi DC. The prevalence and diagnostic value of toxocarasis in unknown eosinophilia. *Ann Hematol* 2006; **85**: 233-238 [PMID: 16463154 DOI: 10.1016/j.jaci.2005.12.320]
- 2 **Pawlowski Z**. Toxocarasis in humans: clinical expression and treatment dilemma. *J Helminthol* 2001; **75**: 299-305 [PMID: 11818044 DOI: 10.1017/S0022149X01000464]
- 3 **Lim JH**. Toxocarasis of the liver: visceral larva migrans. *Abdom Imaging* 2008; **33**: 151-156 [PMID: 17924161 DOI: 10.1007/s00261-007-9325-y]
- 4 **Rahimian J**, Wilson T, Oram V, Holzman RS. Pyogenic liver abscess: recent trends in etiology and mortality. *Clin Infect Dis* 2004; **39**: 1654-1659 [PMID: 15578367 DOI: 10.1086/425616]
- 5 **Jang EY**, Choi MS, Gwak GY, Koh KC, Paik SW, Lee JH, Paik YH, Yoo BC. Enhanced resolution of eosinophilic liver abscess associated with toxocarasis by albendazole treatment. *Korean J Gastroenterol* 2015; **65**: 222-228 [PMID: 25896156 DOI: 10.4166/kjg.2015.65.4.222]
- 6 **Won KY**, Kruszon-Moran D, Schantz PM, Jones JL. National seroprevalence and risk factors for Zoonotic *Toxocara* spp. infection. *Am J Trop Med Hyg* 2008; **79**: 552-557 [PMID: 18840743]
- 7 **Magnaval JF**, Michault A, Calon N, Charlet JP. Epidemiology of human toxocarasis in La Réunion. *Trans R Soc Trop Med Hyg* 1994; **88**: 531-533 [PMID: 7992328 DOI: 10.1016/0035-9203(94)90148-1]
- 8 **Choi D**, Lim JH, Choi DC, Paik SW, Kim SH, Huh S. Toxocarasis and ingestion of raw cow liver in patients with eosinophilia. *Korean J Parasitol* 2008; **46**: 139-143 [PMID: 18830052 DOI: 10.3347/kjp.2008.46.3.139]
- 9 **Park HY**, Lee SU, Huh S, Kong Y, Magnaval JF. A seroepidemiological survey for toxocarasis in apparently healthy residents in Gangwon-do, Korea. *Korean J Parasitol* 2002; **40**: 113-117 [PMID: 12325440 DOI: 10.3347/kjp.2002.40.3.113]
- 10 **Kim HS**, Jin Y, Choi MH, Kim JH, Lee YH, Yoon CH, Hwang EH, Kang H, Ahn SY, Kim GJ, Hong ST. Significance of serum antibody test for toxocarasis in healthy healthcare examinees with eosinophilia in Seoul and Gyeongsangnam-do, Korea. *J Korean Med Sci* 2014; **29**: 1618-1625 [PMID: 25469060 DOI: 10.3346/jkms.2014.29.12.1618]
- 11 **Kim YH**, Huh S, Chung YB. Seroprevalence of toxocarasis among healthy people with eosinophilia. *Korean J Parasitol* 2008; **46**: 29-32 [PMID: 18344674 DOI: 10.3347/kjp.2008.46.1.29]
- 12 **Seo M**, Yoon SC. A seroepidemiological survey of toxocarasis among eosinophilia patients in Chungcheongnam-do. *Korean J Parasitol* 2012; **50**: 249-251 [PMID: 22949755 DOI: 10.3347/kjp.2012.50.3.249]
- 13 **Ryu BH**, Park JS, Jung YJ. Clinical and Serological Findings in Patients with Toxocarasis in the Pohang Region: The Features of Toxocarasis in Pohang. *Korean J Med* 2013; **84**: 203-210
- 14 **Morris PD**, Katerndahl DA. Human toxocarasis. Review with report of a probable case. *Postgrad Med* 1987; **81**: 263-267 [PMID: 3543902]
- 15 **Jacquier P**, Gottstein B, Stingelin Y, Eckert J. Immunodiagnosis of toxocarosis in humans: evaluation of a new enzyme-linked immunosorbent assay kit. *J Clin Microbiol* 1991; **29**: 1831-1835 [PMID: 1774303]
- 16 **Magnaval JF**, Glickman LT, Dorchie P, Morassin B. Highlights of human toxocarasis. *Korean J Parasitol* 2001; **39**: 1-11 [PMID: 11301585 DOI: 10.3347/kjp.2001.39.1.1]
- 17 **Fillaux J**, Magnaval JF. Laboratory diagnosis of human toxocarasis. *Vet Parasitol* 2013; **193**: 327-336 [PMID: 23318165 DOI: 10.1016/j.vetpar.2012.12.028]
- 18 **Magnaval JF**, Berry A, Fabre R, Morassin B. Eosinophil cationic protein as a possible marker of active human *Toxocara* infection. *Allergy* 2001; **56**: 1096-1099 [PMID: 11703226 DOI: 10.1034/j.1398-9995.2001.00284.x]
- 19 **Marx C**, Lin J, Masruha MR, Rodrigues MG, da Rocha AJ, Vilanova LC, Gabbai AA. Toxocarasis of the CNS simulating acute disseminated encephalomyelitis. *Neurology* 2007; **69**: 806-807 [PMID: 17709716 DOI: 10.1212/01.wnl.0000267664.53595.75]
- 20 **Enko K**, Tada T, Ohgo KO, Nagase S, Nakamura K, Ohta K, Ichiba S, Ujike Y, Nawa Y, Maruyama H, Ohe T, Kusano KF. Fulminant eosinophilic myocarditis associated with visceral larva migrans caused by *Toxocara canis* infection. *Circ J* 2009; **73**: 1344-1348 [PMID: 19122304 DOI: 10.1253/circj.CJ-08-0334]
- 21 **Lee S**. Pathophysiology of hypersensitivity in human by *Toxocara canis* larval infection. Chung-Ang University Doctoral Thesis, 2001
- 22 **Despommier D**. Toxocarasis: clinical aspects, epidemiology, medical ecology, and molecular aspects. *Clin Microbiol Rev* 2003; **16**: 265-272 [PMID: 12692098 DOI: 10.1128/CMR.16.2.265-272.2003]
- 23 **Chang S**, Lim JH, Choi D, Park CK, Kwon NH, Cho SY, Choi DC. Hepatic visceral larva migrans of *Toxocara canis*: CT and sonographic findings. *AJR Am J Roentgenol* 2006; **187**: W622-W629 [PMID: 17114516]
- 24 **Stürchler D**, Schubarth P, Gualzata M, Gottstein B, Oettli A. Thiabendazole vs. albendazole in treatment of toxocarasis: a clinical trial. *Ann Trop Med Parasitol* 1989; **83**: 473-478 [PMID: 2694978]
- 25 **Jeanneret JP**. Épidémiologie de la toxocarose dans la région jurassienne: Université de Neuchâtel, 1991

P- Reviewer: Akyuz U, Ferraioli G, He JY, Romero MR, Zielinski J
S- Editor: Qiu S **L- Editor:** A **E- Editor:** Li D





Published by **Baishideng Publishing Group Inc**

8226 Regency Drive, Pleasanton, CA 94588, USA

Telephone: +1-925-223-8242

Fax: +1-925-223-8243

E-mail: bpgoffice@wjgnet.com

Help Desk: <http://www.wjgnet.com/esps/helpdesk.aspx>

<http://www.wjgnet.com>

