**Rawal Medical Journal**

**An official publication of Pakistan Medical Association Rawalpindi Islamabad branch**

Established 1975

**Volume 37** **Number 2** **April – June** **2012**

**Original Article**

# Relationship between *Toxocara canis* infection and schizophrenia

Nagwa Mostafa El-Sayed, Khadiga Ahmed Ismail

Faculty of Medicine, Research Institute of Ophthalmology, Ain Shams, Egypt

## ABSTRACT

### Objective

To investigate the seropositivity rate for anti-*Toxocara* IgG antibodies in patients with schizophrenia and to assess its association with schizophrenia along with some risk factors for toxocariasis, eosinophilia and the presence of other intestinal parasites.

### Methods

Serological examination of 90 schizophrenic patients and 45 healthy controls was carried out by using commercial *Toxocara canis* IgG ELISA kit for the detection of anti-*Toxocara* antibodies. Also, eosinophils in peripheral blood and the presence of other intestinal parasites were investigated.

### Results

Seropositivity for *T. canis* was detected in 21 (23.3%) out of 90 schizophrenic patients and in one subject (2.2%) of 45 healthy controls ($p<0.01$). When seropositive and seronegative schizophrenic patients were compared with respect to sex, residence, owning dogs /cats,

history of geophagia, there was no significant differences between them ($p>0.05$). In contrast, when they were compared with respect to raw food intake and personal hygiene, the differences were statistically significant ($p<0.05$). Eosinophilia in peripheral blood was detected in 61.9% of seropositive schizophrenic patients and in 24.6% of seronegative schizophrenic patients ($p<0.01$). Of 21 schizophrenic patients with positive serology, 47.6% had at least one intestinal parasite comparing to 20.3% in patients with negative serology ($p< 0.05$).

**Conclusion:** There might be a causal relationship between toxocariasis and schizophrenia. Either *Toxocara* may be a possible etiologic agent of schizophrenia or the schizophrenic patients may be at high risk for *Toxocara* infection. (Rawal Med J 2012;37:155-160).

**Key Words**

*Toxocara*, schizophrenia, psychosis.

## INTRODUCTION

Human toxocariasis is an accidental parasitic disease due to infection by larval stages of *Toxocara canis* and *T. cati*, the common roundworms of dogs and cats, respectively.[1] The adult forms live in the upper intestinal tract of their definitive hosts and can produce about 200,000 eggs per day which are excreted into the environment with their feces. *Toxocara* eggs usually become infective within two to five weeks.[2] The embryonated eggs are accidentally ingested by humans, larvae hatch in the small intestine, penetrate the intestinal wall and migrate, via the blood stream, to the liver, lungs, muscles, eye and central nervous system and, during migration, cause an intense inflammatory response and eosinophilia.[1,2] The diagnosis of human toxocariasis is essentially based on immunological tests and the method of choice is the enzyme-linked immunosorbent assay (ELISA) using the excretory-secretory antigens of infective larvae of *T. canis* which is practical and inexpensive.[1]

Although most human infections are asymptomatic, two well-defined clinical syndromes are classically recognized: visceral larva migrans (a systemic disease caused by larval migration through major organs) and ocular larva migrans (a disease limited to the eyes and optic nerves). Two less-severe syndromes have recently been described, one mainly in children (covert toxocariasis) and the other mainly in adults (common toxocariasis).[3] The clinical spectrum of CNS toxocariasis is broad, causing various syndromes: eosinophilic meningoencephalitis and meningitis, meningomyelitis or meningoencephalomyelitis, encephalitis, extramedullary space-occupying lesion, brain vasculitis, seizures and probably behavior disorder.[4,5]

*Toxocara* infection may be increased in mentally retarded patients when adequate personal hygiene is not practiced.[6] Schizophrenic patients typically have poor hygiene and self-care skills. Kaplan et al found that *Toxocara* seroprevalence (45.9%) was much higher in schizophrenic patients than in the general population and schizophrenic state presented a high risk for *Toxocara* infection.[7] To ascertain a causal relationship between *T. canis* infection and schizophrenia, the present study aimed to investigate the seropositivity rate of anti-*Toxocara* IgG antibodies in schizophrenic patients. Also, the association of *Toxocara* seropositivity in those patients with some risk factors for toxocariasis, eosinophilia and the presence of other intestinal parasites was evaluated.

**PATIENTS AND METHODS**

A total of 90 patients with schizophrenia (49 women and 41 men) were selected from patients who admitted to Neuropsychiatry Department, Ain-Shams University Hospital, Giza, Egypt. Their age ranged from 20 to 60 years. Schizophrenia was diagnosed by two independent psychiatrists according to DSM IV diagnostic criteria. The patients either were suffering from a first episode of schizophrenia and had never been medicated with neuroleptics or had a wash-out period of treatment at least 4 months. All patients with known immunological

abnormalities, a co-morbid medical condition or a neurological disease were excluded from the study. Control group consisted of 45 healthy volunteers (24 women and 21 men), and had same age range. It was from health care workers and the relatives/ visitors of the patients. A baseline questionnaire was applied to all participants about some risk factors including sex, residence (urban or rural), ownership of dogs or/and cats and their presence at home, dietary habits (especially with regard to eating uncooked/ undercooked liver, meat), history of geophagia and personal hygiene (frequency of daily hand washes, nail-biting, washing of vegetables). This research had the approval of the Ethical Research Committee of the Faculty of Medicine, Ain-Shams University. All participants gave informed consent to take part in the study.

*Toxocara* IgG antibodies were determined by enzyme-linked immunosorbent assay (ELISA) (Ridascreen *Toxocara* IgG, R-Biopharm, Germany). The test was performed at the Laboratory of Parasitology Department, Research Institute of Ophthalmology, Giza, Egypt. Total leukocyte count was done on coulter counter T660 (Coulter Corp. Miami, Florida, USA) and leukocyte differential formula was obtained by stained-blood smear with Leishman stain. Leukocytosis was defined as cell count more than 10,000 cells/μL and the eosinophilia was defined as absolute eosinophils count more than 500 cells/μL and classified by percentage as normal (< 6%), mild (6-10%), moderate (11-15%), and severe (> 15%) eosinophilia.[8] Coproparasitological examination was realized by direct microscopic observation in fecal smears and by sedimentation technique to detect parasites that can generate cross-reactivity with *Toxocara* IgG ELISA test.

**Statistical analysis** was performed with SPSS v 10. Chi-square test ($\chi 2$) was used to determine statistically significant differences. $P<0.05$ was considered statistically significant and $P<0.01$ was considered statistically highly significant.

## RESULTS

Out of 90 patients with schizophrenia, significant levels of anti-*Toxocara* antibodies were detected in 21 (23.3%) samples, while only one subject (2.2%) had positive level of anti-*Toxocara* antibodies from healthy controls ($p<0.01$) (Table 1).

**Table 1. *Toxocara* seropositivity.**

| Patient group | *T. canis* seropositive Number (%) | *T. canis* seronegative Number (%) | Total Number (%) | P-value |
|---|---|---|---|---|
| Schizophrenic | 21 (23.3%) | 69 (76.7%) | 90 (100%) | 0. 0017* |
| Control | 1 (2.2%) | 44 (97.8%) | 45 (100%) | |

***** Highly significant difference ($p$ <0.01).

From 21 schizophrenic patients with a positive serology result, 12/21 (57.1%) had sample index values range between 1.1-2 and 9/21( 42.8%) had sample index values more than 2. The equivocal group when retested after 2 weeks was proved to be negative (Table 2).

**Table 2. Serofrequency and serointensity between schizophrenic patients and control group.**

| Patient group | Sample index values | | | Total |
|---|---|---|---|---|
| | < 0.9 (-ve) | 0.9 - 1.1 (equivocal) | > 1.1 (+ve) | |
| Schizophrenic group | 64 | 5 | 21 | 90 |
| Control group | 42 | 2 | 1 | 45 |

There was no significant difference between *Toxocara* seropositive and *Toxocara* seronegative schizophrenic patients in terms of sex, residence, owning dogs /cats and history of geophagia that increased the risk of *Toxocara* infection ($p$ > 0.05). When *Toxocara*

seropositive and seronegative schizophrenic patients were compared in terms of raw food intake and personal hygiene, there was statistically significant differences (Table 3).

**Table 3. Association between *Toxocara* seropositivity and some risk factors in schizophrenic patients.**

| Risk factors | Schizophrenia patients | | | |
|---|---|---|---|---|
| | **Total (90)** | ***T. canis* seropositive ( 21) Number (%)** | ***T. canis* seronegative (69) Number (%)** | ***P*- value** |
| **Gender:** | | | | |
| **Male** | 41 | 10 (24.4%) | 31 (75.6%) | 0.828 |
| **Female** | 49 | 11 (22.4%) | 38 (77.6%) | |
| **Residence** | | | | |
| **Urban** | 63 | 15 (23.8%) | 48 (76.2%) | 0.871 |
| **Rural** | 27 | 6 (22.2%) | 21 (77.8%) | |
| **Owning dogs /cats** | | | | |
| **Yes** | 11 | 4 (36.4%) | 7 (63.6%) | 0.275 |
| **No** | 79 | 17 (21.5%) | 62 (78.5%) | |
| **Raw food intake** | | | | |
| **Yes** | 59 | 18 (30.5%) | 41 (69.5%) | 0.026* |
| **No** | 31 | 3 (9.7%) | 28 (90.3%) | |
| **History of geophagia** | | | | |
| **Yes** | 2 | 1 (50%) | 1 (50%) | 0.367 |
| **No** | 88 | 20 (22.7%) | 68 (77.3%) | |
| **Personal hygiene** | | | | |
| **Good** | 24 | 2 (8.33%) | 22 (91.6%) | 0.042* |
| **Bad** | 66 | 19 (28.78%) | 47 (71.21%) | |

*Significant difference ($P<0.05$).

From the patients with positive serology, eosinophilia in peripheral blood was detected in 61.9% (38.1% had mild eosinophilia, 14.3% had moderate eosinophilia, and 9.5% had severe eosinophilia) compared to 24.6% found in the seronegative patients (18.8% had mild eosinophilia, and 5.8% had moderate eosinophilia) with a higher statistically significant difference (Table 4).

**Table 4. Association between *Toxocara* seropositivity and the presence of intestinal parasites and eosinophilia in schizophrenic patients.**

| Variable | Schizophrenia patients (n=90) | | |
|---|---|---|---|
| | ***T. canis* seropositive (21) Number (%)** | ***T. canis* seronegative (69) Number (%)** | **P- value** |
| **Eosinophilia** | **13 (61.9%)** | **17 (24.6%)** | **0.0015**** |
| Mild (6-10%) | 8 (38.1%) | 13 (18.8%) | |
| Moderate (11-15%) | 3 (14.3%) | 4 (5.8%) | |
| Severe (> 15%) | 2 ( 9.5%) | - | |
| **Intestinal parasites** | **10 (47.6%)** | **14 (20.3%)** | **0.0131*** |
| *B. hominis* | 7 ( 33.3% ) | 9 (13%) | |
| *G. lamblia* | 4 ( 19.0% ) | 5 (7.2%) | |
| *E. histolytica* | 3 ( 14.3% ) | 3 (4.3%) | |

**Highly significant difference ($p$ <0.01) *Significant difference ( $p$ <0.05).

The coproparasitological analysis revealed that 47.6% (10/21) of the schizophrenic patients with positive serology had at least one intestinal pathogen parasite [33.3% (7/21) had *Blastocystis hominis* cysts, 19 % (4/21) had *Giardia lamblia* cysts, and 14.3% (3/21) had *Entamoeba histolytica* / *E. dispar* cysts] compared to 20.3% (14/69) of patients with negative serology [13% (14/69) had *Blastocystis hominis* cysts, 7.2% (5/69) had *Giardia lamblia* cysts and 4.3% (3/69) had *Entamoeba histolytica* cysts]. The relationship between the presence of intestinal parasites and seropositivity for *Toxocara* was statistically significant ($p$ <0.05) ( Table 4).

## DISCUSSION

Schizophrenia is a serious neuropsychiatric disease of uncertain etiology. Numerous hypotheses have been proposed in an effort to explain schizophrenia. Some studies have rekindled interest in the possible role of infectious agents in schizophrenia.[9] It is well established that everyone produces marijuana-like compounds known as endocannabinoids which are homeostatic regulators of many body systems including the nervous system. Imbalances in the endocannabinoid system have been considered as .possible causes of various forms of mental illness and abnormal behavior.[10] Melamede et al suggested that a high

level of endocannabinoid activity in the brain is produced in response to parasitic brain infections, and may be crucial for the development of schizophrenia.[10] Against the idea that an infectious parasite might be a cause of schizophrenia, Yolken et al rsuggested that this idea would have met with a major conceptual barrier that schizophrenia is an actual disease of the brain.[11]

The present study found that *T. canis* seropositivity in schizophrenic patients (23.3%) is significantly higher than that in the control group (2.2%). This is in accordance with Kaplan et al who detected seropositivity for *T. canis* in 45.9% of schizophrenic patients and 2.0% of control subjects.[7] Moreover, Huminer et al identified *Toxocara* infection in 8.5% of institutionalized mental retarded adults in Israel.[6] Our observation of the high frequency of *Toxocara* infection in schizophrenic patients suggested that these cases may be considered under risk for *Toxocara* infection. It has been reported that the seroprevalence of toxocariasis varies between 2.4% in Denmark (3247 subjects aged $\leq$ 40 years)[12] to 92.8% in La Réunion island in Indian Ocean (387 subjects aged > 15 years),[13] depending on country, age and socio-cultural level. The seroprevalence of *Toxocara* in childhood is higher than in adults,[2] however, all the schizophrenic patients in this study were adults.

Our study explained the association of *Toxocara* seropositivity in schizophrenic patients with some risk factors for toxocariasis. Many authors suggested that male subjects might be at more risk to toxocariasis infection than female subjects, probably because of their closer contact with soil.[1,2,14,15] On the contrary, other reports showed that females were more frequently infected than males.[16] In this study, no significant differences were found between gender and positive serology, which is in agreement with several other authors.[17-19]

Regarding the residence, seroprevalence of *Toxocara* has been reported among persons living in rural areas than urban areas.[3,19] The association between residence and positive serology in our schizophrenic patients showed differences apparently not significant. About two third of

these patients were from the urban areas. This might be explained by the higher level of awareness and health seeking behavior in the urban areas, compared to rural areas. In Egypt, psychiatric illness is more of a stigma in rural communities.

It has been reported that *Toxocara* seroprevalence is high in those owning dogs and cats.[15,19-21] However, in our schizophrenic cases, the rate of owning dogs and cats at home was low and did not influence *T. canis* seropositivity. It is reported that the risk factor for toxocariasis is contact with soil contaminated with cat and dog feces, rather than actually owning a cat or dog.[22] Kaplan et al reported that the places where mental retarded and control group subjects lived had similar conditions in terms of contamination with cat and dog feces.[22] However, the duration of being in these places and the manner of using these places may be considered as factors that increase contamination of mental retarded patients.

The main risk factor for toxocariasis in adults was the history of raw food intake**,** the prevalence of toxocariasis was found 7.8 times higher in patients with positive history of raw food intake than those with no history of raw food intake.[23] In the present study, seropositivity for *T. canis* was detected in 30.5% of patients with history of raw food intake and 9.7% of patients with no history of raw food intake and the difference was statistically significant. In line with our results, some authors have mentioned the possibility of getting toxocariasis after eating raw or poorly cooked meat from some domestic animals such as chickens, pigs, sheep or cows.[2,24,25] In addition, Hoffmeister et al identified a case of cerebral toxocariasis caused by the consumption of raw duck liver which is usually one of the most heavily affected organs in toxocariasis because it drains portal venous blood containing large amounts of larvae after oral uptake.[26]

Human toxocariasis has been described as a soil transmitted zoonosis. Geophagia (eating soil) is a specific type of pica that increases the risk of toxocariasis as reported in several studies around the world.[27,28] However, in this study, it was found that this type of human behavior was not significantly

associated to *Toxocara* infection. This might be explained by the possibility that adults may not remember their history of pica.

Schizophrenic patients can be expected to behave in uncontrolled fashion with respect to hygiene.[7] Inadequate personal hygiene; nail-biting and infrequency of daily hand washes increases the risk of *Toxocara* infection.[6,29] The present study showed significant association between *T. canis* seropositivity in schizophrenic patients and inadequate personal hygiene. In agreement with the present study results, Kaplan et al suggested that mental retarded subjects constitute a risk factor for *Toxocara* infection, which may be attributed to their behavioral patterns.[22]

A high prevalence of intestinal parasites occasionally was found together with *Toxocara* infection and the statistical association between them was significant. Our results showed that the parasites found in our schizophrenic patients were protozoa that do not cause cross-reactions with the *Toxocara* IgG ELISA test.[30] Therefore, it is believed that our results reflect the real prevalence of toxocariasis in these patients who might be exposed to a fecal contamination of food or poor hygiene. Institutes for mentally retarded have been reported to be endemic foci of protozoan and helminthic infections, including toxocariasis.[6]

In helminthic diseases, moderate to severe eosinophilia occurs as a pathophysiologic response to the parasitic infection.[31] Repetitive and prolonged antigen exposure to T- lymphocytes by parasites is the cause of eosinophilic stimulation as T cells produce interleukin (IL-5, IL-4 and IL-13), which stimulate proliferation and survival of eosinophils.[32] It has been recognized that toxocariasis is one of the causes of eosinophilia in peripheral blood and provokes eosinophilic infiltration in internal organs.[23] A significant association between eosinophilia and infection by *T. canis* was observed in this study as reported by others.[23,33]

## CONCLUSION

There might be a causal relationship between toxocariasis and schizophrenia, either *Toxocara* may be a possible etiologic agent of schizophrenia or the schizophrenic patients constitute a high risk for *Toxocara* infection. Further studies will be needed to realize the actual relationship between them.

Correspondence: Khadiga Ahmed Ismail. Email: khadigaahmed68@yahoo.com
Rec. Date: Oct 13, 2011 Accept Date: Mar 04, 2012

## REFERENCES

1. Despommier D. Toxocariasis: clinical aspects, epidemiology, medical ecology, and molecular aspects. Clin Microbiol Rev 2003;16:265-72.
2. Magnaval JF, Glickman LT, Dorchies P, Morassin B. Highlights of human toxocariasis. Korean J Parasitol 2001;39:1-11.
3. Rubinsky-Elefant G, Hirata CE, Yamamoto J H, Ferreira MU. Human toxocariasis: diagnosis, worldwide seroprevalences and clinical expression of the systemic and ocular forms. Ann Trop Med Parasitol 2010;104:3-23.
4. Vidal, JE, Sztajnbok J, Seguro AC. Eosinophilic meningoencephalitis due to *Toxocara canis*: case report and review of the literature. Am J Trop Med Hyg 2003;69:341-3.
5. Nicoletti A, Sofia V, Mantella A, Vitale G, Contrafatto D, Sabello V, et al. Epilepsy and toxocariasis: a case–control study in Italy. Epilepsia 2008;49:594-9.

6. Huminer D, Symon K, Groskopf I, Pietrushka D, Kremer I, Schantz PM, et al. Seroepidemiologic study of toxocariasis and strongyloidiasis in institutionalized mentally retarded adults. Am J Trop Med Hyg 1992;46:278-81.
7. Kaplan M, Kalkan A, Kuk S, Demirdag K, Ozden M, Kilic SS. *Toxocara* seroprevalence in schizophrenic patients in Turkey. Yonsei Med J 2008;49:224-9.
8. Williams WJ, Beutler E, Erslev AJ, Lichtman AM. Hematology. 4. ed. New York, Mc Graw-Hill; 1991.p.1832-1833.
9. Torrey EF, Yolken RH. *Toxoplasma gondii* and schizophrenia. Emerg Infect Dis 2003;9:1375-80.
10. Melamede R. Parasitic brain infection, endocannabinoids, and schizophrenia. Medical Hypotheses 2009;72:220-2.
11. Yolken RH, Dickerson FB, Torrey EF. *Toxoplasma* and schizophrenia. Parasite Immunology 2009;31:706-15.
12. Stensvold CR, Skov J, Moller LN, Jensen PM, Kapel CMO, et al. Seroprevalence of human toxocariasis in Denmark. Clin Vaccine Immunol 2009;16:1372-3.
13. Magnaval JF, Michault A, Calon N, Charlet JP. Epidemiology of human toxocariasis in La Reunion. Trans Roy Soc Trop Med Hyg 1994;88:531-53.
14. Kanafani ZA, Skoury A, Araj GF, El-Khoury M, Sawaya RA, Atweh SF, et al. Seroprevalence of toxocariasis in Lebanon: a pilot study. Parasitology. 2006; 132:635–639.
15. 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;79:552-7.

16. Theodoridis I, Frydas S, Papazahariadou M, Hatzistilianou M, Adamama-Moraitou KK, Di Gioacchino M, et al. Toxocarosis as zoonosis. A review of literature and the prevalence of Toxocara canis antibodies in 511 serum samples. Int J Immunopathol Pharmacol 2001;14:17-23.
17. Rubinsky-Elefant G, da Silva-Nunes M, Malafronte RS, Muniz PT, Ferreira MU. Human toxocariasis in rural Brazilian Amazonia: seroprevalence, risk factors, and spatial distribution. Am J Trop Med Hyg 2008;79:93-8.
18. Torgerson PR, Rosenheim K, Tanner I, Ziadinov I, Grimm F, Brunner M, et al. Echinococcosis, toxocarosis and toxoplasmosis screening in a rural community in eastern Kazakhstan. Trop Med Int Health 2009;14:341-8.
19. Doğan N, Dinleyici EÇ, Bor Ö, Töz S Ö, Özbel Y. Seroepidemiological survey for *Toxocara canis* infection in the northwestern part of Turkey. Turkiye Parazitoloji Dergisi 2007;31:288-91.
20. Chiodo P, Basualdo J, Ciarmela L, Pezzani B, Apeztequia M, Minvielle M. Related factors to human toxocariasis in a rural community of Argentina. Mem Inst Oswaldo Cruz 2006;101:397-400.
21. Jarosz W, Mizgajska-Wiktor H, Kirwan P, Konarski J, Rychlicki W, Wawrzyniak, G. Developmental age, physical fitness and *Toxocara* seroprevalence amongst lower-secondary students living in rural areas contaminated with *Toxocara* eggs. Parasitology 2010;137:53-63.
22. Kaplan M, Kalkan A, Hosoglu S, Kuk S, Ozden M, Demirdag K, et al. The frequency of *Toxocara* infection in mental retarded children. Mem Inst Oswaldo Cruz 2004;99: 121-5.

23. Kwon NH, Oh MJ, Lee SP, Choi DC. The prevalence and diagnostic value of toxocariasis in unknown eosinophilia. Ann Hematol 2006;85:233-8.
24. Deutz A, Fuchs K, Auer H, Kerbl U, Aspöck H, Köfer J. *Toxocara*-infestations in Austria: a study on the risk of infection of farmers, slaughterhouse staff, hunters and veterinarians. Parasitol Res 2005;97:390-4.
25. Taira K, Saeed I, Permin A, Kapel CM. Zoonotic risk of *Toxocara canis* infection through consumption of pig or poultry viscera. Vet Parasitol 2004;121:115-24.
26. Hoffmeister B, Glaeser S, Flick H, Pornschlegel S, Suttorp N, Bergmann F. Cerebral toxocariasis after consumption of raw duck liver. Am J Trop Med Hyg 2007; 76:600-2.
27. Espinoza YA, Huapaya PH, Roldán WH, Jiménez S, Arce Z, Lopez E. Clinical and serological evidence of *Toxocara* infection in school children from Morrope district, Lambayeque, Peru. Rev Inst Med Trop Sao Paulo 2008;50:101-105.
28. Roldán WH, Espinoza YA, Atúncar A, Ortega E, Martinez A, Saravia M. Frequency of eosinophilia and risk factors and their association with *Toxocara* infection in school children during a health survey in the north of Lima, Peru. Rev Inst Med Trop Sao Paulo 2008;50:273-8.
29. Ajayi OO, Duhlinska DD, Agwale SM, Njoku M. Frequency of human toxocariasis in Jos, Plateau State, Nigeria. Mem Inst Oswaldo Cruz 2000;95:147-9.
30. Roldán WH, Cornejo W, Espinoza Y. Evaluation of the dot enzyme-linked immunosorbent assay in comparison with standard ELISA for the immune-diagnosis of human toxocariasis. Memórias do Instituto Oswaldo Cruz 2006;101:71-4.

31. Klion AD Nutman TB. The role of eosinophils in host defense against helminth parasites. J. Allergy Clin Immunol 2004;113:30-7.
32. Lampinen M, Carlson M, Hakansson LD, Venge P. Cytokine-regulated accumulation of eosinophils in inflammatory disease. Allergy 2004;59:793-805.
33. Karadam, SY, Ertug S, Ertabaklar H, Okyay P. The comparison of IgG antibodies specific to *Toxocara* spp. among eosinophilic and non-eosinophilic groups. New Microbiol 2008;31:113-6.