Journal of the Egyptian Society of Parasitology, Vol. 54, No. 2, August 2024
J. Egypt. Soc. Parasitol. (JESP), 54(2), 2024: 213 - 226
Online: 2090-2549

TOXOCARIASIS VISCERAL AND OCULAR LARVA MIGRANS: IS IT STILL A NEGLECTED ZOONOTIC DISEASE?

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Abstract

Zoonotic toxocariasis (visceral larva migrans or VLM & ocular larva migrans or OLM) refers to human infection caused by helminths that are not natural human parasites. Toxocariasis is an underestimated geohelminthic infection may be worldwide.

Toxocariasis occurs as a result of human infection with the dog ascarid larvae, *Toxocara canis* or less commonly, the cat ascarid larvae, *T. cati*. VLM is mainly a disease of young children, especially those with exposure to playgrounds and sandboxes contaminated by dog or cat feces. While common globally, prevalence in both animals and people is highest in developing countries. In developed countries, more infections are detected among persons in lower socioeconomic strata. TVLM & OLM clinical presentations, although most infections are asymptomatic, yet in VLM, which occurs mostly in preschool children, larvae invade multiple tissues (mainly liver, lung, skeletal muscle, or heart) causing nonspecific symptoms as fever, myalgia, weight loss, cough, rashes, hepatosplenomegaly accompanied by hypereosinophilia. Migration to CNS (neurotoxocariasis) is uncommon and can cause eosinophilic meningoencephalitis and epilepsy. Death can occur with severe cardiac, pulmonary, or neurologic involvement.

Key words: Toxocariasis, VLM, OLM, Man, Review article.

Introduction

Toxocariasis (=visceral larva migrans or VLM) is zoonotic infection caused by non-human roundworms parasite (Morsy, 2020). Toxocariasis occurs by human infection with dog larvae ascarid, *T. canis*, or, less commonly, cat ascarid, *T. cati*. Another VLM form is caused by human ingestion of pig ascarid, *Ascaris suum* (Holland, 2017). Clinical pivtures consist of VLM and OLM; infection may be subclinical (Lloyd *et al,* 1983). Also, four zoonotic *Ancylostoma* species, geographically restricted (South Africa) can cause cutaneous larva migrans (CLM), a serpiginous, pruritic, creeping dermal eruption, form days to weeks, but can prolong in *A. braziliense* infections, and *A. caninum* reaches humans' patency (Ngcamphalala *et al,* 2020).

Review and General Discussion

Epidemiology: Toxocariasis is worldwide infection occur more common in tropical regions than in temperate one and more in rural populations (Won *et al,* 2008). Larvae can develop at temperatures <50°F, but efficiency decreases with decreases in temperature (Azam *et al*, 2012). In the USA, seroprevalence was 13.9% (CDC, 2011), rates increased among persons in poverty and certain minority groups; mainly African and Americans (Liu *et al,* 2018). More than 40% were reported in Indonesia and Brazil (Hotez and Wilkins, 2009).

Brog and Woodruff (1973) in England found *Toxocara* eggs in 24.4% of 800 soil samples from public areas and playground. In Sudan, Woodruff *et al*. (1981) in Sudan found that soil samples 2/75 of soil samples in Khartoum, & 8/25 samples in Juba contained eggs of *Toxocara* species. Duwel (1984) in Germany examined 31 sand pits in Frankfurt for *Toxocara* eggs in children playgrounds; detected in 27 sand pits infective larvae in 40%

of eggs. Bass *et al.* (1987) in industrial countries found that serologic surveys had highest prevalence antibody titers in children up to ten years old . Bouchet (1995) in France found that toxocariasis patients suffered syndrome of fever, malaise (may wax and wane for weeks or months) cough, wheezing, abdominal pain, myalgia, and somewhat hepatomegaly. Mizgajska (1997) in Poznan found *Toxocara* eggs in 10% of soil samples; the most heavily contaminated areas were courtyards downtown (53%) followed by city parks (17%). Okyay *et al.* (2004) in Turkey reportd that toxocaraiasis (45.9%) seroprevalence was much higher in schizophrenic patients than in general population. Chattha *et al.* (2009) in Pakistan reported toxocariasis rates of 30%. Eslahi *et al.* (2020) in Iran reported that toxascaris infection among people was mild, but in dogs and cats were high.

Omer *et al.* (2021) in Aseer, Saudi Arabia assessed knowledge and disease awareness among 285medical practitioners' questionnaires on zoonotic toxocarasis, only 27% gave correct answers, with poor medical community knowledge. Al-Ghabban (2023) in Tabuk Region reported 7.5% of 200 fecal samples randomly obtained from public places, toxocariasis prevalence was the west agricultural area showed high eggs rate of (7.5%). Al-Awadhi and Jamal (2022) in Kuwait reported that 2.5% seroprevalence of toxocariasis among allergic patients.

Life cycle: The life cycle of *T. canis* occurs in dogs and that of *T. cati* occurs in cats; prevalence of *Toxocara* infection is highest among puppies and kittens. In North America, about 5% of dogs and puppies were infected (Mohamed *et al,* 2009). Man acquires toxocariasis as accidental hosts ingestion of eggs shed in the definitive host stools, into environment, embryonate and become infective after about three weeks (Morgan *et al,* 2013). Infective eggs in soils are readily in relatively warm climates to be infective in for many years (Tyungu *et al,* 2020)

After ingestion of infective eggs by dogs or cats, the eggs hatch and larvae (0.5mm in length) penetrate gut wall and migrate via the lungs, bronchial tree, and enter esophagus; adults develop in small intestine, where they lay eggs passed in stool (CDC, 2019a).

Over 100 million dogs are infected with *T. canis,* shedding billions of eggs into the environment annually (Rostami *et al,* 2020). In older animals, larvae penetrate the gut wall and encyst in tissues, reactivate in female dogs during 3rd trimester pregnancy and infect puppies by transplacental and transmammary routes; adults can become established in the puppies' small intestines (Schnieder *et al,* 2011). *Toxocara* can also be transmitted by ingestion of paratenic hosts. Eggs ingested by small non-canine mammals (as rabbits) can hatch and larvae penetrate gut wall with more migrate into various tissues where they encyst, and if dogs eat these encysted larvae develop into adults egg-laying in small intestine (Glickman and Cype-ss, 1977).

Humans are accidental hosts who become infected by ingesting infective eggs in contaminated soil or encysted larvae in the tissues of infected paratenic hosts. Direct contact with infected puppies and kittens is not classically considered to be a risk factor for human infection since the eggs must embryonate to be infective, but sometimes pets carry the embryonated eggs in their fur (Lee *et al,* 2014). After ingestion, eggs hatch and larvae penetrate intestinal wall and carried by circulation to liver, heart, lungs, brain, muscle, or eyes (Zibaei *et al,* 2019). Larvae don't development in these sites, but host inflammatory response against migrating larvae cause mechanical and immunopathological damage to tissues leads to local reactions the basis of clinical toxocariasis (Beaver *et al,* 1952).

Transmission risk factors: Individuals ingesting embryonated eggs in soil develop infection. Also, infection can be acquired via ingestion of raw liver or undercooked meat from an infected intermediate host (cattle chicken, rabbit, or swine) containing encapsulated larvae (Lim, 2008).

Visceral larva migrans is principally a disease of young children, especially those with exposure to playgrounds and sandboxes contaminated by dog or cat feces (Mok, 1968).

A pet dog is a risk factor (Carvalho and Rocha, 2011). Infection is acquired from playgrounds and public parks (Rubinsky-Elefant *et al,* 2010). Low infection may occur in travelers (Van Den Broucke *et al,* 2015).

Clinical manifestations: Clinical manifestations range from asymptomatic infection to severe organ injury; they occur as a consequence of damage caused by migrating larvae and host eosinophilic granulomatous response. The migration cause eosinophilic infiltration, granuloma formation, or eosinophillic abscesses (Baaten *et al,* 2011). Also, larvae are be unable to grow or replicate in human host, but remain viable for at least seven years post infection (Smith *et al,* 2009). Most infections are self-limited as larvae become encapsulated in muscles or liver.

There are two major clinical manifestations simultaneously occur by the visceral larva migrans (VLM) or ocular ones (OLM) (Paul *et al,* 2009). CDC (2019) added that neurotoxocariasis or neural larva migrans (NLM) is uncommon cause's eosinophilic meningoencephalitis, but death can occur in instances of severe cardiac, pulmonary, or neurologic involvement.

Visceral larva migrans: VLM occurs most commonly in young children and results in hepatitis and pneumonitis as larvae migrate through liver and lungs, respectively. Heavy infection caused fever, anorexia, malaise, irritability, hepatomegaly, respiratory symptoms, pruritic urticaria-like cutaneous lesion, and eosinophilia (Huntley *et al,* 1965).

Larvae frequently localize in liver; hepatic manifestations may include hepatomegaly or nodular lesion. Pulmonary involvement may cause dyspnea, wheezing, and a chronic unproductive cough in 20 to 80% of patients (Snyder, 1961). Riles are common on physical examination. Chest radiograph showed abnormalities in ≥40% of patients with symptomatic illness. Bilateral peribronchial infiltration is most common; parenchymal infiltrates can also occur (Shrand, 1964). Computed tomography (CT) can show multifocal subpleural nodules with halo or ground-glass opacities and ill-defined margins (Sakai *et al,* 2006). Severe respiratory tract involvement is an uncommon complication of heavy infection (Beshear and Hendley, 1973).

Toxocariasis can be mistaken for metastatic disease (Anderson *et al,* 2006), but eosinophilia and radiologic findings may be helpful differentiating features.

Larvae can also travel via systemic circulation to muscles, heart, eye, or CNS (Enko *et al,* 2009). Cardiac involvement in *Toxocara* infection is a rare, but potentially life-threatening complication. Clinical presentation may be myocarditis, pericarditis, or Loeffler endocarditis (eosinophilic myocarditis); heart failure or cardiac tamponade can occur and be fatal (Kuenzli *et al,* 2016). The CNS manifestations include eosinophilic meningoencephalitis, space-occupying lesion, myelitis, and cerebral vasculitis causing seizures. Peripheral nervous system presentations include radiculitis, cranial nerves affection, or musculoskeletal involvement (Jabbour *et al,* 2011). Death due to myocardial or CNS was rarely reported. Eosinophils and their products play a marked role in the pathogenesis of various reactive and neoplastic disorders, depending on disease underlying, molecular defect and involved cytokines, hypereosinophilia may develop leading to organ damage (Valent *et al*, 2023).

Ocular larva migrans: OLM involvement may occur as a sole manifestation of VLM in individuals without antecedent symptomatic VLM history (Good *et al,* 2004). OLM occurs most commonly among older childrden and adolts (Marx *et al,* 2007). In USA, ocular toxocariasis in patient age was 8.5 years; ranged 1 to 60 years (Ota *et al,* 2009).

The ocular lesion is due to larval localization in the eye and granulomatous response around larvae. Common symptoms are unilateral visual impairment, causing failing vision and subsequent strabismus. Typical lesion is a whitish elevated granuloma. Occasionally, OLM may present as uveitis, papillitis, or endophthalmitis (Stewart *et al,* 2005). Ocular lesions may resemble retinoblastoma (Chuah *et al,* 2006). The most serious infection consequence is retina invasion with

granuloma formation in periphery or posterior pole, leading to dragging of retina and eventual retinal detachment ending into blindness (Suh *et al,* 2006). But, all children with newly discovered leukocoria must urgently be referred to an ophthalmologist to exclude retinoblastoma and other life- or sight-threatening conditions. If possible, consultation must occur within a week of suspected diagnosis (Canzano and Handa, 1999).

Other presentations: OLM must be differentiated from retinoblastoma, tumors, exudative retinitis, trauma and congenital ocular manifestations (Strickland, 2000). Mild infection may show only eosinophilia. Others include fever, headache, behavioral disturbances, anorexia, abdominal pain, rash, hepatomegaly, nausea, vomiting, wheezing and pulmonary infiltrates (Fan *et al,* 2015). But, wheezing and pulmonary infiltrates, together with eosinophilia, are also hallmark features of childhood asthma. *Toxocara* larvae in lungs may be an underlying factor in the onset of allergic pulmonary, perhaps due to host response to toxocariasis; a causal association remains uncertain, as positive and negative epidemiological associations were reported (Cooper, 2009). A significantly high prevalence of *T. canis* infection occurred among asthmatic patients (Li *et al,* 2014).

Treatment of OLM, Hagler *et al*. (1981) in France successfully treated 15 patients had stability or improvement in visual acuity and and a patient showed decrease in visual acuity. Bertelmann *et al.* (2007) in Germany reported that systemic anthelminthic with Albendazole the pars plana vitrectomy was a substantial therapeutic option in ocular toxocariasis with vitreo-retinal complications. But, Othman (2012) in Egypt reported that ocular toxocariasis treatment is a matter of controversy not only due to limited efficacy of anthelmintic eradication, but also doubts about the therapy benefits as most toxocaraiasis are self-limiting.

Cutaneous manifestations are common, either alone, together with eosinophilia, and/or in conjunction with other clinical manifestations of toxocariasis. Chronic urticaria is the most dermatologic manifestation; others include chronic pruritus, transient rash, different forms of eczema, hypodermic nodules, and vasculitis (Gavignet *et al,* 2008)
Two additional syndromes were covert toxocariasis (mainly children) and common toxocariasis (predominantly adults); with proba bly variations of same clinical entities (McGuinness and Leder, 2014). Covert toxocariasis refers to non-specific symptoms and signs including fever, abdominal pain, anorexia, nausea, vomiting, hepatomegaly, cough, headache, lethargy, behavioral and/or sleep disturbances, skin symptoms, limb pains and lymphadenitis associated with high anti-*Toxocara* antibodies titers, with or without eosinophilia (Taylor *et al,* 1987). Common toxocariasis refers to a syndrome comprised of chronic weakness, breath shortness, abdominal pain, rash, itch, urticarial and arthralgia, with eosinophilia, high IgE levels, and high *Toxocara*-specific antibodies titers (Glickman *et al,* 1987).

Laboratory tests: Generally VLM must be suspected in compatible clinical manifestations setting, together with leukocytesis, eosinophilia, and hypergammaglobulinemia (elevated levels of IgE, IgG, & IgE). Kim *et al*. (2014) in Korea found that toxocariasis antibody positive rate correlated with eosinophil counts. They added that healthy ones with eosinophilia by routine examination and risk factors underwent *Toxocara* tests by multi-antigen ELISA for etiology. There is no peripheral blood eosinophilia in OLM most cases, due to less larval load. Eosinophilia may be absent in long-standing infected patients, but only with cutaneous symptoms (Pawlowski, 2001). Eosinophilic granulomatous hepatitis may develop, causing abnormalities in liver function tests with elevated ALP and /or AST (Azuma *et al,* 2002).

Diagnosis laboratory assays: Several commercial ELISAs can detect human IgG antibodies to *Toxocara* excretory/secretory antigens. The test can detect subclinical or mild infection though it cannot differentiate between *T. canis* and *T. cati* infections. Reliability varies by assay and clinical presentations.

In visceral larva migrans (VLM) and some covert toxocariasis forms, sensitivity and specificity of *Toxocara* enzyme immuno-assay (1:32) were estimated at 78% & 92%, respectively (Jones *et al,* 2008).

A positive ELISA result doesn't necessarily indicate active *Toxocara* infection or prove that clinical symptoms are due to toxocariasis (Glickman *et al,* 1978), but result must be interpreted in the setting of compatible clinical symptoms and epidemiologic exposure. Cross-reactivity of ELISA with other parasite antigens is common, and test may remain positive for several years even after treatment. In some settings, positive ELISA results was confirmed by western blot (Magnaval *et al,* 1991), with better sensitivity and specificity compared to ELISA (Roig *et al,* 1992), but also more expensive and labor intensive (Fillaux and Magnaval, 2013). Definitive diagnosis of VLM may also be by larval detection in the biopsy tissue, which showed *Toxocara* larvae within eosinophilic granulomatous lesions, but biopsy is rarely indicated.

The ELISA sensitivity for OLM is considerably lower than for VLM; the diagnosis of OLM generally relies on the ophthalmologic examination results (Despommier, 2003). It is possible to compare the antibody levels between serum and aqueous humor; if the level of specific IgG in aqueous humor/ level of specific sera IgG & total IgG in aqueous humor/ total IgG is greater than 3.0 that can be considered diagnostic (de Visser *et al,* 2008). PCR-based tests to diagnose *Toxocara* in clinical samples were described but not commercially available (Fogt-Wyrwas *et al,* 2007). Circulating antigen were developed (Rodríguez-Caballero *et al,* 2015).

Stool examinations are not helpful as the parasite does not complete a full life cycle involving the human gastrointestinal tract.

Pulmonary involvement may result in eosinophilia that is detectable in bronchoalveolar lavage (BAL) fluid. But, marked pulmonary infiltration eosinophils were 64% in BAL analysis (Cottin *et al*, 2014).

In the setting of central nervous system involvement, cerebrospinal fluid may show eosinophils (Eberhardt *et al,* 2005) Generally, eosinophilic meningitis is defined by the presence of at least 10% eosinophils in the total cerebrospinal fluid (CSF) leukocyte count. There are several possible causes of eosinophils in CSF, parasitic infection is the main cause. Three common parasites causing eosinophilic meningitis are *Angiostrongnylus cantonensis, Gnathostoma spinigerum* and *Taenia solium* (Sawanyawisuth and Chotmongkol**,** 2013). Also, VLM must be differentiated from capillariasis and peripheral hyper-eosinophilia (Francalanci *et al,* 2023).

Imaging studies: Hepatic and cerebral lesions may be observed with ultrasonography, computed tomography (CT), and magnetic resonance imaging (MRI) (Jain *et al,* 1994) In one review of radiographic changes associated with hepatic VLM, CT and MRI showed multiple, ill-defined lesions, usually measuring 1.0 to 1.5cm in diameter, scattered throughout the liver parenchyma (Zachariah *et al,* 1994). Lesions are usually oval but may be angular or trapezoid. The lesions differ from metastatic nodules in that they are usually uniform in size, non-spherical in shape, and are best seen on portal venous phase. Pulmonary VLM appear on CT as multifocal sub-pleural nodules with halo or ground-glass opacities and ill-defined margins (Ribeiro and Fischer, 2002).

In reviewing radiographic changes associated with pulmonary VLM, four patterns occur: ground-glass opacities (GGOs), solid nodules, consolidation, and linear opacities; predominant lower lung involvement (Lee *et al,* 2015).

Differential diagnosis: The VLM differential diagnosis includes the following: 1- Strongyloidiasis: Transpulmonary migration of larvae can produce dry cough, throat irritation, dyspnea, wheezing, and hemoptysis (Be rk *et al,* 1987). Also, pulmonary toxocariasis involvement may cause dyspnea, wheezing, and a chronic, nonproductive cough, and larvae may be detected in stool, sputum, bronc-

hoalveolar lavage fluid, or pleural fluid (Minamii and Nishioka, 2024). Strongyloidiasis is well documented in Egypt (Zaky *et al,* 2019) and even worldwide (Wilson, 1991).

2- Schistosomiasis: *S. mansoni* infection can cause periportal fibrosis and eosinophilia; in general, there are no apparent signs of liver dysfunction (Elbaz and Esmat, 2013). This disagreed with VLM, which may be associated with abnormal liver function tests including elevated transaminases and/or alkaline phosphatase. Diagnosis is by stool microscopy and/or serologic tests (Abdel-Wahab *et al*, 1979). Both types of schistosomiasis are well documented in Egypt (Abo-Madyan *et al,* 2004). Human infections are caused by *S. mansoni, S. haematobium*, or *S. japonicum* with at least 251.4 million people required preventive treatment in 2021 (WHO, 2024).

3- Ascariasis: Ascariasis may be associated with pneumonitis (=Loeffler's syndrome) in sensitized individuals during larval migration through the lungs (Ramamoorthy, 2015). Pulmonary symptoms are not common in regions with continuous transmission. *Ascariasis* may also involve the biliary tree, but is not associated with hepatitis as with VLM and its larvae cause peripheral eosinophilia (Das, 2014). *Ascaris lumbricoides* (ascariasis) a very large nematode, is one of the soil-transmitted helminthes with 807 million-1.2 billion people in the world are infected particularly in tropical and subtropical areas of Asia, Africa, and Americas (CDC, 2023a)

4- *Echinococcus*: *Echinococcus* is associated with cystic lesions in t liver and lung as well as other organs and detected with ultrasonography or computed tomography (El-Sayed *et al,* 2020), but VLM may be appreciated on computed tomography as nodular lesions. Echinococcosis is always expensive & complicated to treat and may require extensive surgery and/or prolonged drug therapy (Ibrahim and Morsy, 2020). Rupture of the cyst, either spontaneous or traumatic, cause site-specific complications. Rupture of hepatic cysts into biliary tree result in biliary obstruction, super-infection of the cyst, and secondary peritonitis, while rupture of lung cysts into the bronchial tree can cause pneumonitis, pneumothorax, pleural effusion, and secondary pleuritis and can be fatal (Moro and Sc hantz, 2009). CDC (2019b) reported that *E. granulosus* (sensu lato) occurs practically worldwide and more frequently in rural, grazing areas where dog ingests organs from infected animals. Others, *E. multilocularis* cause alveolar echinococcosis, and become increasingly more common. Two exclusive ly New World species, *E. vogeli* and E. *oligarthrus,* are associated with Neotropical infection. *E. vogeli* causes a polycystic form, but *E. oligarthrus* causes extremely rare unicystic one. In *E. multilocularis,* foxes, particularly red one (*Vulpes vulpes*), are the primary definitive host species (CDC, 2019). Others including domestic dogs, wolves, and raccoon dogs (*Nyctereutes procyonoides*), are also competent definitive hosts. Rodents of subfamily Arvicolinae can serve as intermediate hosts (voles, lemmings, and related rodents) are the most typical (CDC, 2023b).

5- Allergic bronchopulmonary aspergillosis: The allergic bronchopulmonary aspergillosis (ABPA) is an inflammatory disease caused by immunologic reactions initiated against *Aspergillus fumigatus* colonized the patients' airways with asthma and cystic fibrosis (Agarwal, 2009). Clinical pictures were recurrent episodes of bronchial obstruction, fever, malaise, cough, and peripheral blood eosinophilia, and chest radiography showed parenchymal infiltrates and bronchiectasis (Rosenberg and Khoury, 2021). Moreover, others cause pulmonary eosinophilia; such as clonorchiasis, coccidioidomycosis, echinococcosis, fascioliasis, gnathostommiasis, opisthorchiasis, paragonimiasis, and schistosomiasis (Kunst *et al,* 2011) that occurred some Eastern Mediterranean Countries (WHO, 2020)

Besides, differential diagnosis of ocular larva migrans (OLM) includes: 1- Retinoblastoma: Both retinoblastoma and OLM may present with strabismus, poor vision, and leukocoria always a danger signal as retinoblastoma, a malignant retinal tumor, is responsible for half of the infants (Balmer and Munier, 2007). Symptoms of retinoblastoma in

children (eye cancer) are white (leukocoria) or red pupil instead of the normal black, misaligned eyes (strabismus) looking toward ear or nose, reddened, painful eye, enlarged pupil, different-colored irises, and poor vision (Fang e*t al,* 2020).

2- Toxoplasmosis: Toxoplasmosis is a zoonotic protozoan disease caused by infection with *Toxoplasma gondii*, one of the worldwide zoological and geographical distributions (Al-Agroudi *et al,* 2017). Congenital ocular toxoplasmosis (chorioretinitis a posterior uveitis) significantly causes blindness. Retinochoroiditis is the most common finding, but other ocular manifestations include microphthalmus, nystagmus, strabismus, and ptosis (Perry and Merritt, 1983). Retinochoroiditis usually presents together with focal necrotizing granulomatous retinitis, reactive granulomatous choroiditis, vitritis and even inflammatory activity of anterior segment. But, many cases may present with substantial clinical variations leading to difficult diagnosis (Bosch-Driessen *et al,* 2002).

3- Ocular tuberculosis: Clinical manifestations suggestive of TBU include granulomatous anterior uveitis with broad-based posterior synechiae, occlusive retinal vasculitis with or without choroiditis, and serpiginous-like choroiditis. These ocular phenotypes raise an alarm about OTB, but when corroborated by the positive investigation test results, merit specific treatment (Testi *et al,* 2020).

4- Other parasitic infections can cause of eosinophilia in the setting of focal lesions; examples include liver lesions caused by capillariasis (Li *et al,* 2010), or brain lesions caused by gnathostomiasis (Anantaphruti *et al,* 2005) and toxocariasis prevalence in patients with unexplained eosinophilia was 70% (Kwon *et al,* 2006).

Also, eosinophilic meningoencephalitis is caused by a variety of helminthic infections, the most common being angiostrongyliasis, gnathostomiasis, toxocariasis, cysticercosis, schistosomiasis, baylisascariasis, and paragonimiasis (Graeff-Teixeira *et al,* 2009).

Treatment: The treatment approach varies according to symptoms and site of the larvae; data are limited. Treatment is generally based on clinical experience and expert opinion. Most individuals with toxocariasis mild symptoms don't require anthelminthic therapy; symptoms are usually self-limited and resolve within a few weeks (Schantz and Glickman, 1978). Eosinophilia may resolve much more slowly over many months, likely due to ongoing antigenic stimulation from dead larvae. In the setting of protracted symptoms, the possibility of reinfection (such as ingestion of contaminated soil) should be considered. For individuals with moderate to severe symptoms due to visceral larva migrans (VLM), treatment consists of albendazole as 400mg orally with fatty meal twice daily for five days (Stürchler *et al,* 1989).

In cases of severe respiratory, myocardial, or CNS involvement, concomitant prednisone (0.5 to 1.0mg/kg/day) is warranted. Some favor treatment in setting of moderate eosinophilia and positive serology even in the setting of minimal symptoms, given risk of larval localization to brain during the infection course (Yoon *et al,* 2018). Long-term treatment with albendazole was safe and effective for larva migrans syndrome (Hombu *et al,* 2019). For ocular larva migrans (OLM) treatment with *s*ight-threatening ocular inflammation, aggressive anti-inflammatory therapy as corticosteroids (e.g., prednisone 0.5 to 1.0mg/kg/ day with slow taper), together with albendazole (800mg/day for adults and 400mg/day for children with fatty meal) is warranted for two to four weeks (Barisani-Asenbauer *et al,* 2001). In complicated cases, surgical intervention may be warranted (Giuliari *et al,* 2011).

Mebendazole (100 to 200mg orally twice daily for five days) is an alternative to albendazole (Magnaval, 1995), albendazole was preferred; particularly in OLM & neurologic disease since it crosses blood-brain barrier. Follow-up consists of monitoring the eosinophil count that usually decreases within one month of treatment (Magnaval *et al,* 2001). Serology was not a good follow-up as IgG decreases very slowly and IgE decreases after therapy, but not reliable (Elefant *et al,*

2006). Nitazoxanide induced a significantly lesser reduction in larval load in *T. canis* infected mice than Mebendazole (Lescano *et al,* 2025). But, levamisole was rarely used to treathuman toxocariasis, with its use only recorded in one case of common/ covert toxocariasis and a case of OT and was withdrawn no longer used (Campillo *et al,* 2022).

Ivermectin was (40%) effective for toxocariasis (Magnaval, 1998). Diethylcarbamazine (3 to 4mg/kg/day for 21 days, starting at 25mg/day for adults) was effective in a small number of cases, but with greater side effects than albendazole so is rarely used (Chaudhuri and Saha, 1959). Ivermectin may be given in a single 12 mg dose and has few side effects; drug was sporadically used for human toxocariasis treatment, but when given orally, it strongly associates with digesta, which reduced potential for absorption and elicited a great variability in half-life (Magnaval *et al,* 2022). However, ocular toxocariasis treatment is more difficult and usually consists of measures to prevent progressive eye damage (CDC, 2017). Granulomas can be surgically removed, or laser photocoagulation and cryoretinopexy can be used to destroy ocular granulomas (Auweerter, 2008).

Prevention: Good hygiene practices, timely disposal of pet feces, and routine deworming of pets are important strategies to prevent zoonotic infection (Guha *et al,* 2015). Handwashing must be encouraged after contact with pets or areas at high risk for soil contamination, such as playgrounds and sandboxes (Macpherson, 2013). Health education as to behaviors is a vital, low-cost, and simple component of most interventions to prevent and control soil transmitted diseases.

What about Egypt: Khalil *et al.* (1976) in Siwa Oasis, and Qena and Aswan Governorates (G), reported toxocariasis in man and dogs. Khalil *et al*. (1978) examined soils for *Toxocara* species eggs; they found 12.5% in Giza, 11.5% in Cairo, 10% in Dakahlia and 7.5% in Qualyobia. Hanafi *et al*. (1987) in Dakahlia G found that soil pollution with T. canis was indoors (100-200 eggs/50gm soil) and outdoors (9001000 eggs/50gm soil), with huge abundance of stray dogs. Oteifa and Moustafa (1997) in Greater Cairo examined 600 soil samples from three sports clubs; reported *Toxocara* eggs of pet dogs and cats in 182 samples (30.3%). El Shazly *et al.* (2003) in Dakahlia G. found *T. canis* eggs in canal water. El-Beshbishi *et al*. (2005) detected toxocariasis eggs in 9.1% of soil samples in four rural villages with high outdoors intensity (12-15 eggs/10gm) than indoors (1-2). Antonios *et al.* (2008) in Tanta City found *Toxocara* IgG antibodies 6.2% among suspected children and 18% among adults. El Shazly *et al.* (2009) in Dakhalia G. found that *T. canis* eggs soil contaminated was risky. Haridy *et al.* (2009) examined 3000 pet dogs over two years at Cairo University Veterinary Teaching Hospital; apart from ecto- and endo-parasites, detected *Toxocara* eggs in 9.83%. Khalafalla (2011) in northern Delta among stray cats detected *T. cati* (9%) and *Toxoascari sleonine* (5%). El-Tras *et al.* (2011) in Kafrelsheikh G. detected *T. canis* eggs in stools of 64 (26.6%) stray and 56 (10.7%) domestic dogs, and recovered embryonated eggs from hair of 11 stray and 9 domestic ones. Farghly *et al.* (2016) reported that toxocariasis occurs especially in the rural populations with poor hygienic levels where stray and pet dogs and cats play a marked role in its spreading among Egyptians

Abdel Aziz *et al.* (2019) examined 296 pet dogs admitted to veterinary hospitals in four governorates, found a high *T. canis* (53.1%). This higher rate might be due to climatic and /or ecological factors (Rostami *et al,* 2020). El-Seify *et al.* (2021) in Alexandria G. found high prevalence *T. cati* in feral cats. Temsah *et al.* (2021) correlated between *Toxocara* and children asthma, urticaria and neurologic manifestations. Abbass *et al.* (2023) in Dakhalia detected in feces of 21/78, (35.9%) had eight parasites species included *T. canis* (19.2%), and *T. leonine* (2.6%). Abd El Wahab *et al*. (2023) among 50 asthmatic, 50 pneumonic children and 50 healthy controls found anti-*Toxocara* IgG antibodies significantly higher in 27.3% (41/150) of asthmatic children compared to controls (26%) and si-

gnificantly highest in pneumonia one compared to bronchial asthma (46%) and controls (10%) with significance between anti-*Toxocara* IgG and eosinophilia, IgE and both.

Conclusions

Toxocariasis occurs as a result of human infection with larvae of *T. canis*, or less common *T. cati*. VLM is a disease of young children, especially those with exposure to playgrounds and sandboxes contaminated by dog or cat feces. Puppies are a major source of environmental egg contamination. Humans are accidental hosts become infected by ingesting eggs in soil or encysted larvae in tissues of infected paratenic hosts. Ingested eggs mature into larvae which penetrate the intestinal wall and are carried by circulation to liver, heart, lungs, brain, muscle, or eyes. Larvae in liver cause hepatomegaly or nodular lesions. Mild infection may be asymptomatic and only suspected by elevated blood eosinophilia. Heavy infection may cause fever, anorexia, malaise, irritability, hepatomegaly, and pruritic urticaria-like lesions. The VLM must be suspected in setting of compatible clinical manifestations together with leukocytosis, eosinophilia, and elevated IgE, IgG, & IgE. In clinical scenario, diagnosis is proved by ELISA for human IgG antibodies to *Toxocara* excretory/secretory antigens.

OLM showed granulomas. Common symptoms are unilateral visual impairment and thus strabismus; visual loss can occur.

Patients with mild toxocariasis symptoms may not require treatment as symptoms are usually self-limited. Those with moderate to severe symptoms, albendazole 400mg orally with fatty meal twice daily for five days, as well as in severe respiratory, myocardial, or CNS cases is recommended (Grade 2C).

Good hygiene practices, timely disposal of pet feces, and routine deworming of pets are important strategies for prevention of toxocariasis in humans. Hand-washing should be encouraged after contact with pets or areas at high risk for soil contamination.

Authors' declaration: They declared that neither have any conflicts of interest nor received any funds.

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Explanation of figures

Fig. 1: Hepatic toxocariasis in 43old man: Contrast-enhanced arterial (A), portal phases (B), and equilibrium phase (C). Computed tomography images display two small triangular and oval nodules in periphery of liver: Nodules on portal venous phase image but not clearly on arterial and equilibrium phase images (Lim, 2008).

Fig. 2: Toxocariasis - Chest: (A) Radiograph of a three-year-old girl, (B) Computed tomography scan in panel A (Ribeiro and Fischer, 2002).