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Enterobius Vermicularis: Does it Invade Central Nervous System?

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Abstract Enterobiasis is a common intestinal parasitic infection of humans caused by *Enterobius vermicularis* (*E. vermicularis*). Although it affects all age groups, in most instances, it causes a self-limiting illness. Infections in children may result in severe morbidity. Enterobiasis can remain as a chronic infection in cases of bad personal hygiene, and those who are not adequately treated. Dissemination of *E. vermicularis* larvae from the perineal and peri-anal regions into the vagina and other associated regions has been adequately documented. There are several reports of ectopic presentations of enterobiasis. This report presents a fatal case of a four-year-old child with *E. vermicularis* infection who presented with central nervous system (CNS) complications.

Keywords: *enterobius vermicularis, enterobiasis, childhood, dissemination, central nervous system (CNS)*

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1. Introduction

Enterobius vermicularis (*E. vermicularis*) is a nematode residing in the large intestine of humans. It is commonly called as pinworm, threadworm, and seatworm. Enterobiasis is a common parasitic infection spread throughout the world and usually causes infection in school going children, presenting as peri-anal itching (pruritus ani), attributed to the mucoid secretions of the eggs on the skin. The adult worms although don't contribute to the pathogenicity, are required for the continuation of the life cycle. Enterobiasis, although is not a life-threatening infection, it could lead to severe morbidity among the infected persons. Children suffering from enterobiasis suffer from sleeplessness, nausea, vomiting, abdominal pain, diarrhea and anemia. Some infected patients might also show symptoms that include nocturnal enuresis, perianal excoriations, biting of nails, nervousness, hyperactivity, and loss of weight. Infection could be symptomless or may present with mild symptoms among adults. In females, the larvae hatched out on the peri-anal skin might crawl back and enter the vagina, and wander through the fallopian tubes, uterus and might enter the peritoneum [1-5].

E. vermicularis does not have the buccal apparatus that helps in penetration, but it still manages to spread hematogenously, through intestinal mucosal abrasions, and may reach various organs of the body including the liver, lungs, and other organs like ovary, kidney, spleen, and are known to cause threadworm granulomas [6,7,8]. Most infections of Enterobius have been diagnosed at sites other than the intestines, which proves its invasiveness especially in susceptible hosts [9-12].

This report presents a case of a four-year-old girl child suffering from enterobiasis who died of central nervous system (CNS) complications.

2. Case Observation and Presentation

A 4-year-old school going girl child was brought to the department of pediatrics at the Prathima Institute of Medical Sciences, Karimnagar, Telangana, India. She presented with complaints of fever, and vomiting's for 1 month, headache in the last 15 days, and abnormal movements of upper and lower limbs. The fever episodes were noted to be intense, sudden onset, and intermittent in nature. On admission, the patient was very irritated, biting the nails, and not cooperating for the treatment. There was no previous history of serious illness or hospitalization, and the child was completely normal before the start of symptoms, except that there was a history of finding tiny, live worms in the stool, and repeated episodes of abdominal pain, and diarrhea a year ago, which was treated by a local private medical practitioner. The child was second among siblings and had reached all milestones normally. The socioeconomic condition of the child appeared to be low, with both the parents being daily wage laborers, living in a poorly ventilated home.

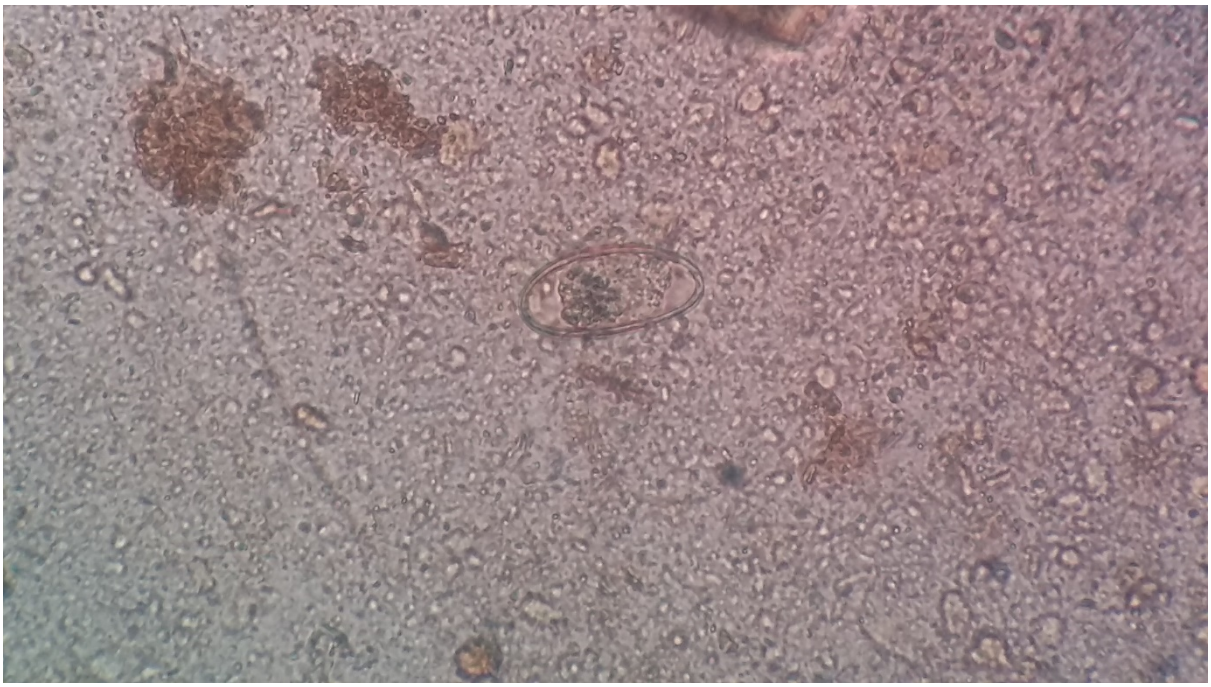

Figure 1. Stool wet mount showing characteristic plano-convex, non-bile stained eggs of *Enterobius vermicularis*

On clinical examination, the cardiovascular system, and the respiratory systems were found to be normal. Central nervous system examination revealed positive for neck stiffness, cerebellar ataxia, and Kernig's sign. Glasgow coma scale of 13/15 (15-best response; <8-comatose; <3-totally unresponsive) was noted, and grade I protein-energy malnutrition was observed.

Computed tomography (CT) scan of the brain revealed prominent ventricles, with slightly effaced basal cistern, and suggestive of obstructive hydrocephalus. A provisional diagnosis of subacute meningitis was made with advice to undergo magnetic resonance imaging (MRI) of the brain for further evaluation and confirmation.

MRI studies revealed T2 hyperintense regions in right thalamus with diffusion restriction and corresponding apparent diffusion coefficient (ADC) showing suppression, suggestive of acute infarction in the right thalamus. MRI also demonstrated lacunar infarct in the left lentiform nucleus, with mild dilatation of the temporal horns and lateral ventricles. An electroencephalogram was performed, which was suggestive of possible encephalitis.

The C-reactive protein (CRP) was raised (1.2 µg), with 12.5 g% haemoglobin, increased erythrocyte sedimentation rate (ESR) (30 mm), leucocytosis (19,000 cells/mm^3), neutrophilia (74%), lymphopenia (20%), and hyponatremia (128 mmol/L). The parasite F test for HRP-2 antigen of *Plasmodium* was negative. Cerebrospinal fluid (CSF) was collected and sent to the clinical laboratory. It was clear, and colourless in appearance, with a total cell count of 6 cells/mm^3 (Normal value: 0-20 cells/mm^3), 90% neutrophils, 10% lymphocytes, with protein (54 mg/dl, Normal value: 20-40 mg/dL) and glucose (27 g% Normal value: 45-80 mg/dL). Gram's stain of the CSF showed no bacteria, and it was negative for acid-fast bacilli. Wet mount of the CSF showed no fungal elements, and other parasites like the Acanthamoeba, and Naegleria.

Stool for pathology and parasites was received in the clinical laboratory. It was negative for reducing substances, and occult blood. Stool wet mount showed the presence of characteristic plano-convex, non-bile stained eggs, which were morphologically resembling *E. vermicularis* as shown in Figure 1. The Images of the parasite egg were sent to the Center for Disease Control and Prevention (CDC), Atlanta, USA, and was positively confirmed (dpdx@cdc.gov).

A diagnosis of possible meningoencephalitis, with cerebral infarctions, was made. The patient was managed for fever. Care was taken for the maintenance of blood pressure and was given vitamin supplements (vitamin E, vitamin B12), valproate and gabapentin. Although the patient was recommended to be under observation, they were discharged against medical advice. The patient got admitted on 30th October 2017, discharged on 8th November 2017. The condition of the patient deteriorated and was brought on 22nd November 2017, only to be declared as brought dead.

3. Discussion

E. vermicularis is previously called Oxyuris, and the disease caused by it was called as oxyuriasis [13]. Human infection with *E. vermicularis* could be as old as 10,000 BC, as evidenced from a report, which found eggs of pinworm in the coprolites (fossilized feces) excavated from Hopug and Danger caves of western Utah [14]. Report of *Enterobius* egg attached to an adolescent female sacral bone, dating back to 7000 AD was excavated in Iran [15]. Evidence of pinworm eggs in the human coprolites, excavated in Chile, which is dated between 400-800 AD was also reported [16].

Human enterobiasis is ubiquitous, prevalent both in the tropical and temperate climate. This could be because its life cycle does not involve any developmental stage in the environment, or the soil as required by most other nematode parasites. Transmission of infection requires close association with infected patients as it does not have a soil development phase, as seen in other nematode

parasites. The Human infection with *E. vermicularis* results from the ingestion of embryonated eggs, which hatches out larvae in the small intestine. These larvae then move into the large intestine (caecum and appendix), develops into adult male and female worms, mature, and undergo fertilization. The female gravid worm has the tendency to migrate towards the rectum, perianal region, and perineal areas to deposit the eggs. These eggs then may cause auto-infection (through nails), and retro-infection (movement of hatched out larvae back into the intestine) or be a source of infection to people living with the infected person [5]. Most human infections resolve automatically without treatment. In some cases, treatment may be necessary to reduce the morbidity and re-infections.

There are fewer reports of prevalence of enterobiasis from India. In a recent study Bharti B et al. have reported that soil-transmitted helminths including the ascariasis, strongyloidiasis, trichuriasis, hookworm infection and enterobiasis account for a combined prevalence rate of 20% [17]. A 2% prevalence rate of enterobiasis and a 35% combined prevalence of soil-transmitted helminthic infections (ascariasis, trichuriasis, taeniasis, infections with hookworm, and *Hymenolepis nana*) among school going children was reported by Ragunathan L et al. from South India [18]. A report from the United States of America (USA), by Burkhart CN et al. has noted that enterobiasis is the most common helminthic infection in the USA, and Western Europe, with prevalence rates of 30-50%. The same study had also highlighted the complications of the genito-urinary tract invasion of the parasite [19]. In a recent report from Iran by Moosazadeh M et al. had reported a prevalence rate of around 17% among various age groups of children [20]. A study by Li H-M et al. from China (Gaozhou, Guangdong Province) reported a 50% prevalence of enterobiasis among children [21].

It was noted that in cases where the parasite develops ectopic lesions in non-habitual sites, the histopathological diagnosis becomes necessary and complicated [22,23,24].

Unusual presentations of human enterobiasis have been reported in the literature recently, where a 55-year-old male originating from Pakistan, had suffered from an inflammatory caecal polyp. This was initially misdiagnosed as a malignancy and was later confirmed as a case of enterobiasis. This patient gave a history of the presence of parasite worms in stool during his teenage [25]. This report signifies the chronicity of the infection and the potential invasiveness of *E. vermicularis*.

4. Enterobiasis and CNS Involvement

A thorough search of the available literature had only identified two studies which had directly associated *E. vermicularis* infection with CNS invasion [26,27]. Interestingly, both reports originated from Iran. The first report had described a fatal case of a 60-year-old man who presented with meningitis. The CSF collected from the patient showed the adult worm, the larvae, and the eggs [26]. The authors were not sure how the parasite had gained access to the brain. The second report describes a case of an eight-year-old boy, who presented with symptoms of severe neck pain radiating to the right shoulder. The magnetic resonance imaging of the spinal cord revealed intra-dural extra-medullary mass arising from the dura on the right anterolateral side of the spinal cord. The pathological observation of the mass-like lesion showed the presence of structures resembling *E. vermicularis* adult worm and the eggs. This report hypothesized the hematogenous spread of the parasite from the capillaries into the spinal cord [27].

5. Association of Other Parasitic/Nematode Infections with CNS Complications

The invasion of the parasite into the brain and the development of CNS complications was only reported in rare circumstances.

Previous research studies have noted that during various parasitic infections, which might cross the blood-brain barrier may contribute to significant pathology, which in most instances could lead to severe consequences, and even death [28,29]. As noted by the previous research, there are possibilities that the infection could prolong for a long time, and in cases of immunosuppression, and increased parasitic burden may lead to severe morbidity and mortality [30,31].

Clinical symptoms, during CNS involvement of nematode parasites as reported previously, may be slowly progressive, and include severe headache, infection of the eyes, encephalopathy (eosinophilic meningoencephalitis), meningitis, altered mental status (restlessness), meningismus (symptoms without pathological changes in the meninges), aneurysms, vasculitis, and intracranial haemorrhages [31-36].

It was also noted that in most cases a biopsy or CSF examination may not show the presence of the parasite, and the diagnosis is made based on the clinical, and radiological findings, along with a history of exposure [31].

Radiological features during nematode infections include hyperintense regions near the basal ganglia may be seen, along with small hemorrhages, and granulomatous changes. White matter abnormality, obstructive hydrocephalus, subarachnoid hemorrhages, and T2 hyperintense regions, and small infarctions on magnetic resonance imaging (MRI) are also reported [37-42].

Previous research has reported the occurrence of secondary bacterial meningoencephalitis caused by *Staphylococcus aureus* in a squirrel monkey, which had an intestinal infestation of nematode larvae. In this case, the CSF showed no larvae, but laboratory results of stool examination, complete blood picture, biochemical profile, and urine analysis revealed systemic disease [43].

6. Other Variable Findings during Parasitic Infections

It was observed that the serum eosinophilia, which is prominent during acute infections, might be absent in chronic cases during nematode infections [44]. Another significant observation is the presence of hyponatremia among the patients suffering from disseminated nematode infection (strongyloidiasis) [45].

The patient in discussion appears to have impaired immune system due to grade 1 protein-energy malnutrition and had chronic intestinal enterobiasis. This is confirmed by the history, as well as the presence of several eggs in the un-concentrated stool specimen, which indicates high parasite burden. Laboratory results revealed increased CRP, ESR, and hyponatremia. The patient was born normal, had no other underlying illness, nor was debilitated, and was going to school regularly before being admitted with CNS symptoms. The patient developed severe headache, neck stiffness, meningeal irritation, and was positive for Kernig's sign on presentation. MRI of the brain showed the presence of T2 hyperintense regions, signs of obstructive hydrocephalus, and small infarctions in the right thalamus. With the available literature, the patient in discussion had CNS symptoms which have already been reported during other nematode infections. And It is not sure if the *E. vermicularis* infestation and associated pathology had caused the CNS complications and death of the patient.

7. Differential Diagnosis

There are several microbes/microbial infections, which could be associated with the central nervous system complications. These include bacterial infections (Leptospirosis, listeriosis, Meningococci, Rickettsiae, tuberculosis, and others), viruses (Enteroviruses, Arboviruses including *Japanese B encephalitis virus*, *Measles*, *Epstein-Barr virus*, *Varicella-zoster virus*, *Mumps*, *Herpes simplex virus*, *Rabies*, and *Human immunodeficiency virus*), fungi (Cryptococcosis), and parasitic infections (Cerebral amoebiasis, *Naegleria*, *Acanthamoeba*, Malaria, Trypanosomiasis, Strongyloidiasis, Schistosomiasis, Echinococcosis, Trichinosis, Onchocerciasis) [46,47,48,49,50].

8. Conclusion

Only two previous research reports had associated *E. vermicularis* infection with CNS complications in humans. In the present case, the infection could have persisted for a long period attributed to the impaired immunological response due to the presence of grade 1 protein-energy malnutrition, continuous re-infections (child hygiene, low socioeconomic status), and due to non-specific, or inadequate treatment. In our patient, who presented with CNS complications, the occurrence of intestinal parasitic infestation could have been an accidental finding. It can be hypothesized that the parasite larvae might have gained access into the brain, and was responsible for the CNS complications, which was, unfortunately not confirmed. Although the patient was negative for malaria, other causes of CNS complication was not completely ruled out. In view of limited data on the prevalence of human enterobiasis and, because there have been reports of ectopic enterobiasis, it is important to understand the pathophysiology of *E. vermicularis* infection, and its potential to invade various organs of the body including the central nervous system.

References

[1] Tsai CY, Junod R, Jacot-Guillarmod M, Beniere C, Ziadi S, Bongiovanni M. Vaginal Enterobius vermicularis diagnosed on liquid-based cytology during papanicolaou test cervical cancer screening: A report of two cases and a review of the literature. Diagn Cytopathol. 2017 Sep 14.

[2] Saleem F, Malik F, Fatima S. Enterobius vermicularis in tubo-ovarian abscess: A rare and interesting incidental finding - A case Report. J Pak Med Assoc. 2017 Apr; 67(4): 630-633.

[3] Shetty JB, Kulkarni DV, Prabhu V. Eggs containing larvae of Enterobius vermicularis in vaginal smear. Journal of Cytology / Indian Academy of Cytologists. 2012; 29(1): 94-96.

[4] Raju K, Verappa S, Venkataramappa SM. Enterobius vermicularis infestation masquerading as cervical carcinoma: A cytological diagnosis. Journal of Natural Science, Biology, and Medicine. 2015; 6(2): 476-479.

[5] Caldwell JP. Pinworms (Enterobius Vermicularis). Canadian Family Physician. 1982; 28: 306-309.

[6] Serpytis M, Seinin D. Fatal case of ectopic enterobiasis: Enterobius vermicularis in the kidneys. Scand J Urol Nephrol. 2012 Feb; 46(1): 70-2.

[7] Dick L, Hannay J. Enterobius vermicularis presentation during laparoscopic cholecystectomy. Journal of Surgical Case Reports. 2017; 2017(1): rjw239.

[8] Little MD, Cuello CJ, D'Alessandro A. Granuloma of the liver due to Enterobius vermicularis. Report of a case. Am J Trop Med Hyg. 1973 Jul; 22(4): 567-9.

[9] Kaniyur V, Chandra Prasad KH, Devan PP, Doddamani SS, Balachandran B, Kulkarni V. Enterobius vermicularis in the nose: A rare entity. Indian Journal of Otolaryngology and Head & Neck Surgery. 2005; 57(2): 148-150.

[10] Dutta LP, Kalita SN. Enterobius vermicularis in the human conjunctival sac. Indian J Ophthalmol. 1976 Apr; 24(1):34-5. Conjunctival sac.

[11] Babady NE, Awender E, Geller R, et al. Enterobius vermicularis in a 14-Year-Old Girl's Eye . Journal of Clinical Microbiology. 2011; 49(12): 4369-4370.

[12] Anuradha S, Bharathi K, Khalique A. Oculosporidial polyp infected secondarily by *Enterobius vermicularis*. *Advanced Biomedical Research*. 2014; 3: 195.

[13] Dass B. Oxyuris Vermicularis, an Unmentioned Cause of Fistula-In-Ano. *The Indian Medical Gazette*. 1876; 11(7): 193.

[14] Fry GF, Moore JG. Enterobius vermicularis: 10,000-year-old human infection. Science. 1969 Dec 26; 166(3913): 1620.

[15] Paknazhad N, Mowlavi G, Dupouy Camet J, et al. Paleoparasitological evidence of pinworm (*Enterobius vermicularis*) infection in a female adolescent residing in ancient Tehran (Iran) 7000 years ago. *Parasites & Vectors*. 2016; 9: 33.

[16] de Araújo AJ, Ferreira LF, Confalonieri UE, Nuñez L, Ribeiro Filho BM. The finding of Enterobius vermicularis eggs in pre-Columbian human coprolites. Mem Inst Oswaldo Cruz. 1985 Apr-Jun; 80(2): 141-3.

[17] Bharti B, Bharti S, Khurana S. Worm Infestation: Diagnosis, Treatment and Prevention. Indian J Pediatr. 2017 Nov 11.

[18] Ragunathan L, Kalivaradhan SK, Ramadass S, Nagaraj M, Ramesh K. Helminthic infections in school children in Puducherry, South India. J Microbiol Immunol Infect. 2010 Jun; 43(3): 228-32.

[19] Burkhart CN, Burkhart CG. Assessment of frequency, transmission, and genitourinary complications of enterobiasis (pinworms). Int J Dermatol. 2005 Oct; 44(10): 837-40.

[20] Moosazadeh M, Abedi G, Afshari M, Mahdavi SA, Farshidi F, Kheradmand E. Prevalence of Enterobius vermicularis among Children in Iran: A Systematic Review and Meta-analysis. Osong Public Health Res Perspect. 2017 Apr; 8(2): 108-115.

[21] Li H-M, Zhou C-H, Li Z-S, et al. Risk factors for Enterobius vermicularis infection in children in Gaozhou, Guangdong, China. Infectious Diseases of Poverty. 2015; 4: 28. More than 50% prevalence, teeth grinding, jaw clenching Bruxism

[22] Pampiglione S, Rivasi F. Enterobiasis in Ectopic Locations Mimicking Tumor-Like Lesions. International Journal of Microbiology. 2009; 2009: 642481. Ectopic tumor like lesions, difficulties in the histopathological diagnosis of oxyurasis in nonhabitual sites, and their importance from a clinical point of view.

[23] Kılıç S, Ekinci S, Orhan D, Senocak ME. Enterobius granuloma: an unusual cause of omental mass in an 11-year-old girl. Turk J Pediatr. 2014 Mar-Apr; 56(2):189-91.

[24] Elsaid N, Mahmood H, Tekkis P, Tan E. Enterobiasis-related inflammatory caecal polyp masquerading as a malignancy. BMJ Case Rep. 2014 Jan 15; 2014.

[25] Efared B, Atsame-Ebang G, Soumana BM, et al. Acute suppurative appendicitis associated with Enterobius vermicularis: an incidental finding or a causative agent? A case report. BMC Research Notes. 2017; 10: 494. Acute appendicitis/inflammation.

[26] Maraghi S: Enterobius vermicularis in Cerebrospinal Fluid. Iran Biomed J. 1997, 1:49-51.

[27] KAMGARPOUR G., RIAZ MONTAZER S., KAMALI PARVIZ: CORD COMPRESSION SECONDARY TO ENTEROBIUS VERMICULARIS INFESTATION. IRANIAN JOURNAL OF RADIOLOGY. 2003, 1:67-70.

[28] Dutta K., Ghosh S., Basu A. (2016) Infections and Inflammation in the Brain and Spinal Cord: A Dangerous Liaison. In: Jana N., Basu A., Tandon P. (eds) Inflammation: the Common Link in Brain Pathologies. Springer, Singapore.

[29] Chow FC, Marra CM, Cho TA. Cerebrovascular disease in central nervous system infections. Semin Neurol. 2011 Jul; 31(3): 286-306.

[30] Tickell KD, Pavlinac PB, John-Stewart GC, Denno DM, Richardson BA, Naulikha JM, Kirera RK, Swierczewski BE, Singa BO, Walson JL. Impact of Childhood Nutritional Status on Pathogen Prevalence and Severity of Acute Diarrhea. Am J Trop Med Hyg. 2017 Nov; 97(5): 1337-1344.

[31] Walker MD, Zunt JR. Neuroparasitic Infections: Nematodes. Seminars in neurology. 2005; 25(3): 252-261.

[32] Chau TT, Thwaites GE, Chuong LV, et al. Headache and confusion: the dangers of a raw snail supper. Lancet 2003; 361: 1866.

[33] Patikulsila D, Ittipunkul N, Theerakittikul B. Intravitreal angiostrongyliasis: report of 2 cases. J Med Assoc Thai 2003; 86: 981-985.

[34] Boschetti A, Kasznica J. Visceral larva migrans induced eosinophilic cardiac pseudotumor: a cause of sudden death in a child. J Forensic Sci 1995; 40: 1097-1099.

[35] Huff DS, Neafie RC, Binder MJ, et al. Case 4: the first fatal Baylisascaris infection in humans—an infant with eosinophilic meningoencephalitis. Pediatr Pathol 1984; 2: 345-352.

[36] Wachter RM, Burke AM, MacGregor RR. Strongyloides stercoralis hyperinfection masquerading as cerebral vasculitis. Arch Neurol 1984; 41: 1213-1216.

[37] Tsai HC, Liu YC, Kunin CM, et al. Eosinophilic meningitis caused by Angiostrongylus cantonensis associated with eating raw snails: correlation of brain magnetic resonance imaging scans with clinical findings. Am J Trop Med Hyg 2003; 68: 281-285.

[38] Rowley HA, Uht RM, Kazacos KR, et al. Radiologicpathologic findings in raccoon roundworm (Baylisascaris procyonis) encephalitis. AJNR Am J Neuroradiol 2000; 21: 415-420.

[39] Cunningham CK, Kazacos KR, McMillan JA, et al. Diagnosis and management of Baylisascaris procyonis infection in an infant with nonfatal meningoencephalitis. Clin Infect Dis 1994; 18: 868-872.

[40] Brant-Zawadzki M, Wofsy CB, Schechter G. CT-evidence of subarachnoid hemorrhage due to presumed gnathostomiasis. West J Med 1982; 137: 65-67.

[41] Germann R, Schachtele M, Nessler G, et al. Cerebral gnathostomiasis as a cause of an extended intracranial bleeding. Klin Padiatr 2003; 215: 223-225.

[42] Xinou E, Lefkopoulos A, Gelagoti M, et al. CT and MR imaging findings in cerebral toxocaral disease. AJNR Am J Neuroradiol 2003; 24: 714-718.

[43] García A, Nambiar PR, Marini RP, Fox JG. Staphylococcal meningoencephalitis, nematodiasis, and typhlocolitis in a squirrel monkey (Saimiri sciureus). J Med Primatol. 2009 Oct; 38(5): 377-81.

[44] Punyagupta S, Bunnag T, Juttijudata P, Rosen L. Eosinophilic meningitis in Thailand: epidemiologic studies of 484 typical cases and the etiologic role of Angiostrongylus cantonensis. Am J Trop Med Hyg 1970; 19: 950-958.

[45] Hassan T, Kamal MU, Reddy P, et al. Anemia, intractable vomiting, chronic diarrhea, and syndrome of inappropriate antidiuretic secretion: a diagnostic dilemma Disseminated strongyloidosis in a patient with newly diagnosed HTLV infection—case report and review of literature. Medicine 2017; 96 (52): p e9229.

[46] Huttunen P, Lappalainen M, Salo E, Lönnqvist T, Jokela P, Hyypiä T, Peltola H. Differential diagnosis of acute central nervous system infections in children using modern microbiological methods. Acta Paediatr. 2009 Aug; 98(8): 1300-6.

[47] Akya A, Ahmadi K, Zehtabian S, Salimi A, Elahi A, Madani SH. Study of the Frequency of Herpesvirus Infections Among Patients Suspected Aseptic Meningitis in the West of Iran. Jundishapur J Microbiol. 2015; 8(10): e22639. Published 2015 Oct 18.

[48] Thomson RB Jr, Bertram H. Laboratory diagnosis of central nervous system infections. Infect Dis Clin North Am. 2001 Dec; 15(4): 1047-71.

[49] Nordholm AC, Søborg B, Andersson M, Hoffmann S, Skinhøj P, Koch A. CNS infections in Greenland: A nationwide register-based cohort study. PLoS One. 2017; 12(2): e0171094. Published 2017 Feb 3.

[50] Finsterer J, Auer H. Parasitoses of the human central nervous system. J Helminthol. 2013 Sep; 87(3): 257-70.