Journal of the Egyptian Society of Parasitology, Vol. 55, No. 2, August 2025
J. Egypt. Soc. Parasitol. (JESP), 55(2), 2025: 203 - 216
Online: 2090-2549

PARASITES AND EPILEPSY, WITH SPECIAL REFERENCE TO EGYPT: AN OVERVIEW

By

TOSSON A. MORSY[1], TAREK ABDEL KADER SALLAM[2], MOHSEN RAGHEB MOHAMAD[3], and EMAN EBRAHIM ABDEL FADIL[4]

[1]Department of Parasitology, Faculty of Medicine, Ain Shams University, Cairo, 11566, and [2]Consultant of Public Health, [3]Department of Gastroenterology, Air Forces General Hospital, Cairo, *11779*, [4]Consultant of Nursing and Hospital Administration, [2,3,4]Military Medical Academy, Cairo 11911, Egypt
(Correspondence: [1]morsyegypt2014@gmail.com; t.morsy@med.asu.edu.eg; ORCID: org/0000-0003-2799-2049, [2]trksaam35@gmail.com, [3]mohsen.ragheb1@yahoo.com, & [4]emanebraim1970@icloud.com)

Abstract

Epilepsy is a brain condition causes recurring seizures, affects people of all races, sexes, ages, and ethnic backgrounds. About half of all epileptic cases have an unknown cause (idiopathic), structural abnormalities, genetic factors, infections, metabolic issues, immune system problems, and prenatal or perinatal injuries. Besides, bacteria, fungi, parasites and viruses are clinical cause of seizures and epilepsy by inducing the brain inflammation to the epileptogenesis process, which normal neuronal circuits transform into epileptic circuits increasing the probability of epilepsy development.

This overview dealt with the eight zoonotic parasites attack the brain tissues causing epilepsy

Key words: Zoonotic parasites, Epilepsy, An overview.

Introduction

Epilepsy causes are varied and multifactorial, including genetic and acquired etiologies, such as stroke, brain injury, and neurotoxicity (Thomas and Berkovic, 2014). Of the acquired infectious etiologies of epilepsy, bacteria, viruses, fungi, and parasites are the known worldwide causes of seizures (Vezzani *et al,* 2015). Infectious parasites causing epilepsy play a significant role in the burden of neurological disorders worldwide (Sander, 2003). Primary parasitic infections are indeed preventable, but their prevention can be hampered by limited access to resources and the uneven geographic distribution of parasitic agents (Ayeh-Kumi *et al,* 2025).

Review, Discussion and Conclusion

The parasites in alphabetical orders are *Cysticercosus cellulosae* (*Tania solium*), *Onchocerca volvulus*, *Paragonimus westermani*, *Plasmodium falciparum*, *Spirometra mansoni*, *Toxocara canis* & *felis*, *Toxoplasma gondii*, and Trypanosomiasis (African sleeping sickness).

1- *Taenia solium* is one of the most common and recognized parasite causing epilepsy is neurocysticercosis (NCC) infecting CNS. Although NCC infection does not always lead to seizures, patients who are symptomatic constitute a significant proportion of persons with epilepsy in endemic regions of Asia, sub-Sahara Africa, and Latin America (Garcia, 2018). This showed high exposure seroprevalence to multiple parasites infections in low-resource regions, establishing a controversial relation between *T. solium* and epilepsy (Wagner and Newton, 2009). But, albendazole® treated patients with viable parenchymal cysts showed decreased risk of seizure recurrence, supporting the role of NCC in epileptogenic process (Garcia *et al,* 2004). Generally, epilepsy associated with NCC is especially important given the preventable nature of the disease is exacerbated without resources; availability of diagnosis, physicial, surgical and pharmacological treatment options (Reddy and Volkmer, 2017).

Consensus about NCC process causes seizures involves two complexes and intertwined mechanisms of epileptogenesis: structural change to CNS parenchyma (Singh, 2011) caused by the larval cyst reaction to an inflammatory infection (Vezzani *et al,* 2016).

The parasite likely disrupts blood-brain barrier and enables influx of inflammatory cells into the parenchyma, consistent with the finding that NCC patients have higher matrix metalloproteinase (a molecule involved in blood-brain barrier breakdown) compared to the healthy persons (Verma *et al,* 2011). The parasite undergoes unique distinguishable changes within the brain: a viable cyst that does not illicit, much inflammatory response, followed by cyst degeneration around brain edema and, subsequent formation of a granuloma, which sometimes led to the calcified brain lesions (Nash *et al,* 2004). Each infective stage illicit variable inflammatory responses and determines the overall the NCC-associated epilepsy course (Fujita *et al,* 2013).

There is evidence from electroencephalogram (EEG) and imaging studies to suggest that the NCC-related structural lesions in the brain parenchyma and recurrent inflammation may also lead to hippocampal sclerosis and subsequent mesial temporal lobe epilepsy (Rathore *et al,* 2018). But, it is important to note that the dual pathology of NCC-related lesions and hippocampal sclerosis frequently exists, a causal relationship between them was yet established (Del Brutto *et al,* 2016). Typical seizure semiology occurred in NCC, and abnormal EEG with NCC is commonly focally as well, manifesting as slowing and epileptiform discharges. While there could be a clinico-electrographic correlation with the anatomical cyst-associated lesions location (Duque and Burneo, 2017), the electrophysiological abnormalities are not always concordant with the NCC lesions site (Issa *et al,* 2018). NCC patients follow-up showed that seizures occurred both acutely and in chronic phase of the infection and about 50% of persons have seizures recurrence (defined as occurrence of any seizures after a week of index seizure). The persistence of brain lesions on imaging is particularly associated with increased the risk of seizure recurrence (Carpio and Hauser, 2002). Besides, younger and adult persons with a higher cysts burden were more likely had seizures (Kelvin *et al,* 2011). Unlike clinical seizures risk, the electrographic abnormalities didn't depend on lesion burden (Chay asirisobhon *et al,* 1999). NCC persons with associated epilepsy and electrographic abnormalities on EEG were likely having hippocampal atrophy (Del Brutto *et al,* 2021). Electro-clinical data reinforce hypothesis that NCC epileptogenesis were mediated by the hippocampal sclerosis (Nash *et al,* 2015).

In Egypt, *T. solium* & *C. cellulosae* were not common as Muslims don't consume pigs' meat (Youssef and Uga, 2014). But, in Cairo Governmental Abattoir (1994-1997) among 6,434,039 animals *C. cellulosae* was 0.7% (Haridy *et al,* 1999). In Minia Governmental abattoir *C. cellulosae* were (12%) among 100 animals and anti-*T. solium*/*C. cellulosae* antibodies in Assiut & Sohag Governorates in humans were 8.1% & 3.33% respectively (Abdel-Hafeez *et al,* 2015). Mansour *et al.* (2023) in Cairo reported a trapped (locked-in) lateral ventricle human case caused by an isolated *C. cellulosae* trapped at ipsilateral foramen of Monro, an atypical neurocysticercosis site. Alattar *et al.* (2025 in press) rep orted an Egyptian neurocysticercosis *cellulosae* and brain TB patient based on MRI findings and clinical presentation (fever, confusion, fatigue, inability of concentration & cachexia) and elevated ESR, leucopenia and positive Quantiferon-TB test.

2- Onchocerciasis epilepsy due to exposure to the *Simulium*-borne nematode *Onchocerca volvulus* has gained attention (Colebunders *et al,* 2021). Hotterbeekx *et al.* (2020) in Belgium reported that *O. volvulus* neither directly enter the CNS nor caused epilepsy. Kaiser *et al.* (2013) based on African Neurology Database up to May 2012, and Mmbando *et al.* (2018) in Tanzanian children and Mukendi *et al.* (2019) in Congo children found a strong related between *O. volvulus* and epilepsy. Also, Chesnais *et al.* (2018) reported that a dose-response relation was between exposure to *O. volvulus,* and epilepsy development. Levite *et al.* (2020) repo-

rted that the nodding syndrome (NS) is not a known fatal pediatric epilepsy etiology, accompanied by multiple neurological causes, and associated with *O. volvulus*, malnutrition, war-induced trauma, and other insults.

Johnson *et al.* (2020) in USA reported that NS was a rare, but is a manifestation along a continuum of *O. volvulus*-associated neurological complications. Formerly, Abdel Fattah *et al.* (1985) in Sudan reported that *O. volvulvus* caused river blindness. Pearlman and Hall (2000) reported that the *O. volvulus* immune response caused sclerosing keratitis, chorioretinitis, or blindness.

Generalized tonic-clonic seizures were commonly reported semiology associated with *O. volvulus* (Siewe Fodjo *et al,* 2019). Onchocerciasis-subtypes associated epilepsy include nodding syndrome, an epileptic encephalopathy characterized by atonic seizures with repetitive dorso-ventral head drop, syndrome name, and that epidemic NS, in parts of East Africa, showed clinical overlap with sub-Saharan Nakalanga syndrome (NLS), and their etiology was dominated by the environmental factors, including malnutrition, displacement, and nematode infection (Spencer *et al,* 2019). Patients who died with NS and other forms of onchocerciasis-associated epilepsy (OAE) showed similar pathological changes but no generalized tauopathy, suggested that NS and OAE other forms are clinical different presentations of the same disease with a common etiology (Hotterbeekx *et al,* 2019). Epidemiological data strongly showed that onchocerciasis direct or indirect trigger epilepsy (Gumisriza *et al,* 2020)

Clinical epilepsies spectrum in sub-Saharan Africa associated with *O. volvulus* might be greater than previously reported, and that the focal onset tonic-clonic seizures, cortical and cerebellar atrophy are dependable brain imaging and clinical features (Ogwang *et al,* 2021). Morin *et al.* (2021) in Cameroon reported that onchocerciasis may induce neurocognitive disorders and epilepsy via a mechanism that involves mainly the brain frontal and temporal regions. Arndts *et al.* (2023) reported elevated eosinophil in onchocerciasis and epilepsy/nodding syndrome patients' increased esoinophilic catonic protein (ECP) and antigen specific IgG levels as compared to free ones.

Steward (1937) in England reported *O. gutturosa* in cattle. In USA, after five years migration, Sudanese refugee's boys (21%) were *O. volvulus* IgG4 positive (Franco-Paredes *et al,* 2007). In Spain, onchodermatitis were in patients migrated from Equatorial Guinea (Puente *et al,* 2017). In Egypt, none detected *O. vulvolus.* However, Khalil (1939) reported *Onchocerca* eye lesions, Nagaty (1947) detected *O. gibsoni* in Sudan imported cattle, and Steyskal and El-Bahy (1967) detected one *Simulium* spp. El Sheikh *et al.* (1986) in Sudan reported onchocerciasis in three village communities along Bahr El Arab, Southern Darfur. Nasher (1986) in Saudi Arabia detected *O. fasciata* in subcutaneous nodules; anterior parts of local *Camelus dromedarius* L. nuchal ligaments, neck, and shoulders; with general high (59%) but, none showed any symptoms.

Helmy and Al Mathal (2003) in King Abdulaziz University Hospitals diagnosed Sowda (chronic hyperactive) among patients in Asir District. Mahdy *et al.* (2018) in Yemen reported that onchocerciasis was one of the most neglected diseases with baseline of onchocerciasis estimation and the ivermectin impact of regularly administered to patients on its transmission were lacking. Steyskal and El-Bahy (1967) reported one *Simulium* in Upper Egypt. Belqat *et al.* (2018) reported that the greatest simuliid species were in Morocco (44 nominal species), followed by Algeria (34 spp.), Tunisia (18 spp.), Libya (five spp.), and the least was Egypt with one *S. ruficorne* Macquart as a new geographical site. Consequently, is human and animal onchoceriasis a neglected or misdiagnosed filarial nematode parasite in Egypt?

3- Malaria: malaria is another common parasitic disease endemic to so many low- and middle-income countries in sub-Saharan Africa, several regions of south and Southeast

Asia, and South America (Saleh *et al,* 2019). Malaria continues to be a significant global health concern, with an estimated 263 million cases and 597,000 deaths worldwide in 2023. WHO African Region found heaviest burden, accounted 94% of cases and 95% of deaths (WHO, 2024).

Malaria is caused by five *Plasmodium* species; *P. falciparum, P. malariae, P. ovale, P. vivax,* and *P. knowlesi* (CDC, 2024), and transmitted to man by infected female *Anopheles* mosquito bite (WHO, 2020). Patient-to-person transmission is rare, but occurs by blood transfusion, organ transplants, or contaminated needles stick injury (Abdel-Motagaly *et al,* 2017). Also, malaria can be transmitted congenitally from an infected mother to her fetus or during delivery (CDC, 2024). Malaria signs and symptoms may include: fever, chills, general discomfort, headache, nausea, vomiting, diarrhea, abdominal pain, muscle or joint pain, fatigue, cough, and rapid breathing, and rapid heart rate (Mayo Clinic, 2023).

Plasmodium does not directly invade the brain, but remains inside erythrocytes. Parasite-infects RCBs sequestered in the intravascular space causes the perivascular damage, or the first ictogenesis step (White and Ho, 1992). Seizures and other epileptics status are the common neurological manifestations among malaria infected children mainly those with CM (Alizadeh Khatir *et al,* 2023). But, a significant proportion of these manifestations may be the simple febrile seizures (Angwafor *et al*, 2019). The cerebral malaria (CM), characterized by coma and parasitaemia is a life-threatening *P. falciparum* infection (Ngoungou and Preux, 2008), and rarely by *P. vivax* (Mukhtar *et al*, 2019). The changes to neurovasculature and its subsequent effect on the brain parenchyma induces an inflammatory reaction (Löscher *et al,* 2008). Other mechanisms of seizure malaria generation were often squealed of non-neurological systemic malaria effects, such as hypoglycemia, acute metabolic acidosis, and shook (Idro *et al,* 2010). Clinically, seizures commonly occurred during acute phase of both cerebral and uncomplicated malarial form; about 10% of CM survivors develop epilepsy within two years with high fever and acute seizures during CM increased risk of subsequent malaria associated epilepsy (Birbeck *et al,* 2010). Seizures semiology of associated with CM is the typical focal with or without secondary generalization (Carter *et al,* 2010). Also, the status epileptics' frequently occurred and contributed to epileptogenesis (Crawley *al,* 1996). CM is clinical definition as a syndrome characterized by coma at least for one hour after termination of a seizure or correction of hypoglycaemia, asexual forms of *P. falciparum* are on peripheral blood smears, and without any other reasoning for the coma (WHO, 2000).

In the CM-associated epilepsy, EEG is a powerful tool in diagnosis, prognosis, and treatment (Crawley *et al,* 2001). EEG data during hospitalization predicted morbidity, and mortality among malaria infected children (Postels *et al,* 2018). The follow-up of 70 CM children didn't show significant difference in the EEG on visual inspection between those with and without epilepsy, but quantitative spectral analysis showed a risky gamma-delta ratio in the epileptic survivors (Nariai *et al,* 2017). Gamma activity serves as a potential biomarker in CM epileptogenesis, and other epilepsies (Patel *et al,* 2020).

Besides, malignant malaria during pregnancy may cause severe disease in mother, who may lead to premature delivery or delivery of a low-birth-weight baby, on rare occasions, *P. vivax* causes splenic rupture, nephrotic syndrome can result from chronic or repeated *P. malariae* infections, and hyperreactive malarial splenomegaly (tropical splenomegaly syndrome) happens infrequently due to an abnormal immune response to repeated malarial infections (CDC, 2024).

In Egypt, Elgohary and Ibrahim (2022) reported a case of UN Peace Keeping Forces back to Egypt with malaria complicated and splenic infarction. The patient was successfully treated and followed-up.

Undoubtedly, in Egypt the malaria sporadic local and imported human cases and local *Anopheles* vectors and introduced ones were reported by many Egyptian and non-Egyptian authors. The Egyptian ancient DNA based evidence of malaria infection in King Tutankhamun's Mummy indicated that malaria was present there as early as 4000 years ago (Hawass *et al,* 2010). Also, the ancient DNA molecules in sixteen heads' mummified recovered from Fayoum showed *P. falciparum* DNA was in 6/16 samples (Lalremruata *et al,* 2013). Apart from the sporadic cases in Egyptian UN Peace Keeping Forces (Al-Agroudi *et al,* 2018) or employees or Hajj or Omra from Saudi Arabia (El-Bahnasawy *et al,* 2010) back to Egypt. Several malaria epidemics from Sudanese visitors or immigrants occurred (Dahesh and Mostafa, 2015; El-Kady *et al,* 2017; Abo Hashim *et al,* 2018; Mohamed *et al,* 2024). In addition to local malaria vectors (Mikhail *et al,* 2009) 3 *Anopheles* vectors were introduced to Toshka district from chloroquine resistant *P. falciparum* in Sudan (El-Bahnasawy *et al,* 2011). No doubt, all these facts will pave the way to a sudden malaria epidemics or outbreaks.

4- Paragonimiasis: Genus *Paragonimus* contains more than 50 different species (Narain *et al,* 2010). Human paragonimiasis is caused by nine species; *P. africanus, P. heterotremus, P. kellicotti, P. mexicanus, P. skrjabini, P. skrjabini miyazakii, P. siamensis, P. uterobilateralis,* and *P. westermani* but, cerebral or spinal involvements are most commonly caused by *P. westermani* (45%), and also *P. skrjabini, P. s. miyazakii,* and *P. mexicanus* (1% for each) in all paragonimiasis patients (Chai, 2013). Young patients (<18 years) were more likely to have cerebral hemorrhage leading to epilepsy, especially grand mal seizures, headache, visual, motor and sensory disturbances are the five major clinical symptoms (Kim and Lee, 2017).

Most cases with *P. westermani* infection were consistently reported from Asia, Africa and South America due to the cultural dietary customs of ingesting raw or undercooked crustacean harboring the infective metacercariae (Kong *et al,* 2021). Generally, paragonimiasis is significant foodborne zoonosis globally transmitted by consumption of infected uncooked or undercooked crabs, crayfish and snails (Shah *et al,* 2023).

In Egypt, Awadallah and Salem (2015) in Sharkia Governorate reported *P. westermani* in a nomadic dog (0.77%), in a nomadic man and an employee (1.33%). Barduagni *et al*. (2008) recommended triclabendazole, a systemic safe and very effective anthelmintic against many trematodes including *Paragonimus* spp. Abou-Bakr *et al.* (2019) reported that paragonimiasis is one of the most common opportunistic lung fluke among the HIV patients. Morsy *et al.* (2020) gave a detailed review on Jellyfish and sea Jellies in the Red Sea and the Mediterranean Sea, but didn't refer to zoonotic parasitosis. Ali *et al.* (2025) in Saudi Arabia, reported a 41-year-old Filipino driver with a scrotal abscess caused by *P. westermani* suffered from breathlessness, cough productive of yellowish sputum, fever, body aches, and painful scrotal swelling for four weeks.

5- *Spirometra mansoni*: Cestodes of genus *Spirometra*, including *S. erinacei S. mansoni, S. mansonoides, S. ranarum,* and the aberrant *Sparganum proliferum*. *Spirometra* species live in the dogs and cats intestines, pass eggs in stool into water to release coracidia that are ingested by or copepods (first intermediate host) and develop into procercoid larvae. Fish, reptiles and amphibians (second intermediate hosts), ingest infected copepods and procercoid larvae develop into plerocercoid larvae. The cycle is completed when dog or cat eats an infected second intermediate host. Man acquires sparganosis by either drinking water contaminated with infected copepods or consuming the flesh of an under-cooked second intermediate or paratenic host and can live up to 20 years in the human host (CDC, 2017). Also, man is infected by ingesting infected under-cooked frogs or snakes meat, or by put frog or snake poultices on flesh or skin on his/her eyes or

open wounds and/or lesions (Li *et al,* 2011).

The sparganosis was reported sporadically around the world, but higher disease prevalence occurred in several Asian countries, especially South Korea, Japan, Thailand, and China (Nithiuthaia *et al,* 2004). In human body in addition to brain and spine (cerebrospinal sparganosis up to 25.6%), sparganosis can live in eyes, face, neck, limbs, subcutaneous tissues of breast and abdomen, liver, lungs, and kidney (Lin *et al,* 2009). Lotfy (2020) in Egypt added that human sparganosis is a zoonotic infection endemic in many parts of the world. Park and Paek (2025) in Korea reported that brain sparganosis must be suspected in a patient exposure to contaminated water or food, especially if imaging showed migratory lesions.

6- Toxocariasis: Human toxocariasis is a nematode cause blindness or meningoencephalitis (Fakhri *et al,* 2018). Toxocariasis (visceral larva migrans or VLM) is zoonotic infection caused by *T. canis,* or, less commonly by *T. cati* (Morsy, 2020). Children are more prone to be infected via the fecal-oral route from contaminated soil or infected pet dog & cat with up to seropositive 80% in Nigeria (Sowemimo *et al,* 2017). Visceral larva migrans VLM & ocular larva migrans (OLM) clinical presentations, although most infections are asymptomatic, yet in VLM, mostly among preschool children, the larvae invade multiple tissues (mainly liver, lung, heart, or skeletal muscle) cause nonspecific symptoms as fever, myalgia, weight loss, cough, rashes, hepatosplenomegaly accompanied by hypereosinophilia, and migration to CNS causes neurotoxocariasis is uncommon but can cause eosinophilic meningoencephalitis and epilepsy (Morsy e*t al,* 2024).

Apart from toxocariasis VLM, four forms of human VLM are caused by ingestion of pig *Ascaris suum* (Holland, 2017), zoonotic *Ancylostoma* species, restricted to South Africa, causes cutaneous larva migrans (CLM), a serpiginous, pruritic, creeping eruption in days to weeks, but can prolong in *A. braziliense* infections, and *A. caninum* reaches humans' patency (Ngcamphalala *et al,* 2020).

Differential diagnosis: Toxocariasis VLM should be differentially diagnosis from: 1- Strongyloidiasis pulmonary migrated larvae (Berk *et al,* 1987). 2- Ascariasis *lumbricoides* pneumonitis or Loeffler's syndrome (Ramamoorthy, 2015). 3- *Echinococcus granulosus* secondary peritonitis (Moro and Schantz, 2009). 4- Allergic bronchopulmonary aspergillosis (Agarwal, 2009). Also, others parasites cause pulmonary eosinophilia as the clonorchiasis, fascioliasis, gnathostomiasis, opisthorchiasis, paragonimiasis, and schistosomiasis (Kunst *et al,* 2011), and fugus coccidioidomycosis or coccidioides meningitis (Hung *et al,* 2019). Also, 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 (Balmar and Munier, 2007). Symptoms of retinoblastoma in children (eye cancer) are white (leukocoria) or red pupil instead of a normal black, misaligned eyes (strabismus) looking toward ear or nose, reddened, painful eyes, enlarged pupil, different-colored irises, and poor vision (Fang e*t al,* 2020).

7- Toxoplasmosis: Toxoplasmosis caused by *T. gondii* is a zoonotic protozoan parasite of worldwide geographical among all warm-blooded vertebrates, including humans with domestic and wild felids are the definitive hosts (Saleh *et al,* 2016). Humans can be infected with toxoplasmosis either congenital or acquired. Congenital transmission is from an infected mother to her fetus vertically or even after delivery (Abdalla *et al,* 1994). Acquired transmission is either by 1- Ingestion of infectious oocysts from soil contaminated with feline feces, or pet cat, 2- Consumption of unpasteurized goat or equine milk, 3- Ingestion of tissue cysts in infected under cooked meat particularly that of lamb and pork and/or raw vegetables or fruits, 4- Via blood transfusion or organ transplantation from an infected donor, 5- Accidentally by

needle-stick or sharps injuries or even handling with viable infective sample for lab diagnosis (Hussein *et al*, 2022).

Healthy *T. gondii*-infected persons typically remain asymptomatic, but chronic toxoplasmosis among immunodeficiency, such as HIV/AIDS patients, transplant recipients, cancer patients, and others on prolonged immunosuppressive therapy, are at risk of severe, life-threatening infections (Morsy *et al*, 2025), and among COVID-19 patients it increased infection risk (Hamdy *et al*, 2024).

Cerebral toxoplasmosis or neurotoxoplasmosis is the commonest cause of expansive brain abscesses among people living with HIV/AIDS (PLWHA) with high morbidity, and mortality (Vidal, 2019). Clinically, cerebral toxoplasmosis in immunocompetent hosts must be suspected in patients with mental status changes, confusion, delirium, agitation, personality changes, rapidly progressive dementia, myoclonus, and seizures with or without fever, but focal neurologic deficits may be absent (Habek *et al*, 2009). Symptoms of cerebral toxoplasmosis in some patients were initially thought to be due to brain tumor and diagnosis only after surgical removed of brain lesion (Manwani *et al*, 2016).

Diagnosis: The detection of *Toxoplasma* 18S rDNA gene by nested-PCR is useful for diagnosis and safer than a brain biopsy (Matsuura *et al*, 2018).

Differential diagnosis: Toxoplasmosis must be considered among immunocompetent patients presented with severe illness of unknown etiology with pulmonary, cardiac, CNS, or multiorgan involvement/failure, or prolonged febrile illness, even without history of common exposure risk factors or common manifestations of toxoplasmosis (e.g., fever, mononucleosis-like illness, lymphadenopathy, chorioretinitis and others), since prompt initiation of anti-*Toxoplasma* treatment can be lifesaving (Layton *et al*, 2023).

8- Trypanosomiasis: Two subspecies of *Trypanosoma brucei* parasite cause human African trypanosomiasis (HAT) or sleeping sickness. Western African sleeping sickness in central Africa and limited areas of West Africa sleeping sickness are caused by *T. brucei gambiense* (up to 95%, year 2022), and East African sleeping sickness in the Eastern and Southeastern Africa is caused by *T. b. rhodesiense*. Both parasites are only found in sub-Saharan Africa and transmitted by the bite of the tsetse fly (*Glossina* species) that carries these parasites is low (CDC, 2025). *T. b. rhodesiense* infection progressed rapidly, with severe inflammation resulting in hemolytic anemia, and death can occur within 6 months. But, in *T. b. gambiense* infection the course takes years, and is known as Winter-bottom sign, may be particularly conspicuous in the posterior cervical triangle with only 20% of the infected patients can develop CNS involvement after 24 months (Pays *et al*, 2023).

Symptoms of African trypanosomiasis are two stages. The hemolymphatic one is the period when parasites are present in blood and lymphatic system (fevers, headaches, itchiness, and joint pains, one to three weeks after bite), before the central nervous system involvement and neurological stage. The neurological stage, or meningoencephalitic phase, begins when trypanosomes cross the blood-brain barrier and invade CNS (Lundkvist *et al*, 2004). Besides, the hemolymphatic stage neurological symptoms may be present, causing difficult clinical differentiation between both stages (CDC, 2020).

Differential diagnosis: A history of staying in endemic areas, without bleeding diathesis, and encephalopathy in the late stages distinguish African trypanosomiasis from babesiosis, brucellosis, dengue fever, ehrlichiosis, malaria, typhoid fever, viral hepatitis, and yellow fever (Singh *et al*, 2021). Ibrahim *et al.* (2022) in Sudan added that African trypanosomiasis late second stage with neurological complications such as non-convulsive status epilepticus must be suspected in any patient who developed progressive cognitive decline and behavioral changes following long standing history of African Trypanosomiasis and routine.

In Egypt, neither Human African Trypanosomiasis nor *Glossina* species vector was reported due to unfavorable ecological conditions. As to animal trypanosomiasis, Ashour and Gaafar (1997) in Siwah Oasis detected *Trypanosoma mega* in the *Bufo viridis* (83.3%), as a new host, and geographical site. Haridy *et al.* (2011) in Al-Basatin Governomental Abattoir (Greater Cairo) reported that camels were naturally infected with *T. evansi* (Steel, 1885) that causes (Surra), in males was 6% (smears), 8% (ELISA) and 5% (urine thymol turbidity test), and in females was 9%, 24% &12% respectively. Also, they reported zoonotic *T. evansi* in 1/30 owners and/or workers was positive by ELISA and blood smear, but was negative by urine thymol turbidity test. Elhaig *et al.* (2013) in Ismailia reported *T. evansi* in camels. Ashour *et al.* (2013) suggested that sheep & goats played a role in transmission of *T. evansi* to camels.

Host genetics in parasites and epilepsy: Parasites have co-evolved with man; providing selective pressure on his genetics (Fumagalli *et al,* 2009). So, on evaluating the parasitic effect on epileptogenesis and humans' factors must be understood, and evidence of host-genetic susceptibility led to an increased risk of neuronal injury due to the parasitic infection (Thierry *et al,* 2020). Several possibilities were considered: a- host-genetic predisposition increases the risk of a parasitic infection, b- host genetics affect the immune response to a parasite leading to epileptogenesis & c- chronic parasitic infections change the inflammatory milieu and thus lower the seizure threshold in persons who are genetically susceptible to develop seizures as a second hit in epileptogenic process (Clocchiatti-Tuozzo *et al,* 2024).

CM-associated epilepsy patients commonly have first-degree relatives with their family epilepsy (Versteeg *et al,* 2003) A multisite study among four African countries found that polymorphism in genes involved in the inflammatory pathways such as IL-10 were associated with acute seizures (Kariuki *et al,* 2013). Also, Timmann *et al.* (2012) in Ghana identified two previously unknown loci associated with severe malignant malaria in patients and controls and added that the Genome-wide association (GWA) is a must approach to provide candidates for the control measures development against human infectious diseases.

In neurocysticercosis patients, host genetics play an important role in epileptogenesis (Lachuriya *et al,* 2016). Polymorphisms in Toll-like receptor-4, which plays an important role in NCC immune response within the CNS, were associated with seizure recurrence due to NCC (Villegas *et al,* 2019). Jain *et al.* (1999) reported that seizures syndrome association with single lesion seemed to be a benign localization-related epileptic syndrome. They added that human leukocyte antigen (HLA) indicated an inherited susceptibility to an infective agent in most cases was of cysticercal etiology. Benedek *et al.* (2020) dealt with *O. volvulus* suggested that immunogenetic fingerprints in HLA peptide-binding grooves tentatively associate with protection or susceptibility to NS, and thus different HLA molecules explained why in similar environmental factors, only some children within the same families, tribes and areas, developed NS, while others didn't. Undoubtedly, high seizure frequency during severe malaria is an epilepsy predictor, but coma is not a predictor of neurocognitive impairment in these children (Njamnshi *et al,* 2021).

Conclusion

Parasitic etiologies of epilepsy play a significant role in the neurological disorders problem worldwide. While primary parasitic infections are preventable, they are hindered by access to resources along with the disparate geographic abundance of the infectious agents. In addition to primary prevention of parasitic infection, future study efforts must focus on understanding how these infectious agents cause neuronal injury that ultimately leads to epilepsy. Electrophysiological and imaging biomarkers continue to emerge, and individuals who are at-risk of developing pa-

rasite-associated epilepsies are being identified with greater reliability.

Clarifying such pathways may allow the development of novel anti-epileptogenic treatment mechanisms and achieve primary epilepsy prevention.

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

Authors' contribution: All authors equally contributed in data collections, wrote, revised and approved manuscript publication.

References

Abdalla, KF, El Fakahany, AF, Arafa, MAS, Salama, MMI, Morsy, TA, 1994: Congenital toxoplasmosis among premature in Saudi Arabia. J. Egypt. Soc. Parasitol. 24, 3:643-8.

Abdel Fattah, A, Youssif, NM, Shenouda, OA, Morsy, TA, 1985: On the diagnosis of onchocerciasis in the Sudan. J. Egypt. Soc. Parasitol. 15, 2:365-70.

Abdel-Hafeez, EH, Kamal, AM, Abdelgelil, N H, Abdel-Fatah, M, 2015: Parasites transmitted to human by ingestion of different types of meat, El-Minia City, El-Minia Governorate, Egypt. J. Egypt. Soc. Parasitol. 45, 3:671-80.

Abdel-Motagaly, AME, Ibrahim, AMA, Morsy, TA, 2017: An intervention program on blood protozoa acquired by needle stick injury and infection control. JESP 47, 2:309-22

Abo Hashim, AH, Zaalouk, TK, Saad, MY, 2018: Imported malaria in Egypt. Egypt. J. Hosp. Med, 67, 1:455-8.

Abou-Bakr, AA, Ibrahim, NE, Morsy, TA, 2019: Opportunistic parasitic pulmonary infections in HIV infected patients: With references to Egyptian Parasites. JESP 49, 2:423-38

Agarwal, R, 2009: Allergic bronchopulmonary aspergillosis. Chest 135:805-26**.**

Al-Agroudi, MA, Ahmad, GMS, Kondo, MK, Morsy, TA, 2018: Malaria situation in Egypt the last three years: Retrospective study in an Egyptian Fever Hospital. JESP 48, 3:635-44.

Ali, SAM, Ahmed, RI, Deeb, M, Hassan, M, Shahein, M, *et al*, 2025: Rare case of pulmonary paragonimiasis in Jubail city. Egypt. J. Bronchol.19 Article number: 59

Alizadeh Khatir, A, Sepidarkish, M, Daryabari, Y, Taghipour, A, Mollalo, A, *et al*, 2023: Malaria infection and the risk of epilepsy: A meta-analysis. Parasitology 150, 4:382-90.

Angwafor, SA, Bell, GS, Njamnshi, AK, Singh, G, Sander, JW, 2019: Parasites and epilepsy: Understanding the determinants of epileptogenesis. Epilepsy Behav. 92:235-44**.**

Arndts, K, Kegele, J, Massarani, AS, 2023: Epilepsy and nodding syndrome in association with an *Onchocerca volvulus* infection drive distinct immune profile patterns. PLoS Negl. Trop. Dis. 17, 8:e001503

Ashour, AA, Gaafar, NA, 1997: *Trypanosoma mega* (Kinetoplastida) from *Bufo viridis* in Siwah Oasis, Egypt. J. Egypt. Soc. Parasitol. 27, 1: 59-65.

Ashour, AA, Abou El-Naga, TR, Barghash, S M, Salama, MS, 2013: *Trypanosoma evansi*: Detection of *Trypanosoma evansi* DNA in naturally and experimentally infected animals using TBR(1) & TBR(2) primers. Exp. Parasitol. 134, 1:109-14

Ayeh-Kumi, PF,Tetteh. PB, Aryee, INA, Baddoo, PNA, Ocansey, JT, *et al*, 2025: Navigating the parasitic landscape: Insights into infection patterns and public health strategies in West Africa. Trop. Med. Infect. Dis. May 6; 10, 5:125doi: 10.3390/tropicalmed10050125.

Balmer, A, Munier, F, 2007: Differential diagnosis of leukocoria and strabismus, first presenting signs of retinoblastoma. Clin. Ophthalmol. 1, 4:431-9.

Barduagni, P, Hassanein, Y, Mohamed, M, Wakeel, AE, Sayed, ME, *et al*, 2008: Use of triclabendazole for treatment of patients co-infected by *Fasciola* spp. and *S. mansoni* in Behera Governorate, Egypt. Parasitol. Res. 102, 4:631-3.

Belqat, B, Adler, PH, Cherairia, M, Boudghane-Bendiouis, CC, 2018: Inventory of the black flies (Diptera: Simuliidae) of North Africa. Zootaxa 4442, 2:201-20.

Benedek, G, El Latif, MA, Miller, K, Rivkin, M, *et al*, 2020: Protection or susceptibility to devastating childhood epilepsy: Nodding syndrome associates with immunogenetic fingerprints in the HLA binding groove. PLoS Negl. Trop. Dis. 14:1-15.

Berk, SL, Verghese, A, Alvarez, S, *et al*, 1987: Clinical and epidemiologic features of strongyloidiasis: A prospective study in rural Tennessee. Arch. Intern. Med. 147:1257-61.

Birbeck, GL, Molyneux, ME, Kaplan, PW, Seydel, KB, Chimalizeni, YF, *et al*, 2010: Blantyre Malaria Project Epilepsy Study (BMPES) of neurological outcomes in retinopathy-positive

pediatric cerebral malaria survivors: A prospective cohort study. Lancet Neurol. 9:1173-81.
Carpio, A, Hauser, WA, 2002: Prognosis for seizure recurrence in patients with newly diagnosed neurocysticercosis. Neurology 59:1730-4.
Carter, JA, Neville, BGR, White, S, Ross, AJ, Otieno, G, *et al*, 2004: Increased prevalence of epilepsy associated with severe falciparum malaria in children. Epilepsia 45:978-81.
CDC, 2017: Sparganosis. https://www.cdc.gov/dpdx/sparganosis/index.html.
CDC, 2020: Trypanosomiasis-Disease. https://www.cdc.gov/dpdx/monthlycasestudies/html.
CDC, 2024a: Malaria. https://www.cdc.gov/malaria/index.html.
CDC, 2024b: How Malaria Spreads. https://www.cdc.gov/malaria/causes/index.html,
CDC, 2024c: Malaria Clinical Features. https://www.cdc.gov/malaria/hcp/clinical/features/index.html
CDC, 2025: What Causes Sleeping Sickness https://www.cdc.gov/sleepingsickness/causes/index.html.
Chai, JY, 2013: Paragonimiasis. Handb. Clin. Neurol. 114:283-96.
Chayasirisobhon, S, Menoni, R, Chayasirisobhon, W, Locke, GE, 1999: Correlation of electroencephalography and the active and inactive forms of neurocysticercosis. Clin. EEG Neurosci. 30:9-11.
Chen, J, **Chen, Z**, **Lin, J, Zhu, G, Meng, H, *et al*, 2013:** Cerebral paragonimiasis: A retrospective analysis of 89 cases. Clin. Neurol. Neurosurg. 115, 5:546-51.
Chesnais, CB, Nana-Djeunga, HC, Njamnshi, AK, Lenou-Nanga, CG, Boullé, C, *et al*, 2018: The temporal relationship between onchocerciasis and epilepsy: A population-based cohort study. Lancet Infect. Dis. 18:1278-86.
Clocchiatti-Tuozzo, S, Rivier, CA, Misra, S, Zelano, J, Mazumder, R, *et al*, 2024: Polygenic risk of epilepsy and post-stroke epilepsy. Stroke 55, 12:2835-43.
Colebunders, R, Njamnshi, AK, Menon, S, Newton, CR, Hotterbeekx, A, *et al*, 2021: *Onchocerca volvulus* and epilepsy: A comprehensive review using the Bradford hill criteria for causation. PLoS Negl. Trop. Dis. 15:1-24.
Crawley, J, Smith, S, Kirkham, F, Muthinji, P, Waruiru, C, *et al*, 1996: Seizures and status epilepticus in childhood cerebral malaria. QJM Assoc. Physicians 89:591-7.
Crawley, J, Smith, S, Muthinji, P, Marsh, K, Kirkham, F, 2001: Electroencephalographic and clinical features of cerebral malaria. Arch. Dis. Child. 84:247-53.
Dahesh, SMA, Mostafa, HI, 2015: Reevaluation of malaria parasites in El-Fayoum Governorate, Egypt using rapid diagnostic tests (Rdts). J. Egypt. Soc. Parasitol. 45, 3:617-28
Del Brutto, OH, Engel, J, Eliashiv, DS, García, HH, 2016: Update on cysticercosis epileptogenesis: The role of the Hippocampus. Curr. Neurol. Neurosci. Rep. 16:1-7.
Del Brutto, OH, Mera, RM, Wu, S, Recalde, BY, Issa, NP, 2021: Epilepsy, interictal EEG abnormalities and hippocampal atrophy in patients with calcified neurocysticercosis: A population study in an endemic milieu. Epileptic Disord. 23:357-65.
Duque, KR, Burneo, JG, 2017: Clinical presentation of neurocysticercosis-related epilepsy. Epilepsy Behav. 76:151-7.
El Bahnasawy, MM, Dabbous, HKh, Morsy, TA, 2010: Imported malaria as a threat to Egypt. J. Egypt. Soc. Parasitol. 40, 3:773-87.
El-Bahnasawy, MM, Saleh, NMK, Khalil, M F, Morsy, TA, 2011: The impact of three anopheline mosquito species in Toshka, on the introduction of chloroquine resistant *P. falciparum* to Egypt. JESP 41, 3:557-92.
El Sheikh, H, Ghalib, H, Hussein, SMA, Barbiero, V, *et al*, 1986: Onchocerciasis in Sudan: The Southern Darfur foci. Trans. R. Soc. Trop. Med. Hyg. 80, 6:902-5.
El-Kady, A, Abdell, OH, Mostafa, A, Hussein, SM, 2017: Locally transmitted malaria focus in aswan governorate, upper Egypt. J. Adv. Parasitol. 4, 1:23-7.
Elgohary, MA, Ibrahim EA, 2022: Imported malaria patient complicated with splenic rupture and bleedy necessities splenectomy. JESP 52, 3: 543-8.
Elhaig, M, Youssef, AI, El-Gayar, AK, 2013: Molecular and parasitological detection of *Trypanosoma evansi* in camels in Ismailia, Egypt. Vet. Parasitol. 198, 1 & 2:214-8.
Fakhri, Y, Gasser, RB, Rostami, A, Fan, CK, Ghasemi, SM, *et al*, 2018: *Toxocara* eggs in public places worldwide: A systematic review and a meta-analysis. Environ. Pollut. 242, PtB:1467-75.
Fang, X, Wang, H, Ma, X, Guo, Y, Yang, W, *et al*, 2020: Clinical features of children with re-

tinoblastoma and neuroblastoma. J. Ophthalmol. Jul 10;2020:9315784. doi:10.1155/9315784.
Franco-Paredes, C, Dismukes, R, Nicolls, D, *et al*, 2007: Persistent and untreated tropical infectious diseases among Sudanese refugees in the United States. Am. J. Trop. Med. Hyg. 77:633-5.
Fujita, M, Mahanty S, Zoghbi SS, De Ferraris, AM, Hong, J, 2013: Pet reveals inflammation around calcified *Taenia solium* granulomas with perilesional edema. PLoS One 10.1371/journal.pone.0074052.
Fumagalli, M, Pozzoli, U, Cagliani, R, Comi, GP, Riva, S, *et al*, 2009: Parasites represent a major selective force for interleukin genes and shape the genetic predisposition to autoimmune conditions. J. Exp. Med. 206:1395-408.
Garcia, HH, 2018: Neurocysticercosis. Neurol. Clin. 36:851-64.
Garcia, HH, Pretell, EJ, Gilman, RH, Martinez, SM, Moulton, LH, *et al*, 2004: A trial of anti-parasitic treatment to reduce the rate of seizures due to cerebral cysticercosis. N. Engl. J. Med. 350:249-58.
Gumisriza, N, Kaiser, C, Asaba, G, Onen, H, Mubiru, F, *et al*, 2020: Changes in epilepsy burden after onchocerciasis elimination in a hyperendemic focus of western Uganda: A comparison of two population-based, cross-sectional studies. Lancet Infect. Dis. 20:30122-5.
Habek, M, Ozretic, D, Zarkovic K, Djakovic, V, Mubrin, Z, 2009: Unusual cause of dementia in an immunocompetent host: Toxoplasmic encephalitis. Neurol. Sci. 30:45-9.
Hamdy, DA, Eid, RA, Abdel-Tawab, H, El-Badry, MA, Abdallah AM, *et al*, 2024: Impact of latent toxoplasmosis on pneumonic and non-pneumonic COVID-19 patients with estimation of relevant oxidative stress biomarkers. Folia Parasitol. 71:8. doi:10.14411/fp.2024.008.
Haridy, FM, El-Metwally, MT, Khalil, HH, Morsy, TA, 2011: *Trypanosoma evansi* in dromedary camel: With a case report of zoonosis in Greater Cairo, Egypt. J. Egypt. Soc. Parasitol. 41, 1:65-76.
Haridy, FM, Ibrahim, BB, Morsy, TA, Ramadan, NI, 1999: Human taenaisis and cysticercosis in slaughtered cattle, buffaloes and pigs in Egypt. J. Egypt. Soc. Parasitol. 29:375-94.
Hawass, Z, Gad, Y, Ismail, S, Khairat, R, Fathalla, D, *et al*, 2010: Ancestry and pathology in King Tutankhamun's family. JAMA. 303, 7:638-47.
Helmy, MM, Al Mathal, IM, 2003: Human infection with *Onchocerca volvulus* in Asir District (Saudi Arabia). J. Egypt. Soc. Parasitol. 33, 2: 385-90.
Holland, CV, 2017: Knowledge gaps in the epidemiology of *Toxocara*: The enigma remains. Parasitology 144:81-94.
Hotterbeekx, A, Lammens, M, Idro, R, Akun, P, Lukande, R, *et al*, 2019: Neuroinflammation, and not tauopathy is a predominant pathological signature of nodding syndrome. J. Neuropathol. Exp. Neurol. 78:1049-58.
Hotterbeekx, A, Raimon, S, Abd-Elfarag, G, Carter, JY, *et al*, 2020: *Onchocerca volvulus* is not detected in the cerebrospinal fluid of persons with onchocerciasis-associated epilepsy. Int. J. Infect. Dis. 91:119-23.
Hung, CY, Hsu, AP, Holland, SM, Fierer, J, 2019: A review of innate and adaptive immunity to coccidioidomycosis. Med. Mycol. 57, 1:S85-92.
Hussein, EH, Megahed, LA, El Sadany, RAR, Morsy, TA, 2022: An overview needle sticks or sharps related injuries and blood-borne pathogens. JESP 52, 2:353-62.
Idro, R, Marsh, K, John, CC, Newton, CR, 2010: Cerebral malaria: Mechanisms of brain injury and strategies for improved neurocognitive outcome. Pediatr. Res. 68:267-74.
Ibrahim, EAA, Elmahal, MGEM, Ahmed, K AHM, Hasabo, EA, Omer, MEA, 2022: Unusual neurological presentation of second stage African trypanosomiasis in a young boy: A case report. BMC Pediatr. May 12;22(1):265. doi: 10.1186/s12887-022-03313-2.
Issa, NP, Sedler, MJ, Del Brutto, VJ, Darsan, E, Milla, L, *et al*, 2018: EEG patterns in patients with calcified neurocysticercosis with or without hippocampal atrophy. J. Clin. Neurophysiol. 35:232-8.
Jain, S, Padma, MV, Kanga, U, Mehra, NK, Puri, A, *et al*, 1999: Family studies and human leukocyte antigen class II typing in Indian probands with seizures in association with single small enhancing computed tomography lesions. Epilepsia 40:232-8.
Johnson, TP, Sejvar, J, Nutman, TB, Nath, A, 2020: The pathogenesis of nodding syndrome. Ann. Rev. Pathol. Mech. Dis. 15:395-417.
Kaiser, C, Pion, SDS, Boussinesq, M, 2013: Case-control studies on the relationship between onchocerciasis and epilepsy: Systematic review

and meta-analysis. PLoS Negl. Trop. Dis. 10. 1371/journal.pntd. 0002147.

Kariuki, SM, Rockett, K, Clark, TG, Reyburn, H, Agbenyega, T, *et al,* 2013: The genetic risk of acute seizures in African children with *falciparum* malaria. Epilepsia 54:9901001.

Kelvin, EA, Carpio, A, Bagiella, E, Leslie, D, Leon, P, *et al,* 2011: Seizure in people with newly diagnosed active or transitional neurocysticercosis. Seizure 20:119-25.

Khalil, M, 1939: Eye lesions due to *Onchocerca* infection in Egypt. Bull. Soc. Ophtal. Egypte. 32:1-8.

Kim, DW, Lee, SK, 2017: Headache and epilepsy. J. Epilepsy Res. 7, 1:7-15.

Kong, L, Hua, L, Liu, Q, Bao, C, Hu, J, *et al,* 2021: One delayed diagnosis of paragonimiasis case and literature review. Respirol Case Rep. Apr 28; 9, 5:e00750.doi:10.1002/rcr2.750

Kunst, H, Mack, D, Kon, OM, Banerjee, AK, Chiodini, P, *et al,* 2011: Parasitic infections of the lung: A guide for the respiratory physician. Thorax 66, 6:528-36.

Lachuriya, G, Garg, RK, Jain, A, Malhotra, HS, Singh, AK, *et al,* 2016: Toll-like receptor-4 polymorphisms and serum matrix metalloproteinase-9 in newly diagnosed patients with calcif ied neurocysticercosis and seizures. Medicine 17:pe3288.

Lalremruata, A, Ball, M, Bianucci, R, Welte, B, Nerlich, AG, *et al,* 2013: Molecular identification of falciparum malaria and human tuberculosis co-infections in mummies from the Fayum depression (Lower Egypt) PLoS One 8, 4:doi: 10.1371/journal.pone.0060307.

Layton, J, Theiopoulou, DC, Rutenberg, D, Elshereye, A, Zhang, Y, *et al,* 2023: Clinical spectrum, radiological findings, and outcomes of severe toxoplasmosis in immunocompetent hosts: A systematic review. Pathogens Mar 31; 12, 4:543. doi: 10.3390/pathogens12040543.

Levite, M, Zelig, D, Friedman, A, *et al,* 2020: Dual-targeted autoimmune sword in fatal epilepsy: Patient's glutamate receptor AMPA GluR3B peptide autoimmune antibodies bind, induce reactive oxygen species (ROS) in, and kill both human neural cells and T cells. J. Autoimmun. 10. - 1016/journal.jaut.102462.

Li, M-W, Song, H-Q, Li, C, *et al,* 2011: Sparganosis in mainland China: Review. Inter. J. Infect. Dis. 15, 3:E154-6.

Lin, XM, Xu, BL, Liu, CJ, *et al,* 2009: Analysis of 20 human sparganosis caused by eating live tadpoles. China J. Zoonoses 25:1126-7.

Löscher, W, Gernert, M, Heinemann, U, 2008: Cell and gene therapies in epilepsy-promising avenues or blind alleys? Trends Neurosci. 31:62-73.

Lotfy, WM, 2020: Neglected rare human parasitic infections: Part I: Sparganosis review article. PUJ. 13, 1:29-34.

Lundkvist, GB, Kristensson, K, Bentivoglio, M, 2004: Why trypanosomes cause sleeping sickenss. Physiology **19**, 4:198-206**.**

Mahdy, MAK, Abdul-Ghani, R, Abdulrahman, TAA, Al-Eryani, SMA, Al-Mekhlafi, AM, *et al,* 2018: *Onchocerca volvulu*s infection in Tihama region, west of Yemen: Continuing transmission in ivermectin-targeted endemic foci and unveiled endemicity in districts with previously unknown status. PLoS Negl. Trop. Dis. 12, 3:e0-006329. doi: 10.1371/j.pntd. 0006329.

Mansour, M, Tahir, M, Ahmadi, Z, 2023: Neurocysticercosis presenting as a locked-in lateral ventricle: A case report, and evidence-based review. IDCases 32:e01778. doi:10.1016/journal. idcr.e01778.

Manwani, N, Ravikumar, K, Viswanathan, V, Rao, SM, Mahadevan, A, 2016: Acquired toxoplasmosis presenting with a brainstem granuloma in an immunocompetent adolescent. Indian Pediatr. 53: 159-61.

Matsuura, J, Fujii, A, Mizuta, I, Norose, K, Mizuno, T, 2018: Cerebral toxoplasmosis diagnosed by nested-polymerase chain reaction in a patient with rheumatoid arthritis. Intern. Med. 57, 10:1463-8.

Mayo Clinic, 2023: Malaria: Symptoms & causes. https://www.mayoclinic.org/diseases-conditions/malaria/symptoms-causes/syc-20351184.

Mikhail, MW, Al-Bursheed, KM, Abdel Halim, AS, Morsy, TA, 2009: Studies on mosquito borne diseases in Egypt and Qatar. J. Egypt. Soc. Parasitol. 39, 3:745-56.

Mmbando, BP, Suykerbuyk, P, Mnacho, M, Kakorozya, A, Matuja, W, *et al,* 2018: High prevalence of epilepsy in two rural onchocercias is endemic villages in the Mahenge area, Tanzania, after 20 years of community directed treatment with ivermectin. Infect. Dis. Poverty. Jun 20; 7, 1:64. doi:10. 1186/s40249-018-0450-3.

Mohamed, SS, Abdou Seleem, M, Etewa, SE, Mohamed, AS, 2024: Prevalence study of malaria among travelers coming from endemic areas

to Egypt ZUMJ, 30:959-69
Moro, P, Schantz, PM, 2009: Echinococcosis: A review. Int. J. Infect. Dis. 13, 2:125-33.
Morin, A, Guillaume, M, Ngarka, L, Tatah, G Y, Siewe Fodjo, JN, *et al,* 2021: Epilepsy in the SanagaMbam valley, an onchocerciasis-endemic region in Cameroon: Electro-clinical and neuropsychological findings. Epilepsia Open 6, 3: 513-27**.**
Morsy, TA, 2020: Toxocariasis: Visceral and ocular larva migrans. JESP 50, 1:41-8**.**
Morsy, TA, Shoukry, NM, Fouad, MA, 2020: Jellyfish stings complications and management. JESP 50, 2:270-80.
Morsy, TA, Alqurashi, YM, Ozbak, HA, 2024: Toxocariasis visceral and ocular larva migrans: Is it still a neglected zoonotic disease? JESP 54, 2:213-26.
Morsy, TA, Mohamad, MR, Metwally, SFA, Sallam, TA, Alattar, MBR, Elsherif, AAI, 2025: An overview on toxoplasmosis in HIV-infected patients. JESP 55, 1:65-76.
Mukendi, D, Tepage, F, Akonda, I, Siewe, JN F, Rotsaert, A, *et al,* 2019: High prevalence of epilepsy in an onchocerciasis endemic health zone in the Democratic Republic of the Congo, des pite 14 years of community-directed treatment with ivermectin: A mixed-method assessment. Int. J. Infect. Dis. 79:187-94.
Mukhtar, MM, Eisawi, OA, Amanfo, SA, Elamin, EM, Imam, ZS, *et al,* 2019: *Plasmodium vivax* cerebral malaria in an adult patient in Sudan. Malar. J. 18:1-3.
Nagaty, HF, 1947: *Onchocerca gibsoni* among the imported cattle. Vet. Rec. 59:145-7.
Narain, K, Agatsuma, T, Blair, D, 2010: *Paragonimus.* In: Molecular Detection of Foodborne Pathogens (Liu, D, Ed.). Boca Raton, Florida, USA: CRC Press Taylor & Francis Group.
Nariai, H, Beal, J, Galanopoulou, AS, Mowrey, WB, Bickel, S, *et al,* 2017: Scalp EEG Ictal gamma and beta activity during infantile spasms: Evidence of focality. Epilepsia 58:882-92.
Nash, TE, Del Brutto, OH, Butman, JA, *et al,* 2004: Calcific neurocysticercosis and epileptogenesis. Neurology 62:1934-8.
Nash, TE, Mahanty, S, Loeb, JA, *et al,* 2015: Neurocysticercosis: A natural human model of epileptogenesis. Epilepsia 56:177-83.
Nasher, AK, 1986: Incidence and intensity of *Onchocerca fasciata* Railliet and Henry, 1910 in local camels in Saudi Arabia. Ann. Parasitol. Hum. Comp. 61, 1:77-80
Ngoungou, EB, Preux, PM, 2008: Cerebral malaria and epilepsy. Epilepsia 49:19-24.
Ngcamphalala, PI, Lamb, J, Mukaratirwa, S, 2020: Molecular identification of hookworm isolates from stray dogs, humans and selected wildlife from South Africa. J. Helminthol. 94. doi: 10.1017/S00- 22149X19000130**.**
Nithiuthaia, S, Anantaphrutib, MT, Waikagulb, J, *et al,* 2004: Waterborne zoonotic helminthiases. Vet. Parasitol. 126:167-93.
Njamnshi, A, Agbor, CA, Evelyn Mah, E, Prosper Sayong, P, Ruffieux, N, *et al,* 2021: Neur ocognitive impairment and epilepsy in school-aged children after severe malaria in a malaria endemic area. J. Neurol. Sci. 429, 68. http://dx. doi.org/10.1016/j.jns.2021.118932
Ogwang, R, Ningwa, A, Akun, P, Bangirana P, Anguzu R, *et al,* 2021: Epilepsy in *Onchocerca volvulus* sero-positive patients from northern Uganda: Clinical, EEG and brain imaging features. Front Neurol. Jun 3; 12: 687281. doi: 10.3389/fneur.2021.687281.
Park, HR, Paek, SH, 2025: Sparganosis of the brain. Neurol. J. **104, 1:** https://doi.org/10.1212/ WNL 0000000000210219
Patel, AA, Jannati, A, Dhamne, SC, Sapuwa, M, Kalanga, E, *et al,* 2020: EEG markers predictive of epilepsy risk in pediatric cerebral malaria: A feasibility study. Epilepsy Behav. 10.1016/j.yebeh. 2020.107536.
Pays, E, Radwanska, M, Magez, S, 2023: The pathogenesis of African trypanosomiasis. Ann. Rev. Pathol. 18:19-45.
Pearlman, E, Hall, LR, 2000: Immune mechanisms in *Onchocerca volvulus*-mediated corneal disease (river blindness). Parasite Immunol. 22, 12:625-31.
Postels, DG, Wu, X, Li, C, Seydel, KB, *et al,* 2018: Admission EEG findings in diverse paediatric cerebral malaria populations predict outcomes. Malar J. 17: 10.1186/s12936-018-2355-9.
Puente, S, Ramirez-Olivencia, G, Lago, M, Perez-Blazquez, E, *et al,* 2017: Dermatological manifestations in onchocerciasis: a retrospective study of 400 imported cases. Enferm Infect. Microbiol. Clin. 20: 30380-4.
Ramamoorthy, KG, 2015: Anaesthesia and *Ascaris* pneumonia (Loeffler's syndrome). Indian J. Anaesth. 59, 2:125-6.
Rathore, C, Thomas, B, Kesavadas, C, Radhakrishnan, K, 2018: Calcified neurocysticerco

sis lesions and hippocampal sclerosis: Potential dual pathology? Epilepsia 10.1111/j.1528-1167. 2011.03386.

Reddy, DS, Volkmer, R, 2017: Neurocysticercosis as an infectious acquired epilepsy worldwide. Seizure 52:176-81.

Richard-Lenoble, D, al Qubati, Y, Toe, L, *et al,* 2001: Human onchocerciasis and "sowda" in Yemen. Bull. Acad. Natl. Med. 185, 8:1447-61.

Saleh, AMA, El Nakib, MM, Malek, DMA, Morsy, TA, 2019: Malaria and human imunodeficiency virus (HIV) in Republic of South Sudan: A mini-overview. JESP 49, 2:313-20

Saleh, AMA, Al-Agroudi, MA, Morsy, TA, 2016: Occupational, nosocomial or hospital acquired toxoplasmosis. JESP 46, 2:407-18.

Saleh, AMA, El Nakib, MM, Malek, DMA, Morsy, TA, 2019: Mini-review on malaria and human immunodeficiency virus in Sub-Sahara Africa. J. Egypt. Soc. Parasitol. 49, 1:61-72

Sander, JW, 2003: The epidemiology of epilepsy revisited. Curr. Opin. Neurol. 16:1650-70.

Shah, P, Sah, R, Pradhan, S, Bhandari, P, Baral, R, *et al,* 2023: Pulmonary paragonimiasis: A case series. JNMA J Nepal Med Assoc. 61, 259:290-3.

Siewe Fodjo, JN, Mandro, M, Mukendi, D, *et al,* 2019: Onchocerciasis-associated epilepsy in the Democratic Republic of Congo: Clinical description and relationship with microfilarial density. PLoS Negl. Trop. Dis. 13, 7:e0007300.

Singh, P, 2011: Infectious causes of seizures and epilepsy in the developing world. Dev. Med. Child. Neurol. 53:600-9.

Singh, G, Njamnshi, AK, Sander, JW, 2021: Vector-borne protozoan infections of the CNS: Cerebral malaria, sleeping sickness and Chagas disease. Curr. Opin. Neurol. 34, 3:439-46.

Sowemimo, OA, Lee, YL, Asaolu, SO, Chuang, TW, Akinwale, O, *et al,* 2017: Seroepidemiological study and associated risk factors of *Toxocara canis* infection among preschool children in Osun State, Nigeria. Acta Trop. 173:85-89.

Spencer, PS, Mazumder, R, Palmer, VS, Pollanen, MS, 2019: Nodding syndrome phenotypes. Rev. Neurol. (Paris) 175:679-85.

Steward, JS, 1937: Occurrence of *Onchocerca gutturosa* Neumann in cattle in England, with an account of its life history & development in in-*Simuliuin ornatum* Mg. Parasitology 29:212-9.

Steyskal, GC, El-Bahy, S, 1967: A List of Egyptian Diptera with a Bibliography & Key to Families. UAR, Ministry of Agriculture. Tech. Bull. No. 3:1-87.

Thierry, A, Falilatou, A, Covalic, B, *et al,* 2020: Epilepsy and malaria in children aged 1 to 15 years in Parakou in 2018: Case-control study. Child. Neurol. Open. 7:2329048X2095411.

Thomas RH, Berkovic SF, 2014: The hidden genetics of epilepsy: A clinically important new paradigm. Nat. Rev. Neurol. 10:283-92.

Timmann, C, Thye, T, Vens, M, Evans, J, **May, J, *et al,* 2012:** Genome-wide association study indicates two novel resistance loci for severe malaria. Nature 489, 7416:443-89.

Verma, A, Prasad, KN, Nyati, KK, Singh, SK, Singh, AK, *et al,* 2011: Association of MMP-2 and MMP-9 with clinical outcome of neurocysticercosis. Parasitology 138:1423-8.

Versteeg, AC, Carter, JA, Dzombo, J, Neville, BG, Newton, CRJ, 2003: Seizure disorders among relatives of Kenyan children with severe *falciparum* malaria. Trop. Med. Int. Heal. 8:12-16.

Vezzani, A, Fujinami, RS, White, HS, Preux, PM, Ingmar Blümcke, I, *et al,* 2015: Infections, inflammation and epilepsy. Acta Neuropathol. 131, 2:211-34.

Vezzani, A, Fujinami, RS, White, HS, Preux, PM, *et al,* 2016: Infections, inflammation and epilepsy. Acta Neuropathol. 131: 211-34.

Vidal, JE, 2019: HIV related cerebral toxoplasmosis revisited: Current concepts and cotroversies of an old disease. J. Int. Assoc. Provid. AIDS Care Jan-Dec; 18:2325958219867315.

Villegas, M, Sciutto, E, Rosetti, M, Fleury, A, *et al,* 2019: Association of TRAF1/C5 locus polymorphisms with epilepsy and clinical traits in Mexican patients with neurocysticercosis. Infect. Immun. 87, 12:e00347-19.

Wagner, RG, Newton, C, 2009: Do helminthes cause epilepsy? Parasite Immunol. 31:697-705.

White, NJ, Ho, M, 1992: The pathophysiology of malaria. Adv. Parasitol. 31:83-173.

WHO, 2000: Severe *falciparum* malaria. Communicable diseases cluster. Trans. R. Soc. Trop. Med. Hyg. 94, 1:S1-90.

WHO, 2020: World Malaria Report. Geneva.

WHO, 2024: Malaria https://www.who.int/news-room/fact-sheets/detail/malaria.

Youssef, AI, Uga, S, 2014: Review of parasitic zoonoses in Egypt. Trop. Med. Hlth. 42, 1:3-14.