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NEUROCYSTICERCOSIS: THE FIRST CASE REPORT IN CYPRUS

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Abstract

Taenia solium or the pork tapeworm belongs to the cyclophyllid cestode (family Taeniidae). It is found worldwide and is most common in countries where pork is eaten. Infection can cause two distinct diseases: taeniasis and cysticercosis larvae (*Cysticercus cellulosae*). C. *cellulosae* are fluid-filled cystic structures of a thin bladder wall and parenchymatous portion with a single invaginated scolex surrounded by a convoluted spiral canal.

This is reported neurocysticercosis in an African immigrant to the Republic of Cyprus.

Key words: First report, Neurocysticercosis, Cyprus, African immigrant patient.

Introduction

Neurocysticercosis, caused by cestode worm Taenia solium, is one of the commonest zoonotic parasite of the central nervous system (CNS) with 30% of seizures in endemic countries (Adjei et al, 2024), with >50 million patients globally, with 50,000 annual deaths (Nyangi et al, 2024). CDC (2019) reported that the cysticercosis cellulose mainly infects countries in the African Sub-Sahara, Asia (India, China, & Nepal) and Latin America (Guatemala, Nicaragua, & El Salvador). Also, cysticercosis was detected in some Arab Countries including Egypt (Morsy et al, 2024). The cysticercosis cellulosae may be present in more than one site in patients with different stages in natural history simultaneously, who at any time may have some viable C. cellulosae enhancing and/or calcified cyst, the usual involved organs are subcutaneous tissue, skeletal muscles, lungs, brain, CNS, eyes, liver and heart (CDC, 2024).

Man acquired infection by the ingestion of food/water contaminated with pig feces contained eggs or proglottids, or by person-to-person spreading, or autoinfection can occur if a patient ingests his own eggs fecal-orally (WHO, 2022). Once man is infected, the oncospheres hatch in the intestine, invade intestinal wall, enter the bloodstream, and migrate to multiple sites in tissues and organs to mature into the cysticerci within 60-70 days (Haridy *et al*, 1999).

Symptomatic parenchymal NCC occurs 3 to 5 years up to >30 post infection; in delayed one with nonviable or calcified lesions patients suffered from seiz-ures and/or headache (Coyle *et al*, 2012). Seizures are the most common clinical picture, focal neurologic and meningitis, but less common is altered vision (Wilson *et al*, 2018).

Cysticercosis diagnosis are based on clinical manifestations; mainly seizures, altered vision, focal neurologic & meningitis (Barrie *et al*, 2020), neuro-imaging such as CT scan, MRI, MTR, MRS (Pretell *et al*, 2005), serologically such as WB, ELISA, MBA, qPCR (O'Connell *et al*, 2020).

Treatment consists of CSF diversion by ventriculoperitoneal shunt (rather than cyst removal with risky complications), followed by albendazole 15mg/kg/day in 2daily doses up 1200mg/day with food for 10 to 14 days and anti-inflammatory therapy (Kelley *et al*, 2002) In patients with extraparenchymal disease, surgical intervention, along with postoperative anti-inflammatory therapy to remove lesions or to relieve elevated intracranial pressure as a result of hydrocephalus (Filho *et al*, 2022).

The case presentation: A 27-year-old African immigrant to Cyprus a year ago with hemodynamically stable, afebrile, and fully alert and oriented, responded coherently to the questions. His past medical history was unremarkable, didn't use any medication and

clinical examination showed no abnormal findings. He was admitted to the Emergency Department after a first-time, sudden-onset generalized tonic-clonic seizure while asleep.

Initial laboratory examination showed mild leukopenia (2,910cells/μl.) and mild neutropenia (1.06cells/μl.), but without any significant pathological findings. Non-contrast brain CT scan showed multiple cystic lesions with 5mm in diameter scattered among the white matter, cerebral cortex, and basal ganglia. Bilateral X-ray of femur, tibia, and fibula showed mild soft tissue edema, but no rice grain calcifications (muscle fiber calcification). Cerebrospinal fluid (CSF) analysis showed clear fluid with one white blood cell, three red blood cells and culture didn't show growth. CSF glucose levels were 66 mg/dL (serum 79mg/dL) and proteins 54.4 mg/dL. The neutropenia along with the CT scan results prompted to serologic examinations for HIV, echinococcosis, toxocarosis, and toxoplasmosis, but all were negative except a positive Toxoplasma IgG. The subsequent brain MRI with IV gadolinium proved the intraparenchymal neurocysticercosis diagnosis by numerous well-dispersed cystic lesions, with contrast uptake, in both cerebral hemispheres, predominantly involved the deep white matter, cortex, and basal ganglia (figures). Most cysts were in stages 1-2, with active inflammation and edema in specific regions, and some cysts showed clear scolices. Screening for latent tuberculosis and strongyloidiosis was done, prior to the antiinflammatory therapy (dexamethasone), but were negative. The patient was examined by the ophthalmologist, but neither he had ocular cysticerci nor elevated ICP.

On neurocysticercosis diagnosis, the patient was treated for 14-day course of antiparasitic therapies, albendazole (15mg/kg/day), and praziquantel (50mg/kg/day). Dexamethasone was given as anti-inflammatory therapy (parasite death by treatment causes an inflammatory response led to brain edema).

Antiepileptic therapy, with levetiracetam,

was started on admission (before confirmed diagnosis) and was continued for the seizure prophylaxis. The patient was cured and he was followed up to six-months without any seizure recurrence.

Discussion

The first introduced neurocysticercosis case to Cyprus, a *Taenia solium* free country. This highlights health problem risky of migrations, and global climate changes (Almeida-Coral *et al*, 2015, Economides 2000). Fischer (2024) reported that the African immigrants are the second largest group of asylum seekers in Cyprus in 2022, moreover increase the risk of transmission of food-borne disease (FBD), especially *Taenia solium*. Eruaga (2024) added that the global climate change not only increased prevalence and incidence of vector-borne diseases (VBD), but also the zoonotic food borne diseases.

Galipo *et al.* (2021) reported that *Taenia* solium eggs can survive up to a year in the favorable environment conditions (5-25°C Celsius and elevated humidity levels).

Generally, Cissé (2019) in Switzerland reported that the highest burden/population of water and food-borne diseases is common in the African low- & middle-income countries, followed by the Southeast Asia and the Eastern Mediterranean subregions, where unsafe water used for cleaning, and food processing is a key risk factors led to foodborne diseases. Habibullah et al. (2022) in Malaysia studied the climatic changes impact on biodiversity loss of the amphibians, birds, fishs, mammals, snails, plants, and reptiles from 115 countries. They found that higher economic development positively impacted on biodiversity loss, and practicing good governance, promoting conservation of the environmental and greenhouse gasses control would mitigate the biodiversity loss. Morsy et al. (2024) in Egypt reported that the global climatic changes already made the conditions more suitable for the spreading of risky vector-borne epidemic diseases to the non-endemic countries. Also, El-Bahnasawy et al. (2025) reported that the world is facing a process of climate changes, with adversely impact on human health in many different ways, such as increasing arthropod-borne diseases, psychiatric disorders, cancers and other microbial parasitic and viral diseases.

Conclusion

There must be local, national and international collaboration to face the climatic changes impact on man, animal and agriculture.

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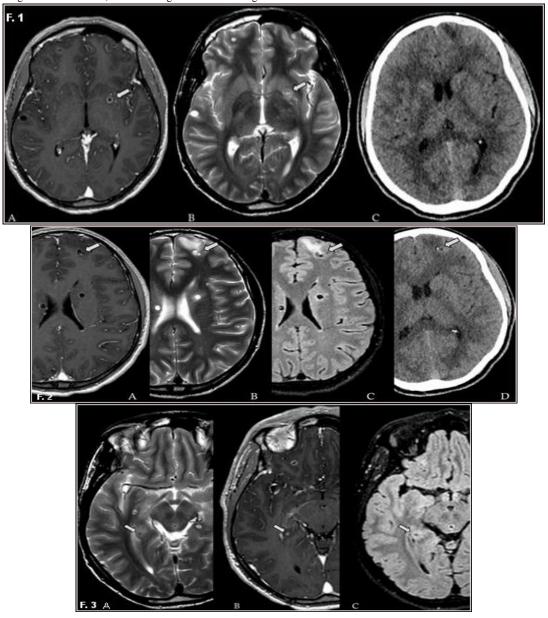
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Explanation of figurers

Fig. 1: A- Post contrast T1W showed a small cyst in left putamen (arrow) with subtle peripheral enhancement. B- T2W CSF-like signal within cyst with neither central dot nor calcification on CT. C- radiological showed a cyst likely in vesicular- early colloidal vesicular stage Fig. 2: Subcortical cyst in left frontal lobe, with thin peripheral enhancement with an eccentric enhancing focus (cyst with dot sign) on post contrast T1W. A- CSF-like signal with peri-lesion edema on T2W and FLAIR (C), and hyper-density of eccentric enhancing focus on CT D-with a visible scolex.

Fig. 3: A focal lesion in right parahippocampal gyrus with hypo-intensity on T2W, A-focal enhancement on post contrast T1W, B- subtle surrounding oedema on FLAIR, C with more granular nodular stage.



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