Journal of Neurological Sciences and Research

Genesis-JNSR-4(1)-34
Volume 4 | Issue 1
Open Access

Case Study on Neurocysticercosis

Kunal Joon*
University: NIIMS greater Noida, India

***Corresponding author:** Kunal Joon, University: NIIMS greater Noida, India.

Citation: Joon K. Case study on Neurocysticercosis. J Neurol Sci Res. 4(1):1-4.

Received: May 16, 2024 | **Published**: June 13, 2024

Abstract

The etiology of this condition is a helminth parasite Taenia solium, which has a cycle eventually affect the human host. This condition follows a cycle of human-to-human fecal-oral route and pigs as an intermediate host with transmittedto humans. Infected humans can get dissemination of the larva through circulation and spread out in the body. In this case, the neural tissue was affected.

Keywords

Host; Parasite; Transmission; Infection; Neurocysticerosis

Introduction

Taenia solium is a larval cyst of the tapeworm that affects a significant number of patients around the world. This condition is transmitted through infected individual with cysticercosis to a new host through fecal-oral route. In low-income countries, the close proximity of individuals in the same household has a higher incidence of larval [1] transmission. Fecal transmission and proximity of humans to the pigs also lead to secondary infections in pigs. Consumption of undercooked pigs subsequently transmits the tapeworm to a new human host and the cycle continues. In the case of [2]. Neurocysticercosis, the larvae circulate in the blood and enter the nervous system. The eggs create cysts in different organs leading to a range of conditions from mild to severe and at times lethal (neural cyst) [3].

Neurocysticercosis could present as an intra-parenchymal or an extra-parenchymal lesion. The location of the lesion can have a significant difference in symptoms [4]. treatment, and prognosis. In the case of intra-parenchymal involvement, the embryos access the gray matter via arteries. Once in place, they grow as a cystic fluid-filled mass surrounded by brain tissue. In most patients there is an equilibrium present with no [5]. symptoms; however, over time, the cyst will damage the surrounding tissue leading to characteristic seizure activity. These cysts may be dormant for years and they may incidentally be detected on imaging .Eventually, the host's immune system overcomes the homeostasis and the larva dies. The released larva particles generate a significant immune response with various symptoms depending on the presence of cyst. The lesion calcifies and leaves a residual calcification in the brain parenchyma. Most of these cysts have benign prognoses with minimal host symptoms. At times, there may [6]. be multiple simultaneous lesions, and this condition is known as cysticercotic encephalitis [7].

The extra-parenchymal lesion can involve subarachnoid space, or ventricles. The enlargement of the cyst in these spaces can obstruct the CSF fluid flow leading to an increase in the intracranial pressure and possible death from herniations. This report presents a case of a patient who presented to the clinic with an extra-parenchymal lesion which was later diagnosed as neurocysticercosis [8].

Case Presentation

A case of a 65-year-old female who presented to the emergency room complaining of a recent headache. Her history is significant for blindness diagnosed secondary to retinitis pigmentosa three years ago. At the time of the presentation, she complained of bilateral vertex headache. The family reported a history of being confused and forgetful at times. She also complained of some vague symptoms of lower left arm numbness. She has no history of upper- or lower-extremity weakness or gait issues. Her past medical history was also significant for type 2 diabetes and a history of hysterectomy. Her medication included amlodipine, atorvastatin, clotrimazole cream, hydrochlorothiazide, indomethacin, sitagliptin, melatonin, and Premarin. On presentation, she was afebrile at 37oc with stable vital signs and a BMI of 36. She appears older than the stated age. She was awake and alert and in no distress. She communicated appropriately and obeyed commands. Blood serology was negative. She was seen by neurosurgery and sent to obtain a computed tomography (CT) scan, which revealed a hyper-dense tumor in the trigone of the left lateral ventricle that measured 30.2 × 17.3 mm. There appeared to be scattered calcifications within the lesion. The mass was blocking the egress of spinal fluid from the left temporal horn and the left occipital horn and the mass was enlarged (Figure1-3).

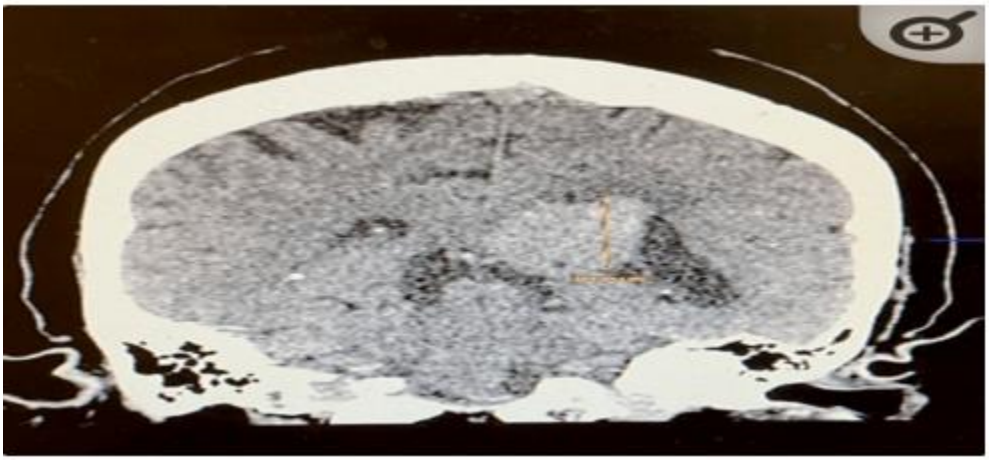

Figure 1: Signs of calcificationand mass lesion seen.

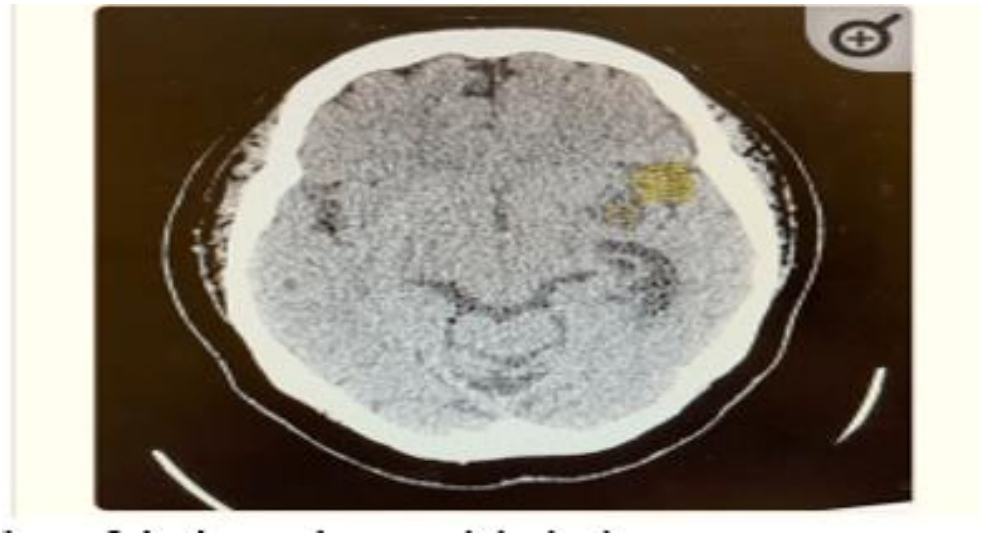

Figure 2: CT scan showing axonal view of lesion.

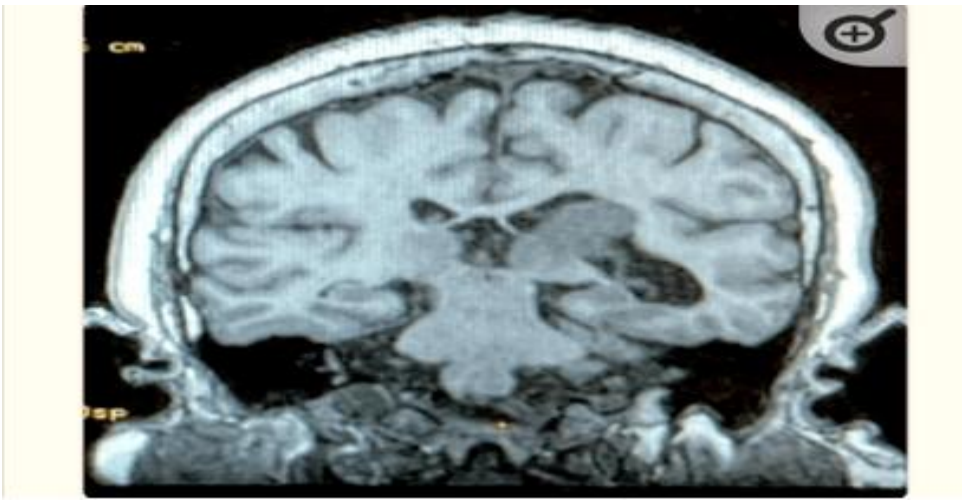

Figure 3: It shows the ventricle lesion.

Differential diagnosis at the time included ependymoma, meningioma, or glioma and possible infections. The subsequent visit to the neurosurgery clinic revealed improvement in the patient's symptoms although the headache now was localized to the occipital region. Her neurologic examination was otherwise negative. The presence of a mass with calcification and the location of the patient's birth placed neurocysticercosis high on the list. Serologic testing with enzyme-linked immunosorbent assay (ELISA). Subsequent visits revealed the lesion to be stable and planned for a one-year follow-up with an MRI scan.

Discussion

The case was the neurocysticercosis and this study shows the signs and symptoms of neurocysticercosis and diagnosis and rule out of different diagnosis possible and mechanism of the occurrence of neurocysticercosis and how calcifications mass formed in the head.

Human Ethics

Consent was obtained from patient to participate in the study.

References

1. Moyano LM, Saito M, Montano SM, Gonzalvez G, Olaya S, et al. (2014) Neurocysticercosis as a cause of epilepsy and seizures in two community-based studies in a cysticercosis-endemic region in Peru. PLoS Negl Trop Dis. 8(2):e2692.
2. Amaral L, Maschietto M, Maschietto R, Cury R, Lima SS, et al. (2003) Ununsual manifestations of neurocysticercosis in MR imaging: analysis of 172 cases. Arq Neurostimulator. 61(3A):533-41.
3. Pal DK, Carpio A, Sander JW. (2000) Neurocysticercosis and epilepsy in developing countries. J Neurol Neurosurg Psychiatr. 68(2):137-43.
4. Suri A, Goel RK, Ahmad FU, Sharma BS, Mahapatra AK, et al. (2008) Endoscopic excision of intraventricular neurocysticercosis in children: a series of six cases and review. Childs Nerv Syst. 24(2):281-5.
5. Proano JV, Torres-Corzo J, Vecchia RRD, Guizar-Sahagun G, Rangel-Castilla L. (2009) Intraventricular and subarachnoid basal cisterns neurocysticercosis: a comparative study between traditional treatment versus neuroendoscopic surgery. Childs Nerv Sys. 25(11):1467-75.
6. Singh G, White AC, Coyle CM, Rajshekhar V, Nash TE. (2018) Diagnosis and Treatment of Neurocysticercosis: 2017 Clinical Practice Guidelines by the Infectious Diseases Society of America (IDSA) and the American Society of Tropical Medicine and Hygiene (ASTMH). Clin Infect Dis. 66(8):e49-75.
7. Sarti E, Schantz PM, Avila G, Ambrosio J, Flisser A. et al. (2000) Mass treatment against human taeniasis for the control of cysticercosis: a population-based intervention study. Trans R Soc Trop Med Hyg. 94(1):85-9.