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Case Report

Neurocysticercosis as an Unrecognized Cause of Sudden Death: A Case-Based Study

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ABSTRACT

Neurocysticercosis (NCC) is a leading global cause of adult-onset epilepsy and the most common parasitic infection of the central nervous system. It is caused by the larval stage of the pork tapeworm, *Taenia solium*, which encysts in the brain after the ingestion of eggs. While often presenting with chronic symptoms, NCC can lead to sudden, unexpected death through acute neurological or cardiac complications. Forensic awareness and a high index of suspicion are critical for identifying subclinical infections in at-risk populations. This case is based on a 43-year-old male who had multiple episodes of seizure for which he was brought to a tertiary care hospital, where he was declared brought dead. This case highlights the significance of conducting a comprehensive medicolegal death investigation, which involves interviews with relatives and a complete forensic autopsy to establish the cause and manner of death.

KEYWORDS

• Sudden death • Neurocysticercosis • Taenia solium • Brain stem dysfunction • Cerebral edema

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INTRODUCTION

Death is said to be sudden or unexpected when a person not known to have been suffering from any dangerous disease, injury or poisoning is found dead or dies within 24 hours after the onset of terminal illness[1]. Sudden death is a major medico-legal and public health issue, as it often occurs without warning and requires post-mortem examination to determine the cause. In developing countries, infectious and parasitic diseases such as neurocysticercosis can contribute to sudden death, especially in young individuals. The World Health Organization classifies neurocysticercosis as a neglected zoonotic disease that poses a significant global health challenge. It is the most common parasitic infection affecting the human central nervous system and a leading cause of adult-onset seizures worldwide. The prevalence of neurocysticercosis as a cause of active epilepsy in India is estimated at about 1 per 1000 population, with 1.2 million people suffering from epilepsy due to neurocysticercosis**.** It accounts for 26–53% of acquired epileptic seizures in India, making it the single most common infectious cause[2]. The high burden of the cause is due to poor hygiene, scavenging pigs, and unregulated slaughtering practices. For forensic pathologists, correctly identifying neurocysticercosis (NCC) during autopsy is crucial because missing it can lead to difficulty in arriving to cause of death.

CASE PRESENTATION

A 43-year-old male had multiple episodes of seizure at his residence, for which he was taken to the nearby hospital for initial treatment. They were then referred to a tertiary care hospital for further treatment. On examination, He was unresponsive, Pulse absent, Blood pressure not recordable, Pupils dilated and ECG showed asystole, then he was declared brought dead. A forensic pathologist performed a medicolegal autopsy. On external examination, a well-built, well-nourished body of a 5ft 5 inches, 43-year-old male subject; Rigor Mortis present all over the body; Postmortem hypostasis present and fixed over dependent parts of the body; no external injury could be detected. On Internal examination, all visceral organs were congested. The brain dissection starts with a bi-mastoid coronal scalp incision, and the scalp is reflected. A circular craniotomy is performed using a saw to remove the skull cap, and the dura mater is opened in a cruciate fashion. The frontal lobes are gently elevated, and the cranial vessels are cut. Finally, the cerebellum is lifted, and the lower cranial nerves are severed. After meticulous coronal dissection. The brain was edematous with multiple small cystic lesions present over the bilateral cerebral hemispheres. On cut section, multiple small fluid-filled cystic lesions are present over the basal cistern (Figure 1), and a small cerebral cyst was retrieved and sent for histopathological examination (Figure 2)

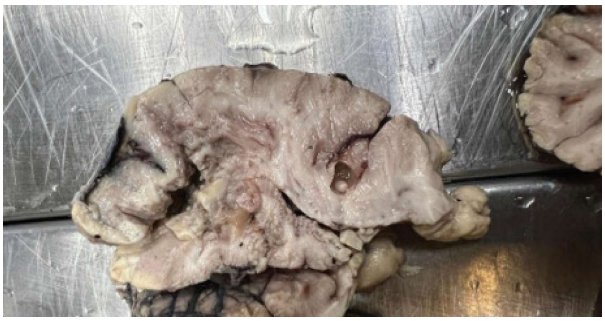

Figure 1: Cyst present over the intraventricular space

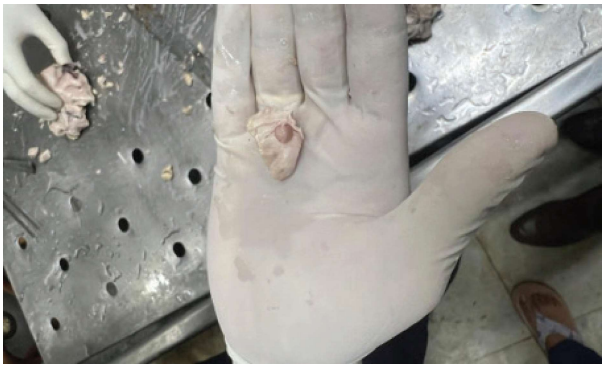

Figure 2: Sample of cerebral cyst sent for histopathological examination

DISCUSSION

Neurocysticercosis is a parasitic infection of the central nervous system caused by the larval stage of the pork tapeworm Taenia solium. Humans acquire the disease by ingesting tapeworm eggs, which subsequently develop into cysts within the brain parenchyma, ventricles, subarachnoid space, or spinal cord. The life cycle of Taenia solium involves humans as the definitive host, harboring the adult worm in the small intestine, and pigs as the intermediate host, containing larval cysticerci in muscle tissue. Eggs released in human feces are ingested by pigs, where oncospheres penetrate the intestinal wall and develop into cysticerci in tissues. Humans acquire intestinal infection by consuming undercooked pork containing cysticerci, which mature into adult

worms. When humans ingest eggs through fecal-oral contamination or autoinfection, larvae disseminate to tissues, including the brain, causing Neurocysticercosis[3] (Figure 3).

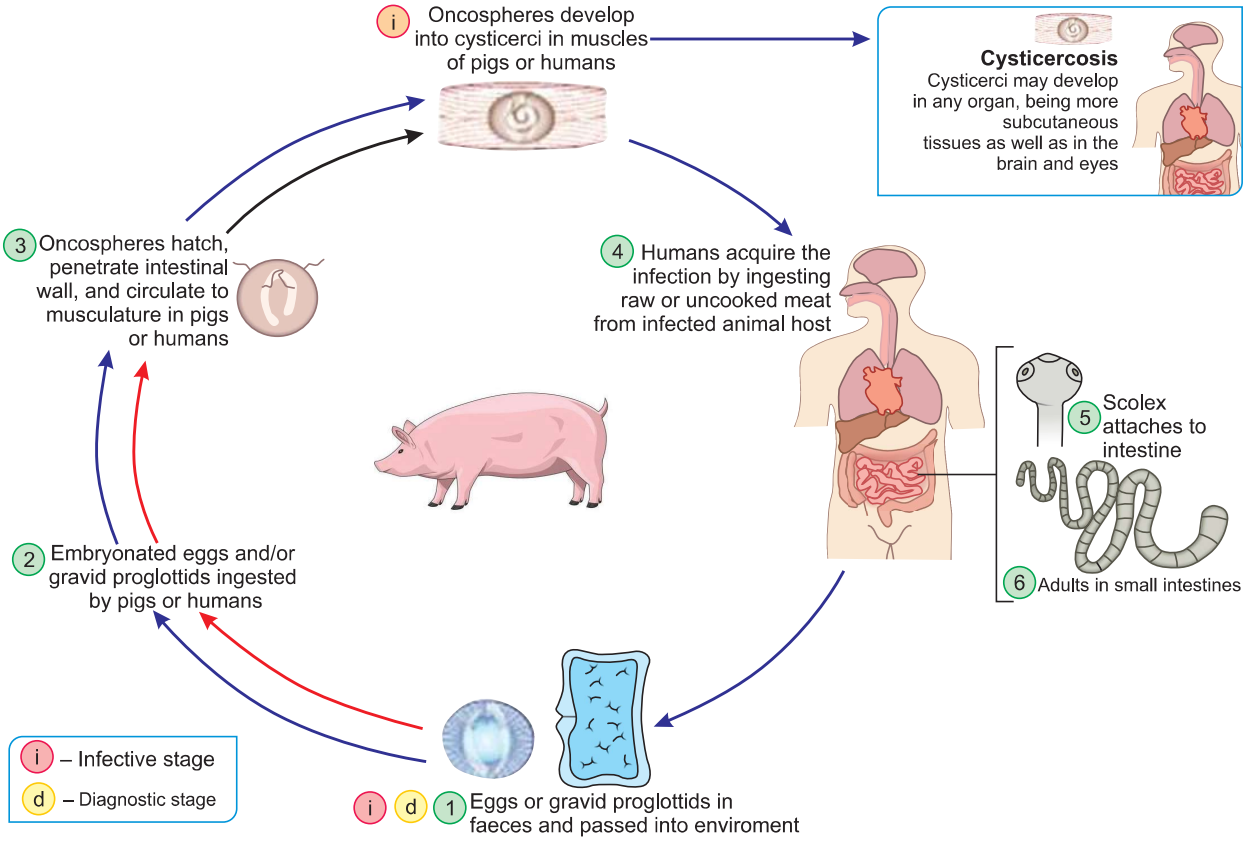

Figure 3: Life cycle of Taenia solium

When Taenia solium larvae localize in the central nervous system, they form cysticerci that progress through vesicular, colloidal, granular, and calcified stages, producing stage-specific effects in the brain, collectively termed Neurocysticercosis. During the viable vesicular stage, cysts may cause minimal symptoms, but as they degenerate, leakage of parasitic antigens triggers an intense host inflammatory response, leading to perilesional edema and seizures, the most frequent manifestation. Parenchymal lesions commonly result in focal epilepsy, while ventricular and subarachnoid cysts obstruct cerebrospinal fluid pathways, producing hydrocephalus, raised intracranial pressure, and risk of sudden deterioration. Chronic calcified granulomas may persist as epileptogenic foci, and subarachnoid disease can cause vasculitis and ischemic stroke. The neurological morbidity largely results from an inflammatory reaction during cyst degeneration rather than the presence of the viable parasite alone[4]. In the early stage, MRI typically demonstrates a thin-walled cyst with cerebrospinal fluid–like signal intensity and an eccentric mural nodule representing the scolex, producing the characteristic "hole-with-dot" sign associated with Taenia solium infection (Figure 4). As cyst degeneration begins, MRI reveals ring enhancement and perilesional edema, allowing disease staging and early therapeutic intervention.[5]

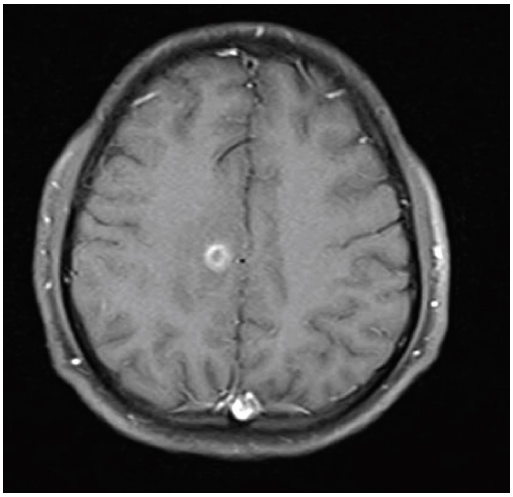

Figure 4: A well-defined thick-walled ring enhancing lesions in MRI

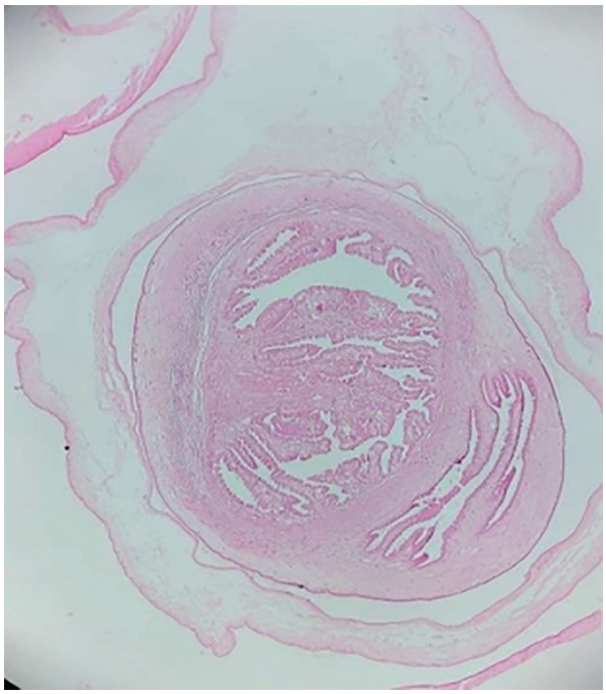

Figure 5: Cysticercus larva with invaginated scolex surrounded by inflammatory gliotic brain tissue

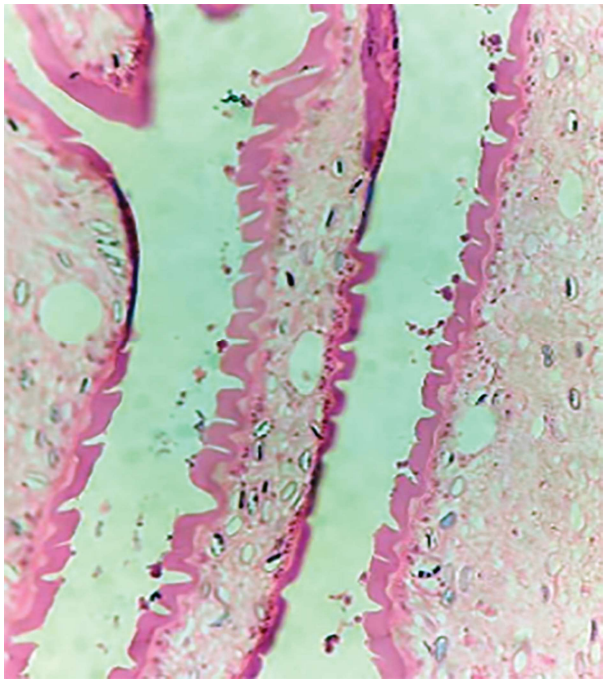

Figure 6: Cysticercus wall with microvilli on the tegument surface

Histopathological findings (Figures 5 and 6) reveal neural parenchyma containing a cyst with a larva. Cross-section of the larva shows hooklets and a scolex resembling neurocysticercosis, with surrounding areas showing reactive gliosis and inflammation[6]. Neurocysticercosis can be treated with Albendazole and corticosteroids to reduce inflammation, plus antiepileptic drugs for seizure control.[7] In neurocysticercosis, degeneration of cysticerci provokes an intense inflammatory response that disrupts the blood–brain barrier, producing cerebral oedema and sometimes hydrocephalus from CSF obstruction; the resulting raised intracranial pressure leads to brain herniation and compression of vital brainstem centres controlling respiration and cardiac activity, while recurrent seizures and autonomic instability may precipitate fatal arrhythmias or respiratory arrest, making brainstem failure due to cerebral oedema the major cause of sudden death in such cases.[8]

CONCLUSION

This case involves a 43-year-old male who experienced a sudden, unexpected collapse with no significant external injuries, and autopsy revealed multiple cystic lesions within the brain consistent with neurocysticercosis. Neuropathological examination demonstrated degenerating cysticerci with surrounding inflammatory reaction, diffuse cerebral edema, and features of raised intracranial pressure, including brain swelling and early herniation. These findings indicate that inflammatory degeneration of the parasite led to breakdown of the blood–brain barrier, resulting in marked cerebral edema and secondary brainstem compression. The absence of trauma, toxicological abnormalities, or other natural disease processes supports a neurological cause of death. Additionally, the possibility of seizure- related autonomic dysfunction contributing to terminal events cannot be excluded. Correlation of scene findings, clinical history where available, and histopathology confirms that the individual died due to brainstem failure following raised intracranial pressure caused by neurocysticercosis, establishing it as the underlying cause of sudden natural death.

REFERENCES

1. The Essentials Of Forensic Medicine And Toxicology by K S Narayan Reddy 36/e 2026 page no:137
2. Devi, K R., Debasish Borbora, Narayan Upadhyay, Dibyajyoti Goswami, S K. Rajguru, and Kanwar Narain. "High prevalence of neurocysticercosis among patients with

epilepsy in a tertiary care hospital of Assam, India." Tropical Parasitology 12, no. 1 (2022): 15-20.

3. Greenfield's neuropathology 9th edition Pg no: 1269
4. "Neurocysticercosis: The Hidden Brain Parasite Crossing Global Borders In 2025." GlobalRPH. 2025.
5. Coyle, Christina M.. "New Insights Into Calcified Neurocysticercosis: Closing the Knowledge Gap." Clinical Infectious Diseases 73, no. 9 (2021).
6. Greenfield's neuropathology 9th edition Pg no: 1271
7. (IDSA), Infectious D., and American S. (ASTMH). "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)." Clinical Infectious Diseases 66, no. 8 (2017).
8. Perez, Alejandro, Gaurav Syngal, Samreen Fathima, Sam Laali, and Sadat Shamim. "Intraventricular neurocysticercosis causing obstructing hydrocephalus." Proc (Bayl Univ Med Cent). 2022; 35(5): 722-724. (2022).