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Case Report

Hydrocephalus Due to Neurocysticercosis in the Cerebral Aqueduct: Case Report

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Abstract: Neurocysticercosis (NCC) is the leading cause of parasitic infection of the central nervous system, resulting from infestation by the larval stage of *Taenia solium*. Intraventricular forms of the disease, although less frequent, are associated with high morbidity and mortality, mainly due to obstruction of cerebrospinal fluid flow and consequent hydrocephalus. We report the case of a patient previously diagnosed with NCC who presented with acute clinical deterioration secondary to obstruction of the cerebral aqueduct by a cyst compatible with a cysticercus, a location rarely described in the literature. The patient underwent emergency neurosurgical intervention with good clinical outcome. Antiparasitic therapy was not initiated, considering the high number of viable lesions and the risk of an exacerbated inflammatory response. This case report reinforces the importance of early diagnosis of complications associated with NCC, particularly ventricular forms, and expands knowledge of atypical topographic presentations, contributing to improved clinical recognition and appropriate management of the disease.

Keywords: Neurocysticercosis; Hydrocephalus; Mesencephalic Aqueduct.

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1. Introduction

Neurocysticercosis (NCC) is an infection of the central nervous system (CNS) caused by the larval form (cysticercus) of the parasite *Taenia solium*. The cysticercus becomes established in the CNS after ingestion of parasite eggs, usually through direct fecal–oral contamination or consumption of contaminated food and water [1]. This parasite is endemic in most developing countries and remains one of the leading preventable causes of epilepsy and neurological morbidity [2,3]. The World Health Organization (WHO) recognizes NCC as a neglected tropical disease, particularly in endemic areas of Latin America, Sub-Saharan Africa, and Southeast Asia, where sanitation conditions are inadequate [4,5]. It is estimated that more than 50 million people are infected worldwide, with NCC accounting for approximately 30% of cases of late-onset epilepsy in endemic regions [6,7].

From a clinical perspective, NCC is characterized by a wide heterogeneity of manifestations, resulting from the interaction between the location, number, and evolutionary stage of the cysticerci, as well as the host's inflammatory response over time [8,9]. NCC can be classified into parenchymal and extraparenchymal forms. Although less prevalent, the extraparenchymal form is associated with greater morbidity and therapeutic complexity, especially when there is intraventricular involvement [9–12].

Intraventricular forms usually progress with obstructive hydrocephalus resulting from blockage of cerebrospinal fluid flow. Involvement of the mesencephalic aqueduct,

also known as the cerebral aqueduct or aqueduct of Sylvius, represents one of the rarest locations [10–12]. This specific topography has clinical relevance disproportionate to its frequency, since small lesions may result in acute neurological deterioration [3,11]. In this context, we present a case of obstructive hydrocephalus secondary to neurocysticercosis located in the cerebral aqueduct, discussing the diagnostic challenges and therapeutic dilemmas associated with this uncommon presentation, with emphasis on clinical decision-making in scenarios of multifocal and chronic disease.

2. Case Report

A 60-year-old male patient, born in the countryside of the state of Minas Gerais and residing in the metropolitan region of São Paulo, with a diagnosis of neurocysticercosis for approximately 10 years and on continuous use of phenobarbital. A family member was unable to report whether the patient had previously received antiparasitic treatment, and it was not possible to retrieve medical records clarifying this information, which represents a relevant limitation in reconstructing the therapeutic history of the disease. Approximately five months prior to hospitalization, the patient experienced a seizure, followed by progressive functional decline, becoming dependent for instrumental activities and partially dependent for basic activities of daily living. Ten days before admission, during an outpatient visit, the family member reported worsening of the general condition, reduced strength in the lower limbs, and hypoactivity with approximately one week of evolution.

A brain MRI report performed seven months earlier was available, showing multiple hypointense foci on T2* sequences compatible with residual calcifications, as well as multiple nodular lesions with peripheral enhancement in the cerebral parenchyma, associated with adjacent vasogenic edema. The patient was referred to the emergency department due to suspected reactivation of NCC. During the initial hospitalization, a cranial CT scan revealed multiple calcified nodules and dilation of the supratentorial ventricular system. Lumbar puncture revealed clear and colorless cerebrospinal fluid, normocellularity (1 leukocyte), glucose level of 56 mg/dL (without a concomitant record of serum glucose in the chart; however, glycemic measurements on the same day ranged between 75 and 88 mg/dL), and elevated protein level of 82 mg/dL. The absence of pleocytosis or hypoglycorrhachia suggested, at that time, the absence of a significant inflammatory process in the cerebrospinal fluid.

The patient remained hospitalized due to decreased level of consciousness and psychomotor agitation, without initial initiation of specific treatment for NCC. Although this management reflects frequent limitations of initial care in emergency services, the delay in defining therapy may have contributed to the observed clinical progression. Given the complexity of the case, transfer to a tertiary care center was requested. After 10 days, the patient was transferred to a specialized tertiary hospital. On admission, he presented with drowsiness (GCS 13), isocoric and photoreactive pupils, and right-sided hyperreflexia. A new cranial CT scan (Figure 1) showed multiple calcified lesions of a sequelae-like appearance and supratentorial ventriculomegaly. Phenytoin and dexamethasone were initiated.

Serology for cysticercosis showed non-reactive IgM and reactive IgG. MRI performed on the third day demonstrated multiple residual calcifications and mild ventriculomegaly. The patient subsequently developed a new seizure, decreased level of consciousness (GCS 9), and left-sided mydriasis, and underwent emergency external ventricular drainage, followed later by definitive ventriculoperitoneal shunt placement. On the 19th day of hospitalization, brain MRI with FIESTA sequence (Figure 2) demonstrated multiple intraparenchymal, intraventricular, and subarachnoid lesions, some with ring enhancement, as well as a structure compatible with a lesion or membrane obstructing the inferior segment of the cerebral aqueduct, with no detectable cerebrospinal fluid flow.

Figure 1. Contrast-enhanced axial cranial computed tomography demonstrating multiple nodular lesions in the parenchyma, subarachnoid space, and lateral ventricle, as well as dilation of the supratentorial ventricular system.

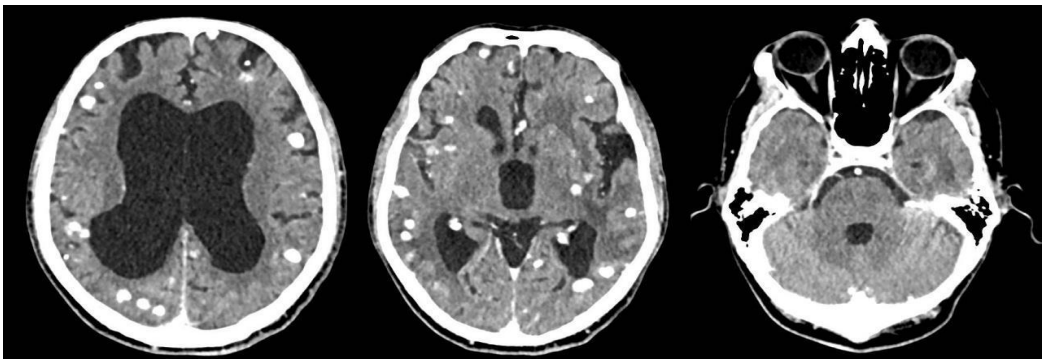
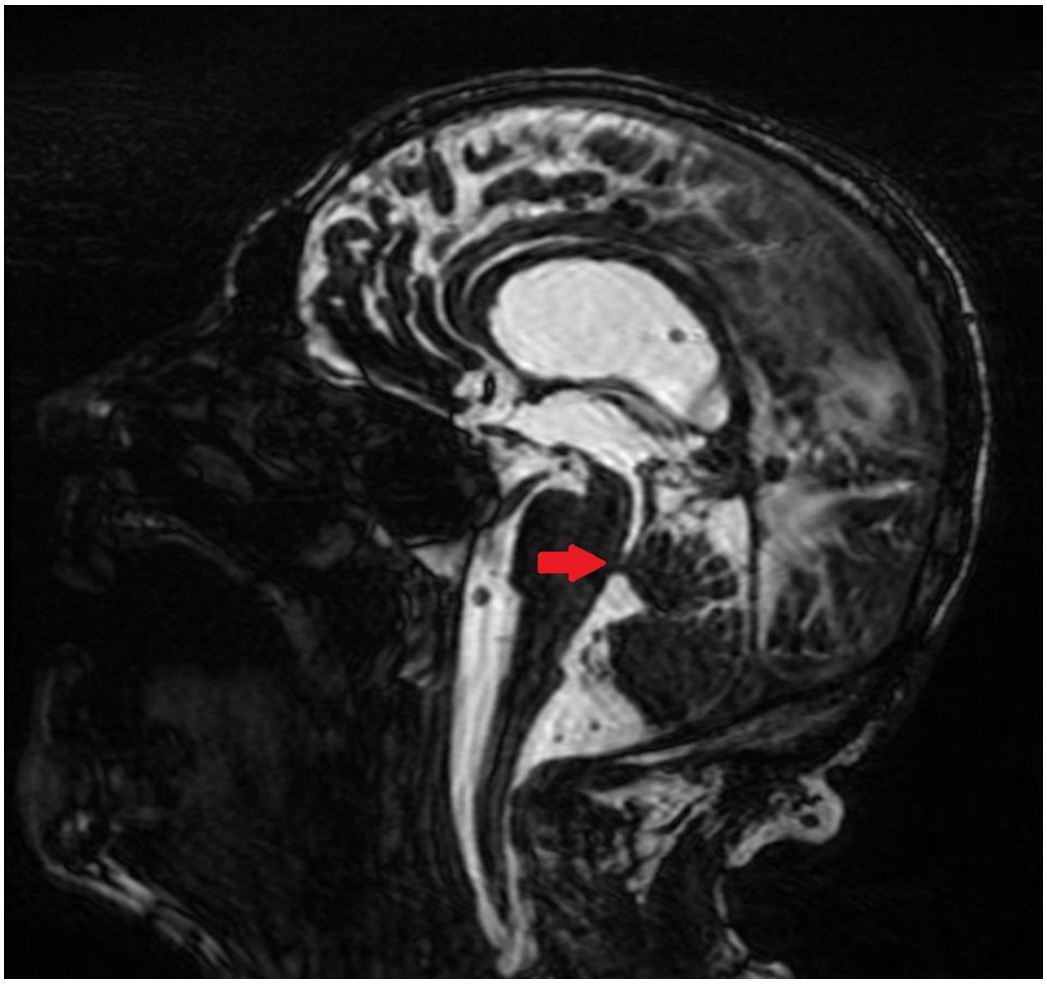


Figure 2. Sagittal brain magnetic resonance imaging in a 3D FIESTA sequence demonstrating obstruction (arrow) of cerebrospinal fluid flow in the cerebral aqueduct.

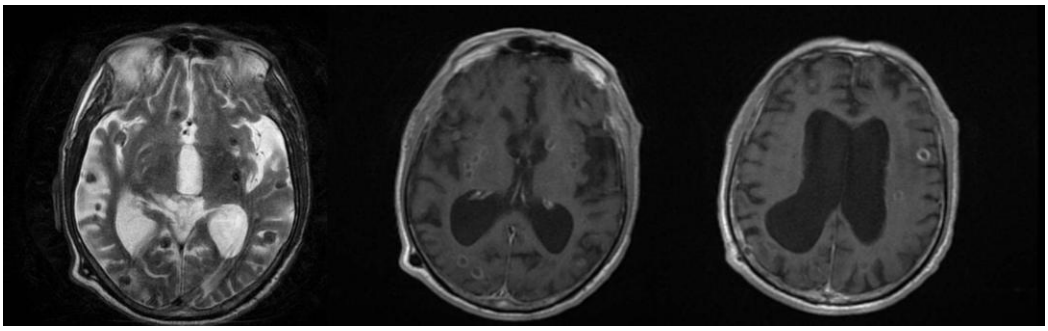


After joint evaluation by the Neurology and Neurosurgery teams, it was decided not to initiate antiparasitic therapy at that time. This decision was based on an integrated assessment that considered the coexistence of lesions at multiple evolutionary stages, the context of established chronic encephalopathy, and the patient's significant functional limitation. In this scenario, it was understood that the potential risk of inflammatory exacerbation and neurological deterioration could outweigh the immediate benefits of etiological treatment. The possibility of staged antiparasitic therapy under intensive corticosteroid

coverage after stabilization of hydrocephalus was discussed but deferred in view of the clinical profile presented at that moment.

The patient was discharged after 29 days of hospitalization. Brain MRI performed one year after discharge (Figure 3) showed persistence of multiple active inflammatory lesions. From a clinical standpoint, he remained globally neurologically stable, although with persistent mild cognitive dysfunction and sporadic seizures. The Neurology team planned to reassess antiparasitic therapy in conjunction with corticosteroid treatment; however, the patient did not attend the last scheduled follow-up visit and attempts to contact him had been unsuccessful up to the time of writing of this manuscript.

Figure 3. Axial brain magnetic resonance imaging demonstrating edema on T2-weighted sequences and ring-enhancing lesions on post-contrast T1-weighted sequences.



3. Discussion

Cysticercosis occurs when humans ingest parasite eggs, which may be transmitted via the fecal–oral route from a human carrier of taeniasis, with the possibility of autoinfection. In the host's digestive tract, the eggs release oncospheres that can migrate and form cysts in organs such as muscles, subcutaneous tissue, heart, and the CNS, the latter resulting in neurocysticercosis [2,3]. From a pathophysiological standpoint, cysticerci pass through four stages: vesicular, colloidal, granular-nodular, and calcified. Vesicular forms correspond to viable cysts and are usually associated with a lower inflammatory response, whereas colloidal and granular-nodular forms trigger a more intense inflammatory reaction, resulting in edema, mass effect, and possible disruption of the blood–brain barrier [1,3,12]. The prevalence of NCC in Brazil is still not well defined due to underdiagnosis. However, an extensive literature review including studies published between 1915 and 2002 showed a prevalence ranging from 0.12% to 9% in autopsy studies and from 0.03% to 13.4% in clinical studies, with higher frequency in the Southeast, South, and Central-West regions [13,14].

The use of imaging studies such as computed tomography and magnetic resonance imaging is essential for the diagnosis of NCC, as they allow visualization of cyst morphology and location, infection severity, evolutionary stages of the cysts, and the presence of adjacent inflammation [3]. Computed tomography (CT) is particularly useful for detecting parenchymal calcifications, which indicate old infection or partial resolution of the disease. Magnetic resonance imaging (MRI), in turn, is considered the method of choice for detailed evaluation of active lesions, localization of cysts in difficult-to-access regions such as the ventricular system and subarachnoid spaces, and for therapeutic planning [1,3,12]. Specific MRI sequences such as FIESTA or CISS (constructive interference in steady state), combined with cerebrospinal fluid flow studies, allow precise detection of intraventricular cysticerci and assessment of ventricular system obstruction, as demonstrated in the present case. In addition, serological tests, such as immunofluorescence assays, may aid in diagnostic confirmation, although they have limitations, especially in extraparenchymal forms or in patients with calcified lesions [1,3,12].

Among the clinical forms of NCC, the parenchymal form is the most prevalent, and among extraparenchymal forms, the intraventricular form is the most common [11,12].

Involvement of the cerebral aqueduct, as in the case described results in obstruction of communication between the third and fourth ventricles, leading to supratentorial ventricular dilation and accumulation of cerebrospinal fluid, a condition that can rapidly progress to neurological deterioration if untreated [3,11]. This presentation is particularly challenging due to the narrow anatomy of the aqueduct and diagnostic difficulty, which explains the severity of the clinical presentation in the patient reported here. Although intraventricular neurocysticercosis represents a relevant proportion of extraparenchymal forms, specific involvement of the cerebral aqueduct is consistently described as rare. Surgical series and MRI-based studies show that among intraventricular cases, most cysts are located in the fourth ventricle, followed by the third ventricle and lateral ventricles, whereas aqueductal localization corresponds to a small fraction of cases, ranging from approximately 3% to 9% when reported. In some Brazilian cohorts, no cases of aqueduct involvement were identified, reinforcing the exceptional nature of this topography and its clinical relevance disproportionate to its frequency [10–12,15,16].

In the present report, the patient showed involvement of both parenchymal and extraparenchymal forms, including intraventricular and subarachnoid lesions. Among the intraventricular lesions, there was involvement of the lateral ventricles and the cerebral aqueduct. Although ventricular involvement is present in a relevant proportion of extraparenchymal cases, specific localization in the cerebral aqueduct is considered one of the most uncommon presentations [10–12,16]. Management of neurocysticercosis should be individualized, considering factors such as lesion location, evolutionary stage of the cysticerci, presence of signs of intracranial hypertension, intensity of the inflammatory response, and the patient's clinical status. In general, treatment may involve three main pillars: antiparasitic therapy, control of the inflammatory response, and neurosurgical interventions when indicated [3,17].

Unlike parenchymal forms, the management of intraventricular NCC remains a matter of debate. Although antiparasitic treatment is widely recommended for viable parenchymal lesions, its use in intraventricular forms may be associated with inflammatory exacerbation, ependymitis, and cerebrospinal fluid shunt dysfunction [3,11,16,17]. In the present case, the decision for a conservative approach after ventricular shunting was not based exclusively on the intraventricular location, but on an integrated clinical assessment. It is recognized, however, that the absence of antiparasitic therapy carries relevant risks, such as persistence of disease activity, lesion progression, shunt dysfunction, and chronic CNS inflammation. Thus, this approach should not be interpreted as curative, but rather as a containment strategy in a specific clinical scenario.

Although staged therapeutic strategies, with delayed introduction of antiparasitic agents under intensive corticosteroid coverage, have been described, there is no consensus regarding their safety in patients with recent hydrocephalus, high parasitic burden, and chronic encephalopathy [3,17]. In this context, the management adopted sought to balance risk and benefit, explicitly acknowledging the limitations of each strategy.

4. Conclusion

Intraventricular neurocysticercosis with involvement of the cerebral aqueduct constitutes a rare and potentially severe presentation, associated with obstructive hydrocephalus and risk of acute neurological deterioration. This case report highlights the importance of advanced neuroimaging for diagnosis and emphasizes that the management of these extraparenchymal forms must be individualized. In similar scenarios, stabilization of hydrocephalus is mandatory, while the decision regarding antiparasitic therapy should be dynamically reassessed, considering parasitic burden, clinical response, inflammatory risk, and realistic therapeutic goals for each patient. Rather than proposing a single management strategy, this case illustrates the complexity of decision-making in aqueductal NCC and reinforces the need for a multidisciplinary, critical, and patient-centered approach.

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Conflicts of Interest: All other authors declare no conflicts of interest.

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