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Dual Burden: Radiologically Confirmed Neurocysticercosis Complicated by Infective Endocarditis in a Young Patient: Case Report

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ABSTRACT

We report the case of a 17-year-old male who presented with progressive headache, fever, vomiting, and neck stiffness. On admission, he was febrile, tachycardic, tachypneic, hypoxic, and severely anemic, with conjunctival hemorrhages and new cardiac murmurs. Neurologically, he had reduced consciousness, left-sided weakness, hypertonia, and meningeal signs. Echocardiography revealed large vegetations on the aortic and mitral valves with moderate regurgitations and pulmonary hypertension, confirming native-valve infective endocarditis. Neuroimaging demonstrated a left parietal mass extending from the choroid plexus with multiple subarachnoid and scalp cystic lesions, consistent with racemose neurocysticercosis, complicated by severe vasogenic edema, obstructive hydrocephalus, subfalcine herniation, and intraparenchymal hemorrhages. The patient underwent burr-hole decompression (yielding mainly blood) and was treated with albendazole, praziquantel, corticosteroids, and antiepileptics for neurocysticercosis, alongside intravenous vancomycin and ceftriaxone for six weeks for infective endocarditis. This case highlights the rare coexistence of infective endocarditis and racemose neurocysticercosis with intracerebral hemorrhage, underscoring the importance of multidisciplinary care in managing overlapping infectious and neurological pathologies.

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Introduction

Neurocysticercosis (NCC) is the most common parasitic infection of the Central Nervous System (CNS) worldwide and a leading cause of acquired epilepsy, particularly in developing regions where Taenia solium remains endemic due to close contact between humans and pigs [1,2]. NCC manifests when the larval form of the tapeworm invades the CNS after ingestion of T. solium eggs. Its clinical presentation varies widely depending on the number, location, and viability of cysts, ranging from seizures and headaches to hydrocephalus, intracranial hypertension, and neuropsychiatric disturbances [3,4]. The diagnosis of NCC is based on epidemiological, clinical, radiological, and serological criteria, with neuroimaging (computed tomography or magnetic resonance imaging) being crucial for the diagnosis [5,6].

While parenchymal NCC is more commonly diagnosed and often manageable with anthelmintic agents, extra parenchymal forms, including intraventricular, subarachnoid (racemose), and spinal involvement, are associated with higher morbidity and mortality [7,8]. Racemose NCC, a less frequent but more aggressive form, involves multilobulated cysts in the basal cisterns or subarachnoid space and lacks a scolex, often mimicking neoplastic lesions or chronic arachnoiditis [9,10]. It is frequently complicated by obstructive hydrocephalus, cerebral edema, herniation, and vasculitis, which may lead to infarcts or hemorrhage [11]. Vascular neurocysticercosis, though rare and underrecognized, may present with infarcts or hemorrhages due to inflammatory occlusion, aneurysmal dilation, or rupture of cerebral vessels, as a result of chronic vasculitis changes [5]. This adds a layer of complexity to both diagnosis and management, particularly in young patients.

On the other hand, Infective Endocarditis (IE), characterized by microbial infection of the endocardial surface of the heart, is uncommon in adolescents but has been increasingly reported

in association with underlying congenital heart disease, dental procedures, or systemic infections [12]. When IE occurs in younger, otherwise healthy individuals, it often signifies systemic vulnerability or undetected routes of microbial dissemination. The coincidental presentation of NCC and IE is exceedingly rare and poses a significant diagnostic dilemma, particularly when systemic signs such as fever, altered mental status, and embolic phenomena may overlap.

To our knowledge, very few cases have reported the coexistence of neurocysticercosis and infective endocarditis, and even fewer have documented this in adolescent patients. Whether this association reflects a causal link, an immunological predisposition, or a coincidental dual infection remains unclear. However, the diagnostic and therapeutic challenges are substantial, particularly in low-resource settings where delayed presentation, limited imaging modalities, and overlapping neurological symptoms may complicate care.

Case

We report a case of a 17-year-old male with radiologically confirmed racemose and vascular neurocysticercosis, complicated by intraparenchymal hemorrhages, hydrocephalus, and subfalcine herniation, alongside echocardiographically confirmed infective endocarditis, highlighting the diagnostic complexity and the need for a multidisciplinary approach. Patient presented with new-onset generalized seizures, fever, and headache He was started on albendazole, praziquantel, dexamethasone, and antiepileptics. Blood cultures done did not yield any growth but echocardiography confirmed mitral valve and aortic valve vegetations, diagnosing native-valve infective endocarditis. A multidisciplinary team initiated intravenous ceftriaxone and vancomycin with good response. Patient underwent surgery for excision of the racemose lesion and also completed antiparasitic and antibiotic treatment without complications and was discharged from the ward, however patient demised one week post discharge.

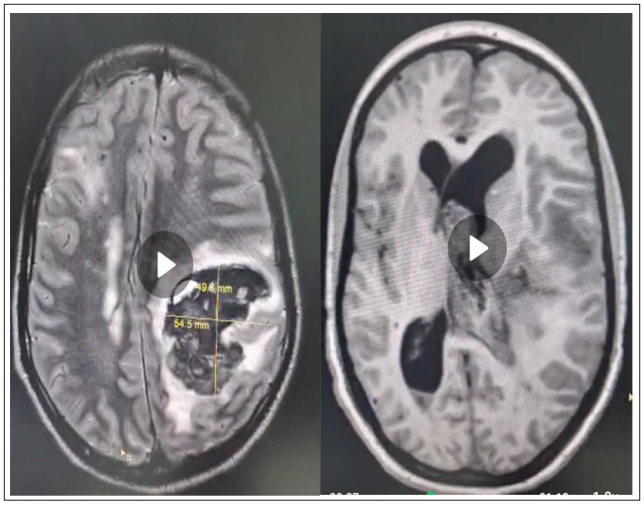

Figure 1: MRI Axial Images of the Brain

Surgical Procedure

Patient was placed in supine position with the head turned to the right, resting on a doughnut headrest. Left temporal region is exposed, prepped, and draped under sterile conditions. A curvilinear skin incision was made, 3–4 cm, approximately 2–3 cm above the external auditory meatus and 2–3 cm posterior to the lateral orbital rim. A temporal burr hole was placed just above the zygomatic arch and anterior to the ear. Dura was identified and a cruciate durotomy incision made, old hemorrhagic blood and debris are evacuated, approximately 60ml.

Progress

Patient stayed in Intensive Care Unit (ICU) for 1 week and High Dependency Unit (HDU) for another week, for extended neuro-resuscitation and did well and was discharged to the ward without incident. He was discharged home six weeks later; however, patient passed away one week post discharge from the hospital

Discussion

This case presents a unique and clinically challenging scenario of concurrent Neurocysticercosis (NCC) and Infective Endocarditis (IE) in a 17-year-old male with altered mental status, systemic inflammatory response, and neuroimaging-confirmed intracranial pathology. While both diseases are independently significant public health concerns in endemic regions, their coexistence is rare and presents diagnostic and therapeutic complexities.

Neurocysticercosis, a parasitic infection of the central nervous system caused by the larval form of Taenia solium, is the leading cause of acquired epilepsy in endemic regions [1,4]. It typically affects impoverished communities where poor sanitation facilitates the fecal-oral transmission cycle. This patient's clinical profile, which includes fever and decreased consciousness (GCS 11/15), correlates with parenchymal NCC complicated by cyst degeneration or increased Intracranial Pressure (ICP) [6,11].

Neuroimaging plays a crucial role in staging the disease and guiding management. There was need for surgical decompression via burr hole evacuation in this case as there was extra parenchymal and ventricular involvement with mass effect and obstructive hydrocephalus, likely secondary to inflammation or hemorrhagic cyst degeneration, both recognized complications in NCC [5].

The diagnosis of infective endocarditis was supported by echocardiographic evidence of large mitral valve vegetations (1.5 x 1.8 cm), moderate aortic regurgitation, and dilated right ventricle with mild pericardial effusion. The white cell counts of 47 x10^9/L, anemia (Hb 4 g/dL), and thrombocytopenia (platelets 144 x10^9/L) reflect a profound systemic inflammatory response, with potential marrow suppression or consumptive coagulopathy. Large vegetations are concerning for septic embolism, which could plausibly explain the intracranial hemorrhagic component observed during surgery. Embolic strokes or hemorrhagic infarcts from septic emboli are well-recognized neurological complications of left-sided IE, particularly involving the mitral valve [12]. The echocardiographic findings in this patient, along with neurological deterioration, support a high embolic risk profile [13].

Furthermore, the moderate aortic regurgitation and right ventricular dilatation raise concern for early biventricular volume overload or pulmonary hypertension, which may have contributed to reduced cerebral perfusion and worsened neurological status [8,13]. Despite these abnormalities, the preserved ejection fraction (EF 58%) indicates maintained left ventricular systolic function at the time of assessment.

The concurrent presentation of NCC and IE is unusual but not implausible in immunocompromised or malnourished individuals, or in the context of systemic inflammation [13]. There is some evidence that NCC-related neuroinflammation can compromise the blood-brain barrier and enhance susceptibility to secondary infections or complications from embolic phenomena [14,15]. Additionally, prolonged bacteremia in IE can seed CNS structures, especially in the setting of preexisting cystic or necrotic brain

lesions [14]. It remains unclear whether the hemorrhagic cyst observed was purely due to NCC progression or exacerbated by embolic ischemia and reperfusion injury from IE. Nevertheless, the presence of old hemorrhagic blood during surgery suggests a sub-acute process, possibly from a previous embolic infarct complicated by secondary infection or cyst rupture [16].

The multidisciplinary approach in this patient, combining cardiac monitoring, antimicrobial therapy, surgical decompression for NCC-related mass effect, and supportive critical care, reflects current best practice in managing complex co-infections with neurological involvement. Surgical evacuation in NCC is generally reserved for cases with raised ICP, ventricular entrapment, or cysts causing mass effect [5]. Infective endocarditis with large vegetations often requires early surgical consultation, especially when complicated by embolic events, heart failure, or uncontrolled infection [12]. The risk of further embolization, especially to the brain, remains significant until the vegetations are removed or stabilized [13].

The patient's postoperative recovery would hinge on adequate control of infection, stabilization of cardiac function, and prevention of recurrent intracranial complications. Long-term neurological and cardiac follow-up is essential.

Conclusion

The coexistence of neurocysticercosis and infective endocarditis is exceptionally uncommon. This case emphasizes the need for vigilance for secondary systemic infections in patients with parasitic CNS disease, particularly when fever and systemic signs persist beyond initial treatment. Early diagnosis and multidisciplinary intervention are essential for optimal outcomes.

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