Case Report

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Radiology Section

Behavioural and Executive Dysfunction as the Initial Manifestation of Disseminated Neurocysticercosis: A Case Report

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ABSTRACT

Neurocysticercosis (NCC) is the most prevalent parasitic infection of the central nervous system globally and the most common cause of acquired epilepsy in endemic areas. Neuropsychiatric manifestations, including cognitive impairment, depression, and behavioural disturbances, are also becoming increasingly common. Multisystem cysticercosis, which involves the central nervous system and extracranial sites such as skeletal muscles, subcutaneous tissues, and visceral organs, is uncommon. This is a case report of a 22-year-old male with progressive behaviour change, which included apathy, social withdrawal, and executive dysfunction. He was initially diagnosed with major depressive disorder with minimal response to antidepressant treatment. Assessment of an asymptomatic forearm swelling showed intramuscular cystic lesions with an eccentric echogenic focus on ultrasonography, suggestive of cysticercosis. Non-Contrast Computed Tomography (NCCT) of the brain revealed numerous calcified nodular lesions in both cerebral hemispheres and deep gray nuclei, without associated edema or mass effect. The cysticercus antibody serological test was positive. The general clinic-radiological picture favoured dissemination of cysticercosis with mixed stages of evolution. After a course of albendazole with corticosteroid, the patient showed improvement in executive functioning and behavioural symptoms. This case underscores how NCC should be considered among the differential diagnoses of treatment-resistant psychiatric manifestations in endemic areas.

Keywords: Albendazole, Cognitive impairment, Cysticercus antibody, Depressive disorder, Myocysticercosis

CASE REPORT

A 22-year-old male from Kuthambakkam, Chennai, presented with a history of progressive behavioural change over a six-month period. His family reported that he slowly became socially withdrawn, emotionally blunted, and disinterested in everyday activities like bathing, eating, and watching television. They also reported decreased motivation, poor planning capacity, and an inability to complete tasks requiring organisation and attention, such as cooking and arranging books.

There was no past history of psychiatric illness/medications. There was no history of seizures, headache, vomiting, focal neurological deficits, fever, weight loss, substance use, or head trauma. There was no significant past medical/surgical, family, drug, or occupational history. The patient had a history of pork consumption once a month for the past 5 years.

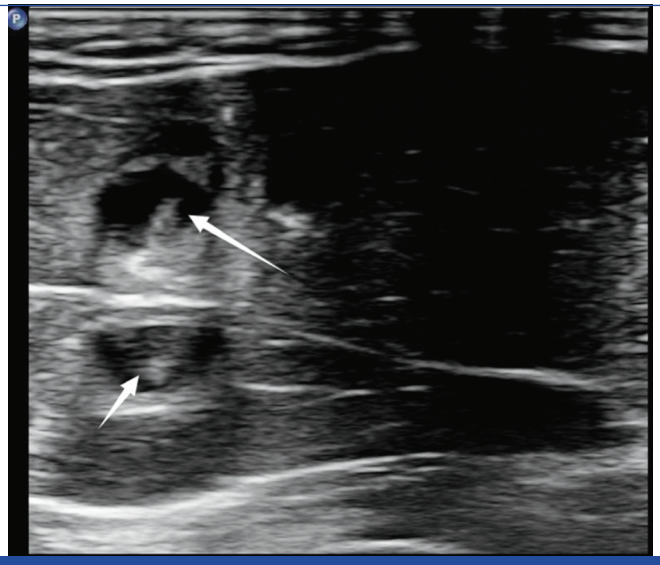
He was also assessed by an in-house psychiatrist and was diagnosed with major depressive disorder. His Hamilton Depression Rating Scale (HAM-D-17) was 20 (normal range 0 to 7) [1], which implied major depression. Three months of antidepressant therapy with oral Escitalopram 10 mg per day showed little improvement, and the repeat HAM-D-17 score was high at 17. Due to the poor response to treatment, a neurological assessment was ordered.

Upon evaluation, severe cognitive deficiency with a Frontal Assessment Battery of 6/18 (normal range 16-18) [2], reflecting severe executive dysfunction (defined by performance ≥2 standard deviations below normative values on at least two executive function tests, accompanied by significant impairment in daily functioning and characteristic deficits in planning, cognitive flexibility, and inhibitory control) was detected [3]. The vitals were normal. Physical examination revealed painless nodular swelling over the left forearm of about 2 cm diameter. No significant chest or eye findings on physical examination.

Ultrasound imaging of the left forearm with a high-frequency linear transducer revealed several discrete intramuscular cystic lesions with an eccentric echogenic focus, representing the scolex, as demonstrated in [Table/Fig-1].

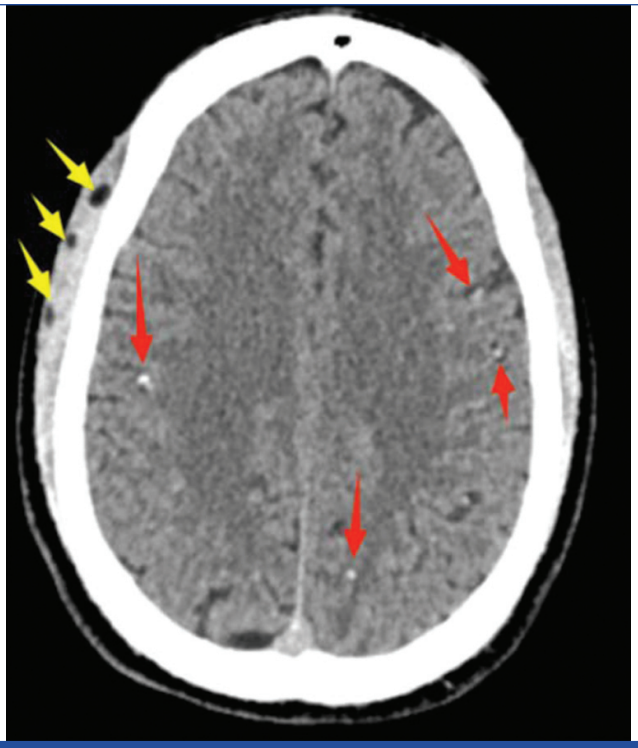
NCCT of the brain showed several calcified nodules scattered throughout the bilateral frontal, parietal, occipital and temporal lobes with preponderance at the grey-white matter junction as shown in [Table/Fig-2].

Similar calcified lesions were observed in deep gray nuclei such as the thalamus as shown in [Table/Fig-3].

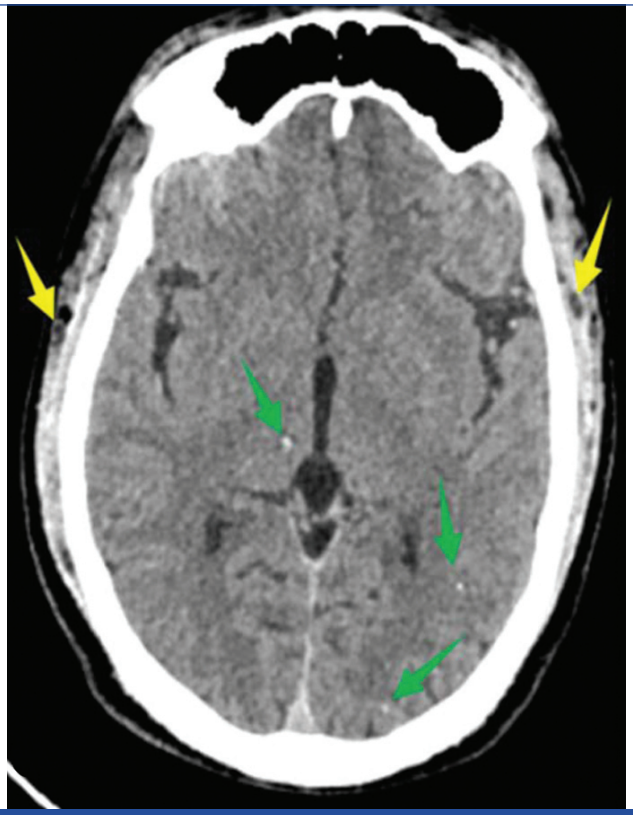


[Table/Fig-1]: Ultrasound forearm - high-frequency ultrasonography of the forearm demonstrates a well-defined intramuscular cystic lesion containing an eccentric echogenic focus representing the scolex (white arrows).

Brain Magnetic Resonance Imaging (MRI) revealed that all lesions were hypointense on T2-weighted images, with blooming on



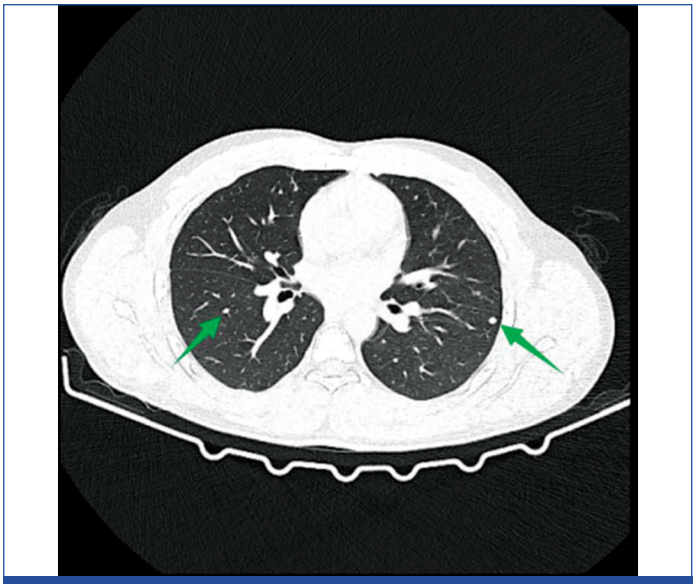
[Table/Fig-2]: Axial Non-Contrast Computed Tomography (NCCT) of the brain at the level of the centrum semiovale demonstrates multiple punctate hyperdense nodular calcifications (red arrows) distributed within the bilateral frontal and parietal subcortical regions. Incidental hypodense lesions also noted in right superficial temporalis muscle (yellow arrows).



[Table/Fig-3]: Axial non-contrast computed tomography of the brain at the level of the thalamus demonstrates multiple punctate hyperdense nodular calcifications distributed in the right thalamus and the left parietal and occipital lobes (green arrows). Coincident hypodense lesions were also noted in the bilateral superficial temporalis muscles (yellow arrows).

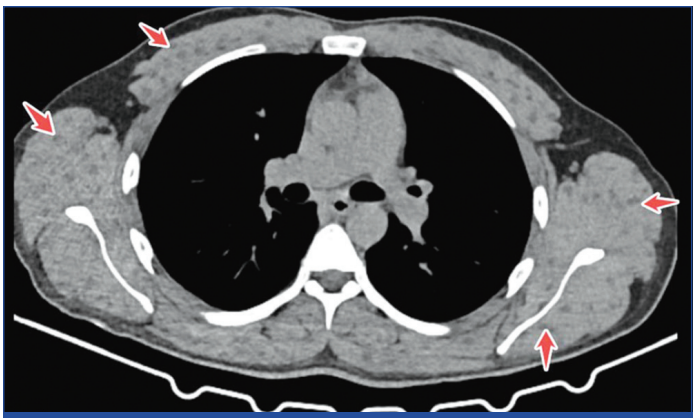
susceptibility-weighted imaging. There was no diffusion restriction, no perilesional oedema, or any contrast enhancement. These findings suggest the calcified nodular phase of NCC, which implies inactive granulomatous disease.

Chest Computer Tomography (CT) showed multiple small nodular opacities randomly scattered across bilateral lung parenchyma as seen in [Table/Fig-4].



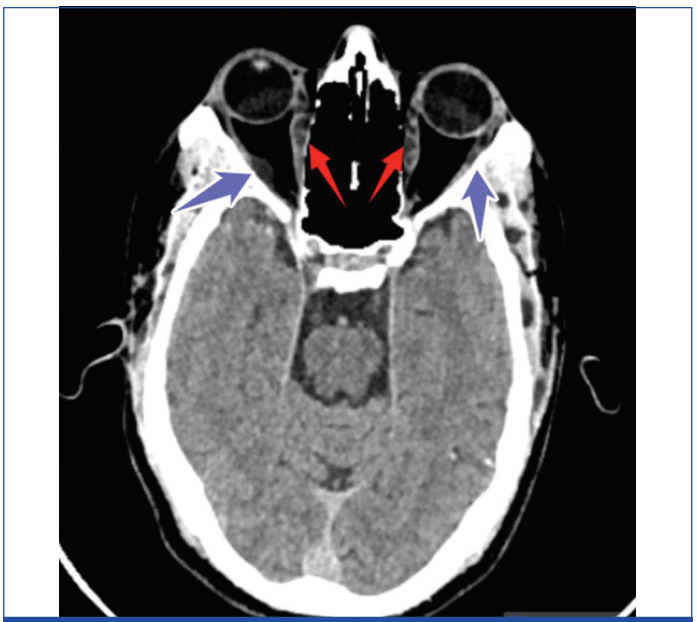
[Table/Fig-4]: Axial CT thorax Lung window images demonstrate multiple randomly distributed pulmonary nodules in both lungs (green arrows) consistent with pulmonary cysticerci.

The nodules were interpreted as pulmonary cysticerci. Moreover, several small cystic lesions were found in pectoralis major, intercostal, and paraspinal muscles as seen in [Table/Fig-5].



[Table/Fig-5]: Axial CT thorax demonstrates multiple rounded low-attenuation lesions within the pectoralis major and intercostal muscles compatible with cysticercus cysts (red arrows). No surrounding inflammatory stranding is seen.

Extraocular muscles of bilateral eye also had similar cystic lesions on CT as noted in [Table/Fig-6].



[Table/Fig-6]: Axial CT brain shows multiple rounded low-attenuation lesions within the bilateral medial rectus (red arrows) and lateral rectus (blue arrows) compatible with cysticercus cysts.

Cysticercus IgG antibody serology via enzyme-linked immunosorbent assay was also positive (2 optical density (OD) units), confirming infection with Taenia solium. Haematological studies, such as complete blood count, were in the normal range, and no peripheral eosinophilia was detected. Inflammatory markers, such as the erythrocyte sedimentation rate and C-Reactive Protein (CRP), were normal. Liver and renal function tests were normal, as shown in [Table/Fig-7].

Parameter	Patient value	Reference range
Haemoglobin (Hb)	13.8 g/dL	13.0-17.0 g/dL
Total Leukocyte Count (TLC)	7200 /µL	4000-11000 /µL
Neutrophils	58%	40-70%
Lymphocytes	34%	20-40%
Eosinophils	2%	1-6%
Platelet count	2.5 $\times 10^5$ /µL	1.5-4.0 $\times 10^5$ /µL
Erythrocyte Sedimentation Rate (ESR)	10 mm/hr	0-20 mm/hr
C-Reactive Protein (CRP)	2 mg/L	<6 mg/L
Serum Bilirubin (Total)	0.8 mg/dL	0.2-1.2 mg/dL
Aspartate Aminotransferase (AST/SGOT)	24 U/L	10-40 U/L
Alanine Aminotransferase (ALT/SGPT)	28 U/L	7-56 U/L
Serum creatinine	0.9 mg/dL	0.6-1.3 mg/dL
Blood urea	28 mg/dL	15-40 mg/dL

[Table/Fig-7]: Haematological and biochemical parameters of the patient.

A lack of systemic inflammatory response and imaging findings of calcified lesions, with no evidence of surrounding oedema, favoured the diagnosis of quiescent end-stage calcified NCC, with potentially vesicular-stage myocysticercosis and pulmonary involvement.

Albendazole was started at 15 mg/kg/day as BD for 14 days. Dexamethasone 0.1 mg/kg/day was administered BID for 10 days orally, then tapered over the next two weeks to reduce potential inflammatory effects associated with parasite degeneration, in line with guideline-based management principles [4,5]. The patient showed substantial improvement in executive functioning and behavioural symptoms at one-month follow-up (Frontal Battery Score- 15/18). HAM-D-17 score improved from 17 to 12 and family members stated that he was now more socially active and able to do routine activities better.

DISCUSSION

The NCC is the most prevalent parasitic infection of the central nervous system globally and the most common cause of acquired epilepsy in endemic areas, with a seroprevalence of 0.1%-6% in India and 0.02%-6% in China, Korea, Vietnam [6].

Radiological literature emphasises that such extracranial lesions may remain clinically silent and can be easily overlooked unless actively sought [7]. In this case, focused imaging of a clinically evident lesion provided a crucial diagnostic anchor.

The presence of both central nervous system and muscular involvement supports the mechanism of haematogenous dissemination of Taenia solium oncospheres following ingestion of infective eggs [8]. Importantly, this patient had a history of pork consumption once a month for the past five years, which represents a potential risk factor in endemic settings. However, it is also recognised that transmission can occur via faecal-oral contamination; therefore, dietary history alone cannot reliably exclude or confirm exposure [9,10].

Ganaraja HV et al., reported one of the largest contemporary Indian series of disseminated cysticercosis. Sixteen (M:F = 13:3) patients were diagnosed with Disseminated Neurocysticercosis (DNCC) with a mean age of presentation of 35.1±14.2 years. Headache was the predominant symptom, followed by seizures (93.75%), vomiting (43.75%), behavioural disturbances (31.25%), fever (12.5%), encephalopathy (12.5%), visual disturbances (6.25%), and muscle pain and limb weakness (6.25%). CT brain showed multiple active parenchymal cysts in all, and calcifications in 68.75%. Albendazole with steroids was used in 15 patients. In 93.3% patients, there was complete improvement in seizures; 12.5% subjects had persistent memory and behavioural abnormalities. One subject required a decompressive craniectomy; mortality was observed in two subjects [11].

An additional diagnostic consideration in this patient was the presence of pulmonary nodules, which can broaden the differential diagnosis to include infectious granulomas or metastatic disease. However, when correlated with characteristic findings in the brain and soft tissues, these lesions were interpreted as part of the spectrum of multisystem cysticercosis.

Similarly, Zou Y et al., reported a Chinese case series of disseminated cysticercosis involving patients ranging from children to middle-aged adults who presented with diverse symptoms, including seizures, headache, subcutaneous nodules, limb pain, visual disturbances, and focal neurological deficits. The disease showed extensive multisystem involvement, with neuroimaging demonstrating numerous intracranial lesions at different stages of evolution, while additional cysticerci were identified in skeletal muscles, subcutaneous tissues, and, occasionally, visceral organs such as the lungs and eyes. The authors emphasised that the marked variability in both clinical presentation and organ distribution necessitates comprehensive multimodality imaging to establish a unified diagnosis and to prevent these lesions from being mistaken for neoplastic or other infectious conditions [12].

CONCLUSION(S)

The case report suggests that multisystem cysticercosis, characterised by quiescent end-stage calcified NCC with potentially vesicular stage myocysticercosis and pulmonary involvement, can manifest as a predominantly chronic, non-progressive dysexecutive behavioural syndrome, which resembles major depression. When depressive or behavioural symptoms are unresponsive to antidepressant treatment, and objective executive dysfunction is present, brain imaging must be considered in endemic areas.

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