

Uncommon presentation of neurocysticercosis

Pooja Dewan, MD
 Jaya Shankar Kaushik, MD, DNB
 Sunil Gumber, MD, DCH, DNB, FIAP
 Amit Gupta, MD

Department of Pediatrics, University College of Medical Sciences and Guru Teg Bahadur Hospital, University of Delhi, Delhi, India

Corresponding author: J S Kaushik (jayashankarkaushik@gmail.com)

Non-epileptic manifestations of neurocysticercosis (NCC) include intellectual deterioration, dementia and parkinsonian behaviour. We report on a child with NCC presenting with abnormal choreiform movement. The case report highlights an uncommon presentation of NCC, and also draws attention to an unusual cause of chorea in children from tropical regions.

Neurocysticercosis (NCC) is a common parasitic infection of the central nervous system caused by the pork tapeworm *Taenia solium*. The clinical presentation of NCC in children includes generalised and partial seizures with or without features of raised intracranial pressure. Presentation with a movement disorder, including chorea, is uncommon in NCC. We report a case of NCC of the midbrain in which chorea was a presenting feature.

Case report

A 10-year-old girl presented to the paediatric emergency department of Guru Teg Bahadur Hospital, a tertiary care hospital in Delhi, India, with complaints of abnormal choreiform movement along with slurring of speech and emotional lability for a period of 4 days. The movements were rapid, continuous and jerky, involving all four limbs. They increased with stress and subsided during sleep. There was no history of seizures, loss of consciousness, visual or hearing defects, fever, drug ingestion, head trauma, ear discharge, bleeding from any site, sore throat, joint pains or subcutaneous swellings. The child's development was in accordance with her age. She attended school in the 3rd standard and her scholastic performance was average.

On examination the child was conscious, alert and responsive. Her vital signs, including blood pressure, were stable at admission. General physical examination was non-contributory. There was no evidence of subcutaneous nodules, an erythematous rash or neurocutaneous markers. The findings on ophthalmic evaluation, including fundus examination, were normal, and no Kayser Fleischer ring was present. Higher mental function was normal. All the cranial nerves were intact. Motor examination revealed quadriparesis, with power in the four limbs of 3/5 - 4/5; deep tendon reflexes were sluggish, with extensor plantar responses. There were rapid, jerky, choreiform movements of all the four limbs, with predominantly distal involvement. The clinical signs of rheumatic chorea such as 'milkmaid grip', darting tongue and the pronator sign were present. Findings of the remainder of the systemic examination, including cardiovascular examination, were normal.

Considering the child's epidemiological background, a diagnosis of rheumatic chorea was considered and she was investigated accordingly. She was found to be anaemic (haemoglobin concentration 9.2 g/dl), with a normal white cell count ($5.6 \times 10^9/l$) and platelet count ($160 \times 10^9/l$). The erythrocyte sedimentation rate (12 mm/1st h) and C-reactive protein level (<6 mg/l) was normal. The antistreptolysin O (ASO) titre was negative and electrocardiography revealed a normal P-R interval. Antinuclear antibody and serum ceruloplasmin

levels were also normal. In view of the focal neurological deficits and negative laboratory parameters for rheumatic activity, neuro-imaging was planned. Magnetic resonance imaging (MRI) of the brain (Fig. 1) revealed a 5.2×4.8 mm ring-enhancing lesion involving the right paramedian midbrain with mild perilesional oedema suggestive of active NCC.



Fig. 1. MRI scan of the patient's brain showing ring-enhancing lesion (1) involving the right paramedian midbrain with mild perilesional oedema (2) suggestive of active NCC.

The child was started on steroids and haloperidol (for the chorea). After 3 days of treatment with steroids, albendazole was started. Over the course of the next 24 hours she developed bradycardia, hypertension, a positive Macewen sign, and bilaterally increased tone of the lower limbs with an extensor plantar response suggestive of raised intracranial tension. Albendazole was withdrawn and mannitol and steroids were initiated, after which the features of raised intracranial pressure decreased. Steroids and haloperidol were continued. The choreiform movements improved dramatically within the next 2 weeks. There was no focal neurological defect, and the child went back to school at 3 months' follow-up. However, we were not able to perform repeat neuro-imaging to ascertain the status of the lesion.



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manifestations of NCC
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Discussion

Clinical features in NCC depend on the number and location of the lesions as well as the extent of associated inflammatory response or scarring.¹ NCC presents with seizures or features of raised intracranial pressure in children. Non-epileptic manifestations of NCC include intellectual deterioration, dementia or parkinsonian clinical features.² Chorea is an uncommon.³⁻⁴ To the best of our knowledge there is only a single paediatric case report of NCC presenting with chorea in the literature. This was a report of a child with NCC of the thalamic region presenting with hemichorea.³

Cosentino *et al.*⁴ reported the case of a 22-year-old woman who presented with hemichorea resulting from multiple parenchymal NCC. NCC can have parenchymal, intraventricular, meningeal, spinal or ocular localisation.² However, brainstem involvement is uncommon.⁵⁻⁹ Midbrain involvement in NCC has been reported in 2 children who presented with ptosis (third cranial nerve palsy).⁵

Treatment of NCC of the brainstem remains controversial. Conservative management has been tried to avoid exacerbation of inflammation and clinical deterioration.^{6,8,9} However, active cysticidal treatment with albendazole under the cover of steroids has also been reported to be safe.^{5,7}

This case report highlights two important facts. One is the uncommon presentation of NCC with chorea. The second is that cysticidal treatment of NCC of the midbrain can result in rapid clinical deterioration, and further studies are required before it can be recommended.

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