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Case Report: A case of neurocysticercosis presenting as a movement disorder

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Abstract

Neurocysticercosis (NCC) is the most common parasitic infection of the central nervous system caused by Taenia solium larvae (pork tapeworm) [1-3]. Seizures are the most frequent clinical manifestation; however movement disorders (MDs) can occur in a small percentage of individuals with NCC and are an uncommon presentation that can pose diagnostic challenges.[7-9]

This case describes an elderly male who presented with fever and neck rigidity, PSP was the initial clinical suspicion or a syndromic presentation, not the final diagnosis which showed good response to levodopa. The overlap of acute febrile illness with chronic neurodegenerative signs and neurocysticercosis and myocysticercosis created a diagnostic challenge.

This case highlights the importance of considering various presentations of neurocysticercosis and having neurocysticercosis in patients presenting with movement disorder and in a patient with history of Pork consumption as a differential diagnosis.

Keywords: Neurocysticercosis, Parkinsonism, Myocysticercosis, Progressive Supranuclear Palsy

Introduction

Neurocysticercosis, resulting from infection with the larval stage of Taenia solium [2,3] is a major cause of seizures globally, especially in low-income areas where pig farming is prevalent. Clinically, it presents with seizures, headache, intracranial hypertension, focal deficits, or hydrocephalus, depending on cyst location.[6,7]. Parasitic infections of the central nervous system can result in a range of movement disorders due to inflammation, granuloma formation, or mass effects on motor control regions such as the basal ganglia, brainstem, and cortex. Cerebral malaria, a common cause, is associated with Parkinsonism, dystonia, chorea, ataxia, and spasticity. Toxoplasmosis, particularly in immunocompromised individuals, may cause Parkinsonism and chorea. Rarely, African trypanosomiasis can present with tremors, choreiform or athetoid movements, dysarthria and cerebellar ataxia. In rare cases, echinococcosis (hydatid disease) may cause dyskinetic or pseudoathetotic movements due to mass effect from cysts in motor pathways[2-6].Neurocysticercosis, caused by the larval form of Taenia solium, is the most common parasitic infection of the central nervous system and a leading cause of acquired epilepsy in endemic regions [1]. Although seizures are the predominant clinical manifestation, a diverse spectrum of movement disorders has also been reported. These include parkinsonism, tremor, dystonia, chorea, ataxia, myoclonus, tics, and hemifacial spasm. Movement disorders in neurocysticercosis may arise from lesions in the basal ganglia, thalamus, cerebellum, or brainstem, or secondary to complications such as hydrocephalus. Progressive Supranuclear Palsy (PSP) is a rare neurodegenerative disorder characterized by early postural instability, vertical gaze palsy, axial rigidity, cognitive decline, and poor response to levodopa. Misdiagnosis is common, especially early in the disease. Fever and neck rigidity frequently prompt evaluation for meningitis, posing diagnostic challenges. This case illustrates the difficulty in distinguishing acute infection from chronic neurodegeneration when signs overlap.

Case Presentation

A 70-year-old male with a history of systemic hypertension and a 40-year smoking history was admitted with a ten-day history of intermittent low-grade fever associated with chills and rigors. While the primary complaint was febrile, the patient's history revealed significant neurological decline over the preceding six months, including generalized slowing of movements, recurrent backward falls, and an inability to look downward while reading due to restricted neck movements. He also exhibited fine involuntary tremors in his fingers at rest and a noticeable decrease in arm swing while walking. Behavioral changes were also reported, specifically frequent episodes of crying and intermittent irrelevant speech.[3]

Physical examination revealed a patient who was conscious but displayed a Mini-Mental State Examination (MMSE) score of 10 and emotional lability. Notable motor findings included mask-like facies, hypophonic speech, and a Unified Parkinson's Disease Rating Scale (UPDRS) motor score of 32, indicating moderate severity. The patient exhibited

significant axial rigidity—which was more pronounced than appendicular rigidity—along with bradykinesia and a positive pull test. Cranial nerve assessment highlighted upward gaze restriction and slow saccades, though no other cranial nerve, cerebellar, or sensory deficits were identified. [3]

Initial investigations showed elevated inflammatory markers, including an ESR of 95 and a CRP of 122. A skeletal survey revealed multiple "rice-grain" calcifications in the soft tissues of the pelvis and thighs, characteristic of myocysticercosis. MRI of the brain demonstrated multiple non-enhancing ring-shaped lesions in cortical and subcortical areas, as well as the midbrain, without surrounding edema. Serology was positive, the presence of these imaging findings in a patient with a history of pork consumption led to a confirmed diagnosis of neurocysticercosis according to the revised Del Brutto criteria

Investigations

CBC	29/5	30/5	8/6
TC	12.200	12.900	10.800
DC	91/4.7	79/10	79/12
RBC	4.9	4.1	4.1
HB	14.9	11.8	11.6
PCV	45	37	37.4
MCV	90	89	89
MCH	29.4	30	29.8
PLT	2.8L	2.9L	3.2L
ESR	95	70	40

	29/5	31/5	8/6
RBS	124	101	112
UREA	75	94	63
CREAT	1.97	1.7	1.77
NA+	144	138	143
K+	3.6	3.9	4.3
CRP	122	40	

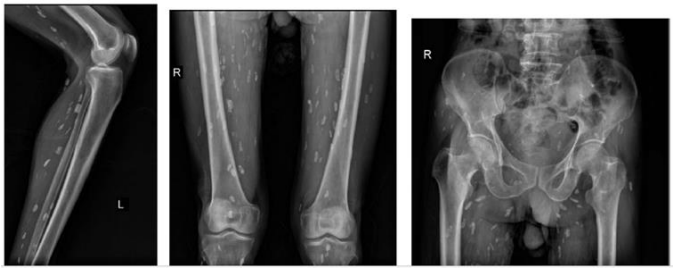
Serology

Cysticercosis IgG – Positive

CSF analysis

The cerebrospinal fluid (CSF) analysis revealed a glucose level of 80 mg/dL and an elevated protein level of 92 mg/dL, while the globulin test remained negative. Cytological examination showed an acellular smear with a cell count of nil, and the Adenosine Deaminase (ADA) level was low at less than 1.0 U/L. Additionally, the CSF culture and sensitivity (C&S) showed no growth after the incubation period.

Xray Skeletel survey



The skeletal survey showing "rice-grain" calcifications in the pelvis and thighs is a critical diagnostic finding for myocysticercosis

MRI Brain

- Grade II small vessel ischemic changes noted
- Age related cerebral cortical atrophic changes

- Chronic lacunar infarct with hemosiderin deposits in left corona radiata, left centrum semiovale, bilateral Basal ganglia and midbrain
- Microhaemorrhages noted in bilateral parietal lobe, left temporal Lobe, right occipital lobe, midbrain
- Few cortical and subcortical ring shaped non - enhancing lesions seen without surrounding edema in cortical and subcortical areas No hydrocephalus.

Diagnostic Criteria — Del Brutto[21]

Neuroimaging (Major) Multiple ring non enhancing lesions seen

Immunologic (Major) Positive serum cysticercosis IgG

Clinical Reversible parkinsonism,

Epidemiologic Pork consumption, endemic region.

Diagnosis: Neurocysticercosis – **probable/confirmed**

Diagnostic criteria
Absolute Histological demonstration of the parasite from biopsy of a brain or spinal cord lesion Evidence of cystic lesions showing the scolex on neuroimaging studies Direct visualization of subretinal parasites by fundoscopic examination
Major Evidence of lesions highly suggestive of neurocysticercosis on neurcimaging studies (CT or MRI) Positive serum immunoblot for the detection of anticysticercal antibodies Resolution of intracranial cystic lesions after therapy with albendazole or praziquantel Spontaneous resolution of small single enhancing lesions
Minor Evidence of lesions compatible with neurocysticercosis on neuroimaging studies Presence of clinical manifestations suggestive of neurocysticercosis Positive CSF ELISA for detection of anticysticercal antibodies or cysticercal antigens Evidence of cysticercosis outside the central nervous system
Epidemiological Individuals coming from or living in an area where cysticercosis is endemic History of travel to disease-endemic areas Evidence of a household contact with T. solium infection
Degrees of diagnostic certainty
Definitive Presence of one absolute criterion Presence of two major plus one minor and one epidemiological criteria
Probable Presence of one major plus two minor criteria Presence of one major plus one minor and one epidemiological criteria Presence of three minor plus one epidemiological criteria

Differential Diagnosis

Idiopathic Parkinson's disease Age of onset, parkinsonism Acute–subacute onset, MRI lesions, rapid recovery

Vascular parkinsonism Falls + bradykinesia No extensive small-vessel ischemia

Wilson Disease Tremor + rigidity Age > 60, normal copper profile

Basal ganglia stroke Movement symptoms Gradual onset, multiple lesions, no acute infarct.

Treatment

The treatment for this case was initiated with a course of Albendazole at a dosage of 15 mg/kg/day administered for 28 days. To manage the host inflammatory response and reduce perilesional edema, Prednisolone was concurrently administered at 1 mg/kg/day and subsequently tapered. Motor symptoms were addressed using Levodopa–Carbidopa, while Levetiracetam was prescribed for seizure prophylaxis. Additionally, the patient underwent physiotherapy focused on improving gait and balance stability.

Outcome of Treatment

Over a period of 6 to 8 weeks, the patient demonstrated significant recovery. A marked improvement in rigidity and bradykinesia was observed after only one week of therapy. By the end of the follow-up period, the patient exhibited improved arm swing and gait, and remained free of seizures.

Discussion

Neurocysticercosis is associated with a remarkably Broad and heterogeneous range of movement disorders, Underscoring its potential to affect multiple motor Control regions within the central nervous system. This systematic review identified both hypokinetic and Hyperkinetic manifestations across varying age groups, Clinical contexts, and anatomical sites. Among hypokinetic Disorders, parkinsonism emerged as the most frequent Presentation, followed by bradykinesia and rigidity—Often resembling idiopathic parkinsonian syndromes and Typically associated with lesions in the basal ganglia or Midbrain. Conversely, hyperkinetic manifestations were More varied, including chorea or hemichorea, hemifacial Spasm, facial dyskinesias, and myoclonus[8-10]. Less common But noteworthy presentations included dystonia, tremor, Ballism, and

asterixis. Several cases involved overlapping. Movement abnormalities, suggesting multifocal central Nervous system involvement. This wide clinical spectrum reflects the ability of neurocysticercosis to imitate Various extrapyramidal disorders, frequently with acute or subacute onset and variable clinical outcomes based On factors such as lesion location, patient age, and Promptness of therapy. The pathogenesis of movement disorders in Neurocysticercosis is multifactorial and depends on the Location, stage, and type of the cystic lesion. Parenchymal Neurocysticercosis, particularly in the basal ganglia, Midbrain, or thalamus, is commonly associated with Hypokinetic disorders such as parkinsonism. In these Cases, cystic or granulomatous inflammation may disrupt The nigrostriatal dopaminergic pathway, leading to Bradykinesia, rigidity, and tremor. Inflammatory edema or immune-mediated neuronal dysfunction—rather than Irreversible damage—is likely responsible in some cases, Explaining the partial or complete response to antiparasitic and steroid therapy. Hyperkinetic movement disorders, on The other hand, are more often associated with subthalamic .Nucleus, thalamic, or caudate lesions, which may produce Chorea, dystonia, or ballism due to disinhibition of Thalamocortical pathways. Intraventricular or subarachnoid Cysts can lead to hydrocephalus or raised intracranial Pressure, secondarily affecting motor circuits. Experimental Studies show that larval products directly excite neurons by Releasing glutamate and aspartate, activating glutamate Receptors and inducing seizure-like activity, suggesting A role in abnormal brain signalling and seizures [8].

In Some cases, movement disorders may also arise from Cortical irritation, epileptogenic spread, or post-infectious Immune responses. Thus, the type of neurocysticercosis Parenchymal, extraparenchymal, intraventricular, or Mixed—determines the pathophysiological mechanism and clinical presentation[8,22,23].

Antiparasitic therapy, particularly albendazole, is The mainstay of treatment for movement disorders Associated with neurocysticercosis, especially in patients with active or parenchymal cystic lesions. Albendazole Is typically administered at a dose of 15 mg/kg/day Generally for 2 to 4 weeks. In approximately 65% of Reviewed cases, it was used either alone or in combination with corticosteroids. Concomitant corticosteroids Help attenuate the host inflammatory response to Degenerating cysticerci, reduce perilesional edema, and prevent clinical worsening during treatment [25].

“In addition to the parasitic load, the MRI revealed Grade II small vessel ischemic changes and microhemorrhages in the bilateral parietal, temporal, and occipital lobes, as well as the midbrain. These vascular factors likely compounded the neurological deficit, contributing to the complexity of the parkinsonian presentation. The coexistence of chronic lacunar infarcts in the basal ganglia and midbrain alongside active neurocysticercosis suggest a multifactorial disruption of the nigrostriatal pathways. This interplay between infectious inflammation and vascular compromise underscores why the clinical picture so closely mimicked the progressive decline seen in neurodegenerative conditions like PSP.”

Hyperkinetic movement disorders, such as hemichorea, Hemiballismus, and myoclonus, generally responded Rapidly and favorably to antiparasitic therapy, often Resolving within days to weeks—particularly when the

Lesions were localized to areas such as the thalamus or Motor cortex and detected in the early inflammatory Phase. In contrast, hypokinetic disorders, particularly Parkinsonism, showed a more variable and often Prolonged clinical course. While some patients improved with adjunctive dopaminergic therapy, especially in The presence of reversible hydrocephalus or midbrain Involvement, others experienced persistent or worsening Symptoms due to irreversible neuronal damage, delayed diagnosis, or complications such as shunt Malfunction or extensive brainstem involvement. In a Reported series of 23 patients with movement disorders [7,11,12]. Due to neurocysticercosis, most achieved complete Recovery following antiparasitic treatment without Requiring long-term therapy. However, parkinsonism Cases were more complex, often necessitating multiple Interventions. Seven patients required repeat courses of albendazole with corticosteroids, eight underwent cyst excision, and eight required ventriculoperitoneal Shunting. Twelve patients received levodopa, though Its benefits were limited, particularly in severe cases. Long-term neurological outcomes ranged widely, with some patients achieving full recovery and others remaining with significant deficits, often accompanied by complications such as epilepsy, cognitive decline, and Intracranial hypertension [7].

Symptomatic treatment is essential, particularly in movement disorders. Hypokinetic patients with Parkinsonism may benefit from dopaminergic therapy, such as levodopa, especially when reversible disruption of the nigrostriatal pathway is suspected. Hyperkinetic Disorders require individualized approaches—Antipsychotics for chorea, benzodiazepines or valproate for myoclonus, and botulinum toxin for focal dystonias or hemifacial spasm. In cases with raised intracranial Pressure due to intraventricular cysts, neurosurgical Intervention like ventriculoperitoneal shunting may be necessary. Most patients respond well to antiparasitic Treatment, with over 80% achieving partial or complete recovery, though delayed diagnosis or calcified lesions may result in persistent symptoms or incomplete resolution [25]

Prognosis

NCC-induced parkinsonism generally has a good prognosis, with 60–80% showing complete or near-complete recovery. Our patient's improvement over 6–8 weeks aligns with published outcomes and supports the reversibility of secondary parkinsonism when timely therapy is provided.

Clinical Significance of This Case

This case underscores several key concepts:

1. Parkinsonism can be a presenting feature of NCC, even without basal ganglia involvement.
2. Neuroimaging is essential for diagnosing atypical parkinsonism in endemic areas.
3. Timely antiparasitic therapy can reverse symptoms, preventing unnecessary long-term dopaminergic therapy.
4. The presence of seizures, cortical lesions, and subacute progression should raise suspicion for secondary causes rather than idiopathic PD.

Conclusion

This case illustrates that Neurocysticercosis (NCC) can present as a "pseudo-PSP" syndrome, characterized by vertical gaze palsy and significant axial rigidity. While the initial clinical presentation strongly suggested a neurodegenerative disorder, the identification of multiple non-enhancing ring lesions on MRI and characteristic

"rice-grain" calcifications on skeletal survey led to a diagnosis of probable NCC.

Unlike idiopathic PSP, which is progressive and poorly responsive to therapy, the movement disorder in this patient was secondary and reversible. The patient achieved significant motor recovery following a targeted regimen of antiparasitic therapy (Albendazole) and steroids, supplemented by short-term Levodopa. This highlights the necessity of neuroimaging and a high index of clinical suspicion for parasitic infections in patients from endemic regions presenting with atypical parkinsonism.

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