

Case Report

ATHETOSIS AS A SEQUELAE OF ACUTE ENCEPHALITIS WITH DEVELOPMENTAL REGRESSION: A CASE REPORT

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ABSTRACT

Background: Acute encephalitis is an inflammatory disorder of the central nervous system that can lead to significant neurological sequelae, particularly in children. Among these, movement disorders such as athetosis are rare but represent profound injury to the basal ganglia and motor control pathways. Post-encephalitic syndromes may present with spasticity, cognitive decline, and loss of previously acquired developmental milestones, profoundly affecting long-term neurodevelopmental outcomes. This case highlights an uncommon presentation of post-encephalitic athetosis associated with developmental regression in a young child following acute viral encephalitis.

Case Presentation: A 4-year-old female child, previously developmentally normal and born of a non-consanguineous marriage, presented with inability to feed herself, sit, stand, or walk for six months following an episode of acute febrile illness with seizures. The child had developed status epilepticus during hospitalization and required non-invasive ventilatory support. She was treated with intravenous acyclovir for 21 days, corticosteroids, and intravenous immunoglobulin due to suspicion of autoimmune encephalitis. After discharge, the child showed persistent abnormal writhing movements of all four limbs, spastic quadriparesis, and severe loss of motor, language, and social milestones. On examination, the child was conscious but bedridden with choreo-athetoid movements more prominent on the right side. Tone was increased in all limbs with exaggerated reflexes and bilateral extensor plantar responses. Cranial nerve evaluation revealed impaired swallowing, regurgitation of feeds, and hearing loss. MRI brain revealed bilateral gyriform thickening and T2/FLAIR hyperintensities with diffusion restriction in both cerebral hemispheres, involving the hippocampus and thalami, consistent with viral encephalitis. The findings were suggestive of cortical and basal ganglia involvement, explaining the observed athetosis and developmental regression.

Results: The child was managed symptomatically with sodium valproate (40 mg/kg/day), levetiracetam (40 mg/kg/day), and clobazam (0.45 mg/kg/dose), along with physiotherapy and nutritional support. She remained seizure-free on follow-up, though persistent choreo-athetoid movements and spastic quadriparesis were noted. Developmental recovery was limited despite rehabilitation measures, indicating irreversible post-encephalitic neurological sequelae.

Discussion: Athetosis as a sequela of encephalitis is an uncommon but severe manifestation, typically resulting from inflammatory or ischemic injury to the basal ganglia. The coexistence of spasticity and athetosis in this child suggests

combined involvement of pyramidal and extrapyramidal systems. Similar outcomes have been reported in post-encephalitic states, particularly in viral or autoimmune etiologies, where neuronal damage leads to abnormal dopaminergic signaling. The diagnostic challenge lies in differentiating post-viral from autoimmune encephalitis, especially in resource-limited settings where antibody testing may be unavailable. Early recognition of such sequelae and institution of multidisciplinary rehabilitation are essential for improving quality of life.

Conclusion: This case emphasizes the rare occurrence of post-encephalitic athetosis with developmental regression in a child with acute viral encephalitis. The combination of cortical and basal ganglia injury leads to persistent neurological deficits, underscoring the need for early neuroimaging, timely antiviral and immunomodulatory therapy, and long-term neurorehabilitation follow-up to minimize morbidity and enhance recovery potential.

Keywords: Athetosis, Acute Encephalitis, Developmental Regression.

INTRODUCTION

Acute encephalitis represents a significant neurological emergency in pediatric medicine, characterized by the acute onset of fever and altered sensorium resulting from inflammation of brain parenchyma. It encompasses a wide spectrum of infectious and immune-mediated etiologies, leading to high morbidity and mortality despite aggressive medical therapy. Globally, the incidence of encephalitis in children is estimated at 3.5–7.4 per 100 000 population per year, with viral causes such as Japanese encephalitis, herpes simplex virus, enteroviruses, and autoimmune encephalitis constituting major contributors. The clinical picture varies from mild confusion to profound coma, frequently accompanied by seizures, focal neurological deficits, and abnormal involuntary movements depending on the site and extent of central-nervous-system (CNS) involvement.^[1]

A critical determinant of neurological outcome following acute encephalitis is the degree of cortical and basal-ganglia injury. Post-encephalitic movement disorders, though uncommon, represent a devastating complication in survivors. Among these, athetosis—characterized by slow, writhing, involuntary movements primarily involving the distal extremities—is particularly rare and typically signifies basal-ganglia dysfunction, especially of the putamen and globus pallidus. The pathophysiology of post-encephalitic athetosis involves neuronal loss, gliosis, and altered dopaminergic and GABAergic neurotransmission secondary to the inflammatory insult. Lesions of the basal ganglia detected on neuroimaging correlate strongly with subsequent movement-disorder manifestations.^[2]

In pediatric patients, acute viral encephalitis often produces heterogeneous outcomes. While some recover completely, others exhibit developmental regression, seizures, or movement abnormalities such as chorea, dystonia, or athetosis. Developmental regression—loss of previously acquired milestones in motor, language, or social domains—reflects higher-order cortical injury and denotes poor neurocognitive prognosis. The concurrence of

developmental regression and athetosis following encephalitic illness underscores simultaneous involvement of both cortical and extrapyramidal structures, posing substantial challenges in rehabilitation and long-term care.^[3]

Autoimmune mechanisms have increasingly been recognized in the etiology of encephalitis with movement disorders. Anti-N-methyl-D-aspartate (anti-NMDA) receptor encephalitis, for instance, may present with dyskinesias and behavioral changes even in the absence of overt infection. However, in resource-limited settings, viral encephalitis continues to predominate, with autoimmune causes often underdiagnosed. The diagnostic approach therefore mandates careful clinical correlation supported by neuroimaging, cerebrospinal-fluid analysis, and exclusion of metabolic or degenerative disorders.^[4]

Several reports describe post-encephalitic movement disorders in children, but isolated athetosis with global developmental regression remains exceedingly uncommon. Historical accounts such as von Economo's encephalitis lethargica epidemic (1917-1928) first highlighted the association of encephalitis with extrapyramidal sequelae, including parkinsonism and athetosis. Modern case series continue to note basal-ganglia-predominant lesions as a predictor of poor neurological recovery. In the Indian subcontinent, studies of acute encephalitis syndrome (AES) indicate diverse etiologies but seldom document choreo-athetoid movement disorders as chronic sequelae.^[5]

The present case is reported because of its unusual combination of post-encephalitic athetosis, spastic quadriparesis, and profound developmental regression in a 4-year-old girl following a presumed viral encephalitic illness. The case emphasizes the diagnostic complexity, the characteristic neuroimaging findings suggesting cortical and basal-ganglia involvement, and the long-term neurological consequences of AES in the pediatric population. Early recognition and comprehensive rehabilitation strategies are essential to mitigate disability and improve quality of life in such survivors.

CASE DESCRIPTION

A 4-year-old female child, C. Kasturi Anuradha, born of a non-consanguineous marriage, was brought by her mother to the outpatient department of the Department of Pediatrics, Konaseema Institute of Medical Sciences (KIMS), Kakinada, for evaluation of inability to feed herself, sit, stand, or walk for the past six months (since March 2023). The mother served as the informant and provided a reliable history.

History of Present Illness: The child was developmentally normal until 6 March 2023, when she developed high-grade fever associated with headache. She was initially treated at a private hospital. On the third day of illness she developed up-rolling of eyes and frothing from the mouth, followed by a generalized tonic-clonic seizure and loss of consciousness. She was admitted and managed as status epilepticus. Neuroimaging (CT and MRI brain) was performed. Subsequently, she was referred to Government General Hospital (GGH), Kakinada, where she required non-invasive ventilation for ten days, later transitioned to nasal prongs for another nine days.

During hospitalization she received intravenous acyclovir for 21 days, intravenous prednisolone for 5 days followed by oral prednisolone for 15 days, and supportive therapy. On the 21st day of admission, she developed abnormal involuntary movements of all four limbs (right > left), along with inability to sit, stand, walk, or feed herself, which she had previously performed independently. She was treated with Levetiracetam (Levipil); due to recurrent seizures, Sodium Valproate was added, titrated to 60 mg/kg/day, and Clobazam was introduced. Considering possible autoimmune encephalitis, intravenous immunoglobulin (IVIG) was administered for 3 days. After partial stabilization, she was discharged on antiepileptic medication and presented to KIMS OPD for follow-up and further assessment.

Developmental and Family History: All developmental milestones had been attained appropriately prior to the illness. Following the encephalitic episode, global developmental regression was noted:

Gross motor: loss of head control and ability to sit or stand.

Fine motor: loss of bidextrous grasp.

Social: loss of social smile and interaction.

Language: loss of cooing and monosyllabic speech. Thus, her developmental quotient could not be calculated. There was no family history of neurological disorders. Antenatal, natal, and postnatal histories were uneventful, and there was no history of birth asphyxia or kernicterus. The child was exclusively breast-fed for six months and currently receives a mixed diet with an estimated calorie intake of 870 kcal/day (expected 1300 kcal).

General and Systemic Examination: On examination, the child was conscious but bedridden,

with all four limbs extended at rest and abnormal writhing (choreo-athetoid) movements of upper and lower limbs more prominent on the right side when awake; movements subsided during sleep. Contractures at tendo-Achilles were noted. No dysmorphic features or neurocutaneous markers were seen.

Anthropometry: weight 12 kg (< 3rd centile), height 97 cm (3rd–15th centile), head circumference 48 cm (15th–50th centile).

Vital signs: temperature 98.5 °F, heart rate 94/min, respiratory rate 24/min, SpO₂ 98 % (RA), BP 80/60 mm Hg.

Central Nervous System

Higher mental functions: attention, memory, communication, and cognition were globally impaired; child failed to recognize mother.

Cranial nerves: CN II—no response to bright light, menace reflex present; CN V—jaw clenching present; CN VII—no facial asymmetry; CN VIII—hearing impairment; CN IX–X—swallowing impairment with regurgitation; CN XII—assessment limited by jaw clenching.

Motor system: generalized hypertonia (spastic quadriparesis) with tone increase (right > left). Power: upper limbs 2+/5, lower limbs 1+–2+/5.

Reflexes: deep-tendon reflexes exaggerated; ankle clonus present; bilateral extensor plantar responses.

Gait: not elicitable.

Other systems: cardiovascular — S₁S₂ present, no murmurs; respiratory — bilateral air entry +, no added sounds; abdomen — soft, non-tender, no organomegaly.

Investigations

Laboratory findings: Hemoglobin 9.5 g/dL, HCT 29 %, MCV 69 fL, WBC 13 100/mm³ (N 90 %, L 6 %, M 4 %), platelets 3.51 × 10⁵/mm³, ESR 102 mm/h, C-reactive protein 62.97 mg/L. Mantoux test and malarial parasite smear were negative.

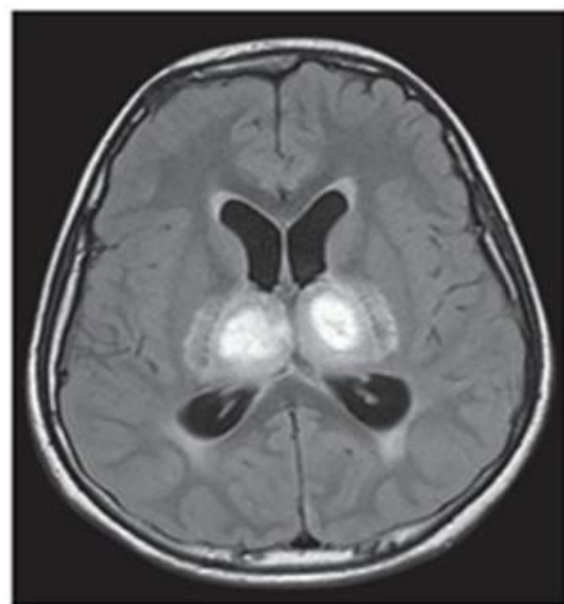


Figure 1: MRI Brain T₂/FLAIR images showing bilateral cortical hyperintensities;

Neuroimaging

CT Brain: multiple tiny (3–5) calcified granulomas in right parietal and occipital lobes, largest 3×3 mm, one with mild perilesional edema.

MRI Brain: large areas of gyriform thickening and T₂/FLAIR hyperintensity with diffusion restriction in both cerebral hemispheres; involvement of left mesial temporal lobe, hippocampus, and posteromedial thalami with diffuse sulcal effacement—suggestive of viral encephalitis.

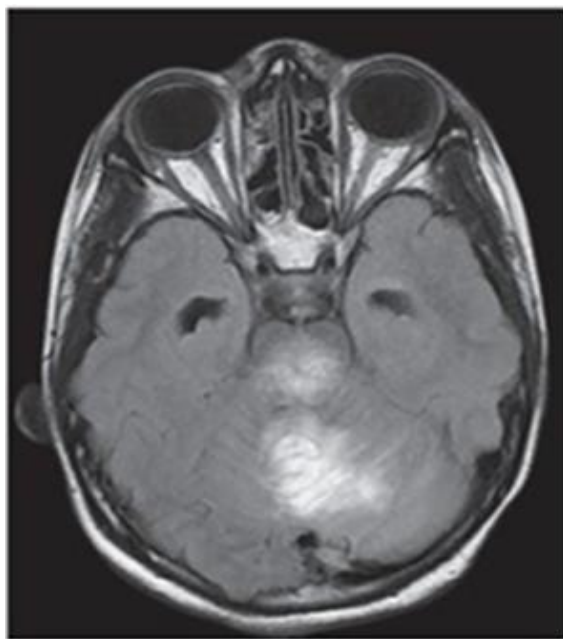


Figure 2: CT Brain demonstrating right-sided calcified granulomas.

Diagnosis and Differential Considerations

Based on the history, examination, and investigations, the following differential diagnoses were considered:

Sequelae of acute viral encephalitis — most likely, given acute febrile illness, seizures, and MRI findings.

Sequelae of autoimmune encephalitis — possible, supported by partial response to IVIG.

Neurodegenerative disorder — less likely due to abrupt onset and stable course.

The final diagnosis was choreo-athetosis as a sequela of acute encephalitis with developmental regression, secondary to significant cortical and basal-ganglia involvement.

Management and Follow-Up

Current pharmacotherapy included:

Sodium Valproate (40 mg/kg/day) – 2-0-1 schedule.

Levetiracetam 500 mg (40 mg/kg/day) – ½ tab morning and ½ tab evening.

Clobazam 5 mg (0.45 mg/kg/dose) – 1-0-1.

Oral prednisolone taper completed; maintenance discontinued.

Nutritional support via orogastric feeds; physiotherapy and contracture-prevention exercises initiated.

Regular monitoring of vitals, antiepileptic drug levels, and neurological status was advised.

The patient was referred to pediatric neurology and rehabilitation for ongoing management.

At follow-up, the child remained seizure-free but continued to have persistent athetotic movements and profound motor and cognitive regression, indicating irreversible post-encephalitic sequelae.

DISCUSSION

This case underscores the complex neurological aftermath that can follow acute encephalitic illness in early childhood, emphasizing how severe cortical and basal ganglia involvement may culminate in both developmental regression and persistent movement disorders such as athetosis. The 4-year-old child described here presented with post-encephalitic sequelae marked by spastic quadriplegia, choreo-athetoid movements, and profound loss of previously acquired developmental milestones. Such a constellation of features, though uncommon, represents a devastating outcome of acute viral or autoimmune encephalitis in the pediatric population. Encephalitis, by definition, involves inflammation of the brain parenchyma, leading to widespread neuronal injury. The clinical spectrum ranges from acute febrile illness with altered sensorium to chronic neurological disability. In India, Acute Encephalitis Syndrome (AES) remains a major public-health problem, with viruses such as Japanese encephalitis virus (JEV), herpes simplex virus (HSV), and enteroviruses among the most prevalent agents Xavier JC et al (2024).^[6] Post-encephalitic sequelae arise from direct cytopathic damage, immune-mediated demyelination, or secondary gliosis affecting critical motor and cognitive circuits. The presence of basal ganglia lesions, as demonstrated in this case's MRI, provides an anatomical correlate for the patient's athetotic movements and developmental regression.

Athetosis represents a specific form of hyperkinetic movement disorder characterized by slow, involuntary, writhing movements predominantly affecting distal extremities. It reflects dysfunction of the extrapyramidal system, particularly the putamen and globus pallidus. The pathophysiology involves disordered dopaminergic and GABAergic neurotransmission secondary to neuronal injury or gliosis within basal ganglia circuits Donnellan EP et al (2024).^[7] In post-encephalitic states, such injury often occurs due to viral neurotropism or immune-mediated inflammation directed against basal ganglia neurons. The clinical pattern seen in this child—choreoathetoid movements that disappear during sleep—strongly supports basal ganglia localization rather than cortical myoclonus.

MRI findings in the present case revealed gyriform thickening and T₂/FLAIR hyperintensity in both cerebral hemispheres, along with involvement of the hippocampus and posteromedial thalami—hallmarks

of viral encephalitis. These findings are consistent with prior literature describing selective vulnerability of gray-matter structures to viral infection Jain R et al (2021).^[8] The additional presence of small calcified granulomas in the occipital and parietal lobes could represent incidental healed neurocysticercosis, but the lack of edema or mass effect makes it less likely to explain the acute neurological presentation. Thus, the neuroimaging strongly points to encephalitic etiology with secondary basal ganglia involvement as the substrate for movement disorder.

The loss of previously acquired developmental milestones in gross motor, fine motor, language, and social domains constitutes global developmental regression, suggesting significant cortical damage. This aligns with earlier studies indicating that 20–30% of children surviving severe encephalitis exhibit long-term cognitive and motor deficits, with cortical involvement being the most important determinant de Oliveira LF et al (2024).^[9] The coexistence of cognitive impairment, pseudo-bulbar palsy, and spasticity in our case supports diffuse cortical and subcortical pathology rather than a focal lesion.

A diagnostic challenge in such cases lies in differentiating between post-viral and autoimmune encephalitis. Autoimmune encephalitis, such as anti-NMDA receptor encephalitis, frequently manifests with abnormal movements, seizures, and behavioral disturbances, sometimes mimicking post-viral states. The administration of intravenous immunoglobulin (IVIG) in this patient was therefore justified, particularly in the absence of definitive viral isolation. While the partial clinical stabilization after IVIG suggests a possible immune-mediated component, the persistence of athetosis and regression highlights irreversible structural damage. The lack of cerebrospinal fluid (CSF) antibody testing in resource-limited settings represents a diagnostic limitation, as early immunotherapy may improve outcomes in confirmed autoimmune cases Ozlu C et al (2024).^[10]

Several published reports have described post-encephalitic movement disorders in children. Dale Jafarpour S et al (2022),^[11] observed that 8% of pediatric encephalitis survivors developed extrapyramidal manifestations, predominantly chorea and dystonia, and only a few presented with isolated athetosis. Similarly, Felipe BE et al (2024),^[12] noted that basal ganglia involvement on imaging correlated strongly with the occurrence of movement disorders and poorer cognitive recovery. The present case supports these findings, illustrating how bilateral basal ganglia injury can produce severe hyperkinetic movements accompanied by developmental regression.

From a therapeutic standpoint, management of post-encephalitic athetosis is largely symptomatic. Antiepileptic drugs such as valproate, levetiracetam, and clobazam were used in this case to control seizures and modulate abnormal movements. While agents like tetrabenazine or baclofen have been used

for refractory choreoathetosis, their safety in young children remains limited. The cornerstone of long-term care remains multidisciplinary rehabilitation, encompassing physiotherapy, speech therapy, and occupational interventions aimed at preventing contractures and optimizing residual motor function Rezende LV et al (2024).^[13] Nutritional support, as initiated through orogastric feeding in this case, is equally critical given the high metabolic demands of hyperkinetic states and feeding difficulties due to pseudo-bulbar involvement.

The clinical reasoning in this case rested on correlating a temporal sequence of acute febrile encephalopathy with subsequent motor and cognitive decline. The exclusion of perinatal hypoxia, kernicterus, or genetic/metabolic disorders strengthened the attribution to post-encephalitic etiology. Moreover, the characteristic disappearance of abnormal movements during sleep, together with MRI evidence of bilateral basal ganglia lesions, further supported the diagnosis of post-encephalitic choreoathetosis. The possibility of a coincident neurodegenerative process remains remote due to the abrupt onset and plateauing of symptoms over months rather than progressive deterioration. Le Roux M et al (2021).^[14]

Nevertheless, certain limitations merit acknowledgment. First, the absence of serial MRI follow-up precluded assessment of lesion evolution and recovery potential. Second, detailed CSF or serologic testing for viral and autoimmune markers was unavailable, limiting etiological specificity. Finally, long-term neuropsychological assessment was not feasible at the time of reporting, leaving residual cognitive function incompletely quantified. Despite these constraints, the case provides valuable insights into the natural history of post-encephalitic sequelae in resource-limited settings.

The implications of this case for clinical practice are twofold. First, it reinforces the need for early recognition and aggressive management of acute encephalitis, including timely initiation of antiviral or immunomodulatory therapy. Second, it underscores the importance of long-term neurological follow-up in survivors, focusing on early detection of developmental regression and institution of neuro-rehabilitation strategies. From a research perspective, larger prospective studies correlating MRI findings, immune profiles, and long-term neurobehavioral outcomes could elucidate predictive markers for post-encephalitic sequelae, guiding targeted interventions.

CONCLUSION

This case illustrates the devastating sequelae of acute encephalitis in a young child, manifesting as choreo-athetoid movements, spastic quadriparesis, and global developmental regression secondary to combined cortical and basal ganglia injury. The clinical and imaging findings demonstrate how

severe encephalitic inflammation can disrupt both pyramidal and extrapyramidal pathways, resulting in persistent motor and cognitive deficits. Early recognition of encephalitis, prompt antiviral and immunomodulatory therapy, and vigilant neurological follow-up are vital to minimize such outcomes. Despite the absence of curative treatment for post-encephalitic athetosis, multidisciplinary rehabilitation focusing on physiotherapy, speech, and occupational therapy can significantly enhance functional recovery and quality of life. This case emphasizes the importance of integrating neuroimaging, clinical evaluation, and long-term rehabilitation in managing pediatric encephalitic sequelae and highlights the need for further research into early biomarkers predicting adverse neurological outcomes.

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