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Case Report

Acute Necrotizing Encephalitis Following Influenza A Infection in a Child: A Rare Radiological Case Report

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Abstract

Acute necrotizing encephalitis (ANE) is a rare and severe neurological complication predominantly affecting children, typically following viral infections, most commonly Influenza A. It is characterized by bilateral and symmetric brain lesions, mainly involving the thalami, with possible hemorrhagic transformation. We report the case of a previously healthy 5-year-old girl admitted to the intensive care unit for acute febrile altered consciousness occurring two days after a flu-like illness. Nasopharyngeal PCR was positive for Influenza A virus. Brain computed tomography (CT) revealed bilateral symmetric hypodense thalamic lesions with brainstem involvement. Magnetic resonance imaging (MRI) confirmed the diagnosis by demonstrating bilateral thalamic and subcortical signal abnormalities with diffusion restriction and hemorrhagic components on susceptibility-weighted imaging. This case highlights the crucial role of imaging, particularly MRI, in the early diagnosis of this rare but potentially fatal condition.

Keywords: *Acute Necrotizing Encephalitis; Influenza A; Pediatric Neuroradiology; Thalamus; MRI; Post-Viral Encephalopathy*

Introduction

Acute necrotizing encephalitis (ANE) is a rare clinico-radiological entity first described by Mizuguchi in 1995 [1]. It primarily affects children and occurs following common viral infections, most frequently Influenza A and B [2]. ANE is thought to result from an excessive immune-mediated inflammatory response rather than direct viral invasion of the central nervous system. Imaging plays a pivotal role in diagnosis, with bilateral symmetric thalamic lesions being the hallmark finding.

Case Presentation

A 5-year-old girl with no prior medical history was admitted for acute febrile altered consciousness occurring two days after a flu-like illness. Nasopharyngeal PCR testing was positive for Influenza A virus. Emergency brain CT demonstrated bilateral symmetric hypodense lesions involving both thalami with extension to the brainstem. Subsequent brain MRI revealed bilateral and symmetric thalamic and subcortical signal abnormalities, hyperintense on T2-weighted and FLAIR images, hypointense on T1-weighted images, with marked diffusion restriction and hemorrhagic components on SWI. MR spectroscopy showed no metabolic abnormalities. These findings were consistent with acute necrotizing encephalopathy of likely viral origin.

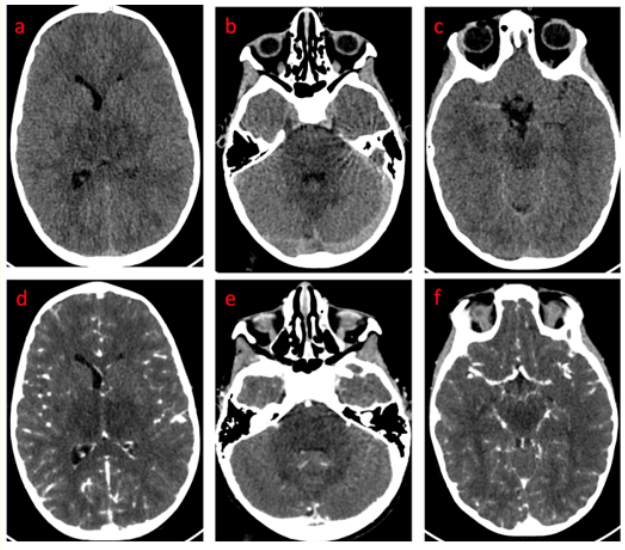

Figure 1: *CT scans before (a; b; c) and after contrast administration (d; e; f) showing bilateral, symmetric mass-like hypodensity involving the thalamic nuclei, the posterior capsulo-lenticular regions, the mesencephalic and pontine structures, and the middle cerebellar peduncles.*

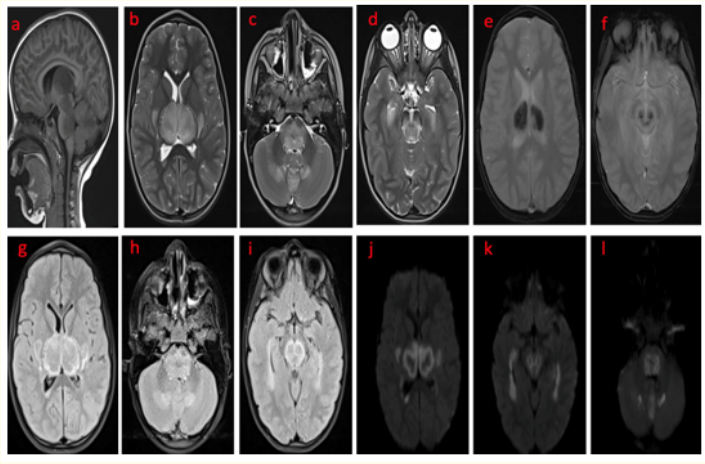

Figure 2: *MRI sequences including sagittal T1 (a) -weighted, axial T2 (b, c, d)-weighted, FLAIR (g, h, i), diffusion-weighted imaging (j, k, l), and T2 gradient-echo images (e, f) showing bilateral and symmetric signal abnormalities involving both thalami, which appear swollen, the lenticular nuclei, the mesencephalon, the pons, and the middle cerebellar peduncles, as well as the periventricular frontal, temporal, and occipital white matter. These lesions are isointense on T1-weighted images, hyperintense on T2-weighted and FLAIR images, show diffusion restriction, and contain hemorrhagic foci appearing as signal voids on susceptibility-weighted sequences.*

Treatment with intravenous immunoglobulins was initiated; however, no clinical improvement was observed, and the disturbance of consciousness persisted.

A follow-up brain MRI performed 10 days later demonstrated worsening of the previously described lesions.

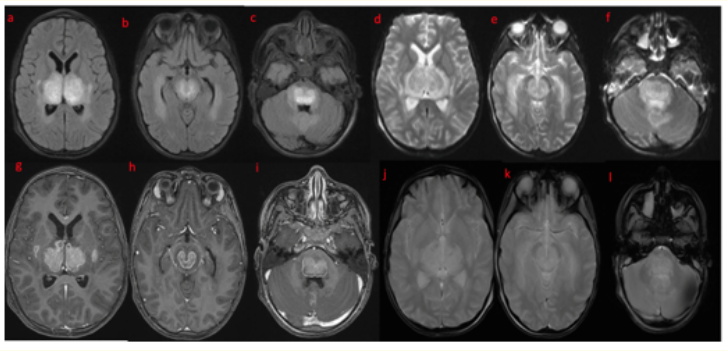

Figure 3: *Control MRI 10 days later showing an increase in the previously described lesions.*

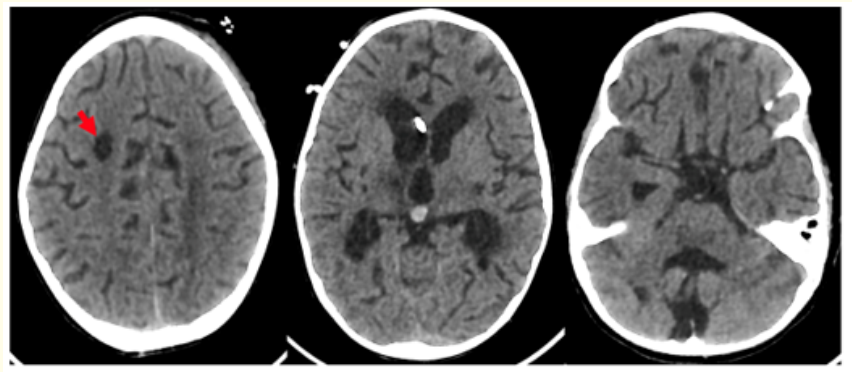

Figure 4: *Follow-up brain CT scan at 1 month showing the development of cystic fluid-filled cavities in the bilateral frontal white matter (red arrow), right occipital region, bilateral periventricular parietal regions, and bilateral cerebellar hemispheres, associated with cerebral edema and active shunted quadriventricular hydrocephalus, with persistence of the previously described necrotizing encephalitis lesions.*

Discussion

Acute necrotizing encephalitis (ANE) is a rare but life-threatening pediatric encephalopathy, most commonly triggered by viral infections such as Influenza A [3,4]. Unlike classical viral encephalitis, ANE is thought to result from a hypercytokinemic response leading to blood-brain barrier disruption, cerebral edema, microvascular injury, and necrotic-hemorrhagic lesions, rather than direct viral invasion [3,4,11]. Genetic susceptibility, particularly RANBP2 mutations, has been reported in familial or recurrent cases, suggesting that host factors may influence disease severity [5,12].

Neuroimaging plays a pivotal role in diagnosis. Bilateral symmetric thalamic involvement is the most consistent imaging feature and is often accompanied by lesions in the brainstem, basal ganglia, cerebellar peduncles, and subcortical white matter [6-8]. Computed

tomography may reveal hypodense thalamic lesions in the acute phase but can be normal early on, whereas MRI is the modality of choice, demonstrating T2/FLAIR hyperintensity, restricted diffusion, and hemorrhagic components on susceptibility-weighted imaging [7,8,13]. Hemorrhagic lesions and brainstem involvement have been associated with poor prognosis [9,14].

In our patient, despite early initiation of intravenous immunoglobulin therapy, no clinical improvement was observed, and follow-up MRI performed 10 days later revealed progression of the previously described lesions. Such clinico-radiological dissociation has been previously documented and likely reflects ongoing cytokine-mediated injury or the aggressive natural course of ANE [15-17]. This emphasizes that immunoglobulin therapy alone may be insufficient, particularly in cases with extensive bilateral thalamic involvement or hemorrhagic changes. Combined immunomodulatory strategies, including high-dose corticosteroids, plasma exchange, or cytokine-targeted therapies, have been proposed in severe ANE, although current evidence is limited to case reports and small series [11,16,18].

The main differential diagnoses include viral encephalitis, deep cerebral venous thrombosis, metabolic or mitochondrial encephalopathies, and acute disseminated encephalomyelitis. However, the combination of acute post-viral onset, characteristic MRI findings, and normal MR spectroscopy strongly supports the diagnosis of ANE. Early recognition by radiologists and pediatric clinicians is essential, as timely diagnosis may allow prompt supportive and immunomodulatory interventions, which can potentially improve outcomes [10,15].

Conclusion

Acute necrotizing encephalitis following Influenza A infection is a rare but devastating pediatric condition. Recognition of its characteristic MRI features particularly bilateral symmetric thalamic lesions with diffusion restriction and hemorrhagic components is crucial for early diagnosis. This case illustrates that disease progression can occur despite immunoglobulin therapy, highlighting the aggressive nature of ANE and the need for close clinico-radiological monitoring. Early identification of poor prognostic indicators, such as brainstem involvement and hemorrhagic transformation, may guide more aggressive or combined immunomodulatory strategies, including corticosteroids or plasma exchange. Further research is needed to establish optimal therapeutic approaches and improve outcomes in this rare but severe disease.

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