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UNMASKING PARECHOVIRUS ENCEPHALITIS – A RADIOLOGIC ODYSSEY THROUGH THE BRAIN.

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ABSTRACT

This case report describes the radiological findings in a 5-month-old infant diagnosed with parechovirus encephalitis, caused by human parechovirus type 3 (HPeV-3). The patient presented with fever, seizures, and altered mental status, with brain magnetic resonance imaging (MRI) revealing characteristic findings. This report emphasizes the radiological manifestations of HPeV encephalitis, which can aid in diagnosis and differentiation from other causes of pediatric encephalitis.

INTRODUCTION

Neonatal meningoencephalitis, which frequently presents with subtle symptoms resembling sepsis, is increasingly linked to Human parechovirus type 3 (HPeV-3). This infection often exhibits normal cerebrospinal fluid (CSF) cell counts and protein levels, making diagnosis challenging and potentially delaying treatment.1-4 HPeV-3 primarily affects the white matter in neonates by targeting glial cells and immature oligodendrocytes, leading to inflammation, demyelination, and edema. MRI typically reveals restricted diffusion in the periventricular and subcortical white matter, especially in the frontal regions, even when CSF results are normal. This case report confirmed through PCR testing, underscores the importance of combining imaging techniques with molecular diagnostics for prompt identification of HPeV-3 infections in neonates.

CASE STUDY

A 5-month-old previously healthy male infant presented to the emergency department with a 2-day history of high-grade fever (39.2°C), irritability, and new-onset generalized tonic-clonic seizures. The parents reported a prodrome of mild upper respiratory symptoms and decreased oral intake over the preceding three days. On examination, the infant was lethargic. Vitals were within normal limits.

Neurological examination revealed no focal deficits, but the anterior fontanelle was tense, suggesting possible increased intracranial pressure. No rash or meningeal signs were observed. Laboratory findings included a white blood cell count of 11,500/μL (normal: 6,000–17,500/μL) with 65% neutrophils, C-reactive protein of 18 mg/L (normal: <10 mg/L), and normal electrolytes. Cerebrospinal fluid (CSF) analysis showed pleocytosis (150 white cells/μL, 75% lymphocytes) and elevated protein (85 mg/dL, normal: 15–45 mg/dL)CSF Gram stain and bacterial culture were negative. Real-time polymerase chain reaction (PCR) testing of CSF was negative for herpes simplex virus (HSV) and enterovirus but positive for HPeV-3.

Brain MRI was performed on hospital day 2 using a 3T scanner. Diffuse T2 /FLAIR hyperintensities were noted in the bilateral frontal, temporal, and periventricular white matter, with predominant involvement of the subcortical white matter. Hypointense areas corresponding to the T2 hyperintensities were seen in T1 weighted imaging. On Diffusion weighted imaging , restricted diffusion in the affected white matter regions, particularly in the frontal lobes.MR spectroscopy showed elevated lactate peak and reduced N-acetylaspartate (NAA) in the affected regions. No cortical gray matter involvement or cerebellar abnormalities were noted. The ventricles appeared normal in size, with no evidence of hydrocephalus.

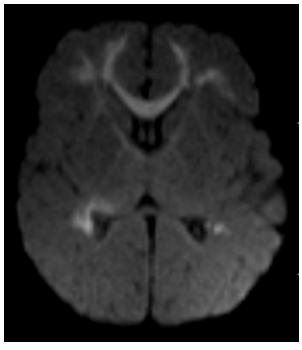

Figure 1.1

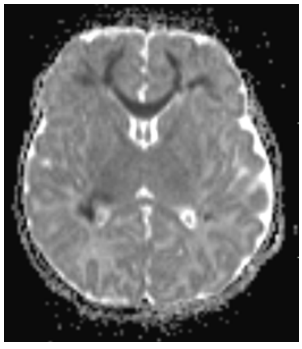

Figure 1.2

Figure 1.1 , 1.2 : MRI brain axial sections of DWI and ADC sequences showing near symmetrical ill-defined areas of diffusion restriction with corresponding signal changes in T1/T2 sequences along the subcortical white matter fibers of bilateral frontal, parietal and anterior temporal lobes.

The patient was empirically started on IV Acyclovir (20 mg/kg every 8 hours) and Ceftriaxone (50 mg/kg every 12 hours) pending diagnostic results. Levetiracetam (20 mg/kg loading dose, followed by 10 mg/kg every 12 hours) was initiated for seizure control. After HPeV-3 was confirmed, antibiotics and acyclovir were discontinued. Supportive care included IV fluids, antipyretics, and monitoring for intracranial pressure.

DISCUSSION

The radiological findings in HPeV encephalitis, particularly due to HPeV-3, are distinct and primarily involve the white

matter, unlike other viral encephalitides (e.g., HSV, which often affects the temporal lobes and gray matter). The diffuse T2/FLAIR hyperintensities and restricted diffusion in the subcortical and periventricular white matter are characteristic of HPeV-3 encephalitis, reflecting its neurotropism for white matter tracts. The absence of contrast enhancement differentiates it from conditions like acute disseminated encephalomyelitis (ADEM), which may show patchy enhancement. MRS findings of elevated lactate and reduced NAA further support neuronal injury, consistent with acute encephalitis.

These imaging features are critical for diagnosis, especially when clinical symptoms are nonspecific. The predominance of white matter involvement in HPeV encephalitis may be related to the virus's tropism for developing white matter in infants, potentially due to immature myelination. The resolution of MRI abnormalities on follow-up aligns with the generally favorable prognosis in many HPeV cases, though severe cases may result in long-term sequelae such as developmental delay or epilepsy.

CONCLUSIONS

The radiological manifestations of HPeV-3 encephalitis, including diffuse white matter T2/FLAIR hyperintensities, restricted diffusion, and MRS abnormalities, are key diagnostic features in infants presenting with fever, seizures, and altered mental status. Brain MRI is essential for confirming the diagnosis and guiding management by differentiating HPeV encephalitis from other causes of pediatric CNS infection. Clinicians should consider HPeV in the differential diagnosis and ensure CSF PCR testing includes HPeV when imaging suggests white matter involvement.

REFERENCES:

[1] Verboon-Maciolek MA, et al. "Human parechovirus causes encephalitis with white matter injury in neonates." *Annals of Neurology*, 2008;64(3):266-273.
[2] Amarnath C, et al. "Neuroimaging findings in parechovirus encephalitis: A case series of pediatric patients." *Pediatric Radiology*, 2019;49(5):614-621.
[3] Harvala H, et al. "Human parechoviruses: Biology, epidemiology, and clinical significance." *Journal of Clinical Virology*, 2010;49(1):1-7.