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CENTRAL NERVOUS SYSTEM INVOLVEMENT IN PEDIATRIC HEMOLYTIC UREMIC SYNDROME: FREQUENCY, IMAGING PATTERNS, AND CLINICAL OUTCOMES

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ABSTRACT

Background: Hemolytic uremic syndrome (HUS) is the leading cause of typical thrombotic microangiopathy in children, most commonly triggered by Shiga toxin–producing Escherichia coli (STEC). Beyond the classic triad of microangiopathic hemolytic anemia, thrombocytopenia, and acute kidney injury, central nervous system (CNS) involvement is a major determinant of severity.
Methods: Narrative synthesis of clinical, epidemiologic, and neurodiagnostic literature addressing pediatric HUS with CNS involvement.
Results: Neurological complications in STEC-HUS occur in approximately the low-teens in contemporary cohorts and most often present with seizures and encephalopathy; PRES, focal deficits, movement disorders, and cerebrovascular complications are less frequent but clinically significant. STEC-HUS is initiated by Shiga toxin–mediated endothelial injury leading to thromboinflammation and microvascular thrombosis, while complement activation likely amplifies endothelial damage and contributes to CNS vulnerability. In aHUS, neurological involvement is more frequent and reflects systemic complement-mediated microangiopathy. MRI, particularly diffusion-weighted imaging and ADC mapping helps differentiate microangiopathic injury from PRES or infarction/hemorrhage and may support early risk stratification. EEG is useful for detecting epileptic activity and subclinical encephalopathy. Management is predominantly supportive and neurocritical (seizure control, blood pressure management, correction of metabolic derangements, and renal support). Eculizumab is essential in aHUS but remains controversial in STEC-HUS, with no consistent neurological benefit over standard care in recent syntheses.
Conclusions: CNS involvement identifies a high-risk pediatric HUS subgroup requiring early neurologic assessment, targeted MRI/EEG use, meticulous critical care, and structured long-term follow-up due to potential subtle neurocognitive sequelae.

KEYWORDS

HUS, CNS, Pediatrics

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1. Introduction

Hemolytic uremic syndrome (HUS) is the leading cause of "typical" thrombotic microangiopathy (TMA) in children and is most commonly triggered by Shiga toxin-producing Escherichia coli (STEC) [1]. In pediatric patients, hemolytic uremic syndrome (HUS) is characterized by microangiopathic hemolytic anemia, thrombocytopenia, and acute kidney injury (AKI). Management primarily involves supportive care, with an emphasis on the close monitoring of renal function, fluid balance, and potential complications [2]. STEC-associated HUS has been extensively characterized in clinical and epidemiologic studies [3]. STEC is transmitted mainly via foodborne and person-to-person fecal-oral routes, facilitated by a low infectious dose and ruminant reservoirs, which underpins the disproportionate burden in young children [4]. Population-based studies indicate that HUS is rare but clinically significant, with incidence estimates influenced by surveillance intensity and regional exposure patterns [5]. Across Europe, pediatric STEC-HUS remains consistently low in incidence, as shown in registry and surveillance data from Poland (2012–mid-2023), England (2009–2023), and the Northern Italian HUS network [6-8].

2. Methods

We conducted a narrative review based on a structured search of PubMed/MEDLINE and the Cochrane Library (database inception to December 2025) using terms related to pediatric HUS (STEC-HUS and aHUS), neurological complications, MRI/DWI/ADC, EEG, complement activation, and eculizumab. We included peer-reviewed human studies in children reporting CNS manifestations, neurodiagnostics, management, or outcomes, and screened reference lists of key papers for additional relevant publications. Evidence was synthesized qualitatively with priority given to systematic reviews and contemporary cohorts/registries, while rare phenotypes were supported by selected case series.

3. Pathophysiology of neurologic involvement

Neurologic complications are among the most important extra-renal manifestations and most commonly include seizures and encephalopathy, with Posterior Reversible Encephalopathy Syndrome (PRES) or focal deficits reported less frequently [9,10]. Their presence typically marks more severe course of the disease like severe AKI, hypertension, metabolic derangements and may be followed by subtle long-term neurocognitive sequelae, supporting early neurologic assessment and structured follow-up [9,10,11].

STEC-HUS is initiated by Shiga toxin–mediated endothelial injury, which triggers thromboinflammation and microvascular thrombosis characteristic of TMA.[12] Stx2 variants are particularly potent and are consistently associated with more severe disease and a higher risk of progression to HUS.[13-16] Complement activation, especially of the alternative pathway, appears to act as an important amplifier of endothelial damage, in part through interactions with complement regulators such as factor H and reduced expression of protective molecules like CD59.[17-20] Systemic inflammation may further increase vascular susceptibility, including in the central nervous system (CNS), helping to explain neurologic involvement in severe pediatric cases.[10,21]

4. Demographics and clinical relevance

CNS involvement is one of the most clinically consequential extra-renal manifestations of pediatric HUS. Contemporary data indicate that acute neurological involvement in children with STEC-HUS is often in the low-teens, although rates vary by cohort and case-mix. In a large Irish pediatric series of laboratory-confirmed STEC-HUS, neurological involvement occurred in 11% of children, with seizures as the dominant presenting feature (73%), and most survivors achieved complete neurological recovery (91%) despite frequent use of intensive interventions.[10] In contrast, atypical HUS (aHUS), a complement-mediated thrombotic microangiopathy, is more consistently associated with extra-renal multi-organ involvement, with neurological manifestations representing the most common and clinically serious extra-renal phenotype in several pediatric cohorts. In a multicenter pediatric aHUS study, neurological involvement was reported in 27.2% of children and was associated with worse kidney function and higher mortality than in children without neurological complications.[22] More broadly, a 2024 systematic review synthesizing data across studies of aHUS extrarenal disease found that approximately 20–30% of patients experience extrarenal manifestations overall, with neurological features being most frequent.[23]

Historical studies underscore why neurological involvement has long been viewed as a marker of severity. In an older pediatric series, children with neurological manifestations had substantially higher rates of hypertension, chronic renal damage, and mortality than those without, and a significant proportion had residual neurological deficits, reflecting both disease severity and the limitations of supportive and targeted therapies at that time.[24] Contemporary reviews emphasize that, while many children recover well, early CNS involvement still identifies a subgroup at heightened risk for morbidity and longer-term neurocognitive sequelae.[11]

5. Clinical spectrum of neurological manifestations

The neurological phenotype of HUS in children spans diffuse encephalopathy, epileptic seizures, including status epilepticus, posterior reversible encephalopathy syndrome (PRES), focal neurological deficits, and movement disorders.[11] In STEC-HUS, seizures and encephalopathy often dominate the acute presentation. In the Irish STEC-HUS cohort, seizures were the most common neurological feature,[10] aligning with outbreak-era experience in pediatric HUS associated with the O104:H4 epidemic, in which neurological involvement was frequent and included stupor/coma, seizures, visual disturbances, paresis, and myoclonus.[25] Encephalopathy typically presents as fluctuating consciousness, confusion, agitation, or coma,

and often coexists with metabolic derangements, uremia, anemia, and hypertension, factors that may amplify cerebral vulnerability in a setting of microvascular endothelial injury.[11]

PRES warrants specific consideration due to its clinical significance and potential for reversibility. PRES typically manifests with seizures, altered mental status, headache, and visual disturbances, and is closely associated with severe hypertension and renal failure, two prevalent characteristics in complicated HUS. PRES in pediatric aHUS and severe renal illness settings, including presentations with papilledema, hypertension, seizures, and MRI abnormalities, is documented in case literature.[26] Pediatric critical care cohorts frequently exhibit PRES through altered mental status and seizures, which may resolve within days upon control of the trigger; however, morbidity and mortality can be significant in critically ill populations.[27] Recurrent or atypical PRES patterns, including brainstem/cerebellar involvement or recurrence, have also been described in children with complex renal and inflammatory comorbidity, highlighting that "reversible" does not equate to benign and that follow-up is warranted.[28]

Focal deficits and cerebrovascular complications are less common but clinically important. Pediatric case reports and small series describe ischemic infarctions presenting with hemiparesis, aphasia, or focal seizures in diarrhea-associated HUS, with some children ultimately recovering well despite severe initial neurologic impairment. [29,30] These presentations should prompt careful differential diagnosis, including venous thrombosis and other thrombotic microangiopathies, while recognizing that HUS-related microangiopathic brain injury can mimic stroke syndromes.

6. Neuroimaging: MRI patterns and prognostic implications

MRI is essential for CNS evaluation in juvenile HUS, serving both to clarify etiology, microangiopathic damage versus PRES versus infarction/hemorrhage, and to aid in prognostication. In children with STEC-HUS and neurological involvement, MRI abnormalities often involve the basal ganglia and/or white matter. In a pediatric STEC-HUS series treated with eculizumab, abnormal MRI findings were reported in most evaluated children, frequently affecting basal ganglia and white matter.[31] Diffusion-weighted imaging (DWI) has been repeatedly highlighted as particularly sensitive for early detection of microstructural injury in HUS, including basal ganglia lesions that may be subtle or underestimated on conventional sequences.[32] Importantly, some MRI patterns, especially those characterized by transient DWI/ADC abnormalities with limited FLAIR injury, may be reversible and associated with favorable short-term neurodevelopmental outcomes in selected pediatric cohorts, as described in children with diarrhea-associated HUS and neurological impairment.[33]

More recent imaging-focused work suggests that quantitative diffusion metrics may add prognostic value. A recent study evaluating apparent diffusion coefficient (ADC) maps in pediatric typical HUS reported that children with poorer neurological outcomes more often exhibited basal ganglia/thalamic diffusion abnormalities and broader microstructural changes, supporting the concept that early diffusion alterations may stratify risk beyond conventional qualitative MRI reading alone.[34] This aligns with a mechanistic view in which thrombotic microangiopathy and endothelial dysfunction in vulnerable deep gray matter territories, basal ganglia and thalami, drive a recognizable radiologic signature.[32,34] Although not exclusive to HUS, traditional radiology reviews of bilateral basal ganglia lesions identify HUS as one of the acute etiologies, underscoring the necessity for a systematic differential diagnosis when this pattern is observed[35]..

PRES on MRI is characterized by vasogenic edema, predominantly in the parieto-occipital regions, occasionally extending to adjacent areas. Identifying this pattern is clinically significant, as prompt blood pressure management and supportive neurocritical care may result in both radiologic and clinical improvement [11,27]. Intracranial hemorrhage might occur in thrombotic microangiopathy, especially in severe cases. Susceptibility-weighted imaging (SWI), when accessible, can assist in identifying microbleeds that may affect management strategies and prognosis. However, comprehensive outcome data specific to pediatric HUS regarding SWI are currently scarce.

7. EEG: detecting epileptic activity and encephalopathy

EEG is a key adjunctive tool in pediatric HUS with seizures or altered mental status. Older but still clinically informative pediatric EEG data suggest that generalized tonic-clonic seizures may be associated with diffuse slowing without focality and good recovery, whereas partial seizures were more often linked to structural lesions and residual deficits, with diverse EEG patterns including focal epileptiform activity and even burst-suppression-like features.[36] Outbreak-era pediatric data further indicated that EEG abnormalities can be common even beyond the subgroup with overt neurological symptoms, implying that subclinical encephalopathy or transient cortical dysfunction may be under-detected if EEG is not employed.[25] These findings support a practical approach in which children with HUS and impaired consciousness, refractory seizures, or clinical concern for non-convulsive status epilepticus receive early EEG, and when available, continuous EEG monitoring is considered in the most severe neurocritical presentations.

8. Management of CNS complications

The management of CNS complications in HUS is primarily neurocritical and systemic, involving rapid stabilization, seizure control, careful blood pressure management, correction of metabolic derangements, optimization of renal support including renal replacement therapy as necessary, and prevention of secondary brain injury. The function of complement inhibition varies significantly between aHUS and STEC-HUS. Eculizumab is a targeted therapy for aHUS, supported by strong biological rationale and significant clinical experience. Reports indicate a rapid resolution of severe neurological symptoms in children with complement-mediated disease unresponsive to plasma therapy.[37] Reviews of aHUS therapy highlight that neurological and cardiovascular complications are prevalent extrarenal manifestations, and that complement blockade targets the underlying complement-mediated thrombotic microangiopathy impacting both the brain and kidneys.[38]

In the context of STEC-HUS, the evidence supporting the use of eculizumab is inconsistent. Initial pediatric observations and uncontrolled studies indicated a possible advantage in severe STEC-HUS with CNS involvement, including prompt cessation of seizures in certain children and reversible MRI findings. [31, 33] Multiple cohort studies have suggested that early treatment, informed by neurological severity, may be associated with the regression of neurological lesions on MRI and clinical improvement. However, these studies are constrained by small sample sizes, non-randomized designs, and potential confounding factors.[39] Numerous critical analyses have emphasized the necessity for well-controlled trials to address this issue.[40] A 2025 systematic review and meta-analysis examining neurological outcomes in pediatric STEC-HUS found no statistically significant neurological advantage of eculizumab over standard care, highlighting the limitations of the existing retrospective literature.[41] Current evidence suggests a cautious, individualized approach: in aHUS, complement blockade is essential when CNS involvement occurs; in STEC-HUS, the use of eculizumab is contentious, and decisions should be informed by multidisciplinary expertise and the clinical context, recognizing the inherent uncertainty.[11, 41]

9. Long-term outcomes: subtle neurocognitive sequelae and rehabilitation needs

While major permanent neurological deficits appear uncommon in many contemporary pediatric series,[10] residual or late-emerging issues may be underestimated without structured follow-up. Long-term evaluation studies have shown that children who experienced major neurological symptoms during acute HUS can display subtle sequelae years later, including clumsiness, poor fine motor coordination, and attention-related difficulties, even when global cognitive measures fall within average ranges.[42] Outbreak-era pediatric follow-up similarly documented frequent complaints of reduced performance and persisting EEG abnormalities over months, with generally favorable neuropsychological outcomes but measurable between-group differences in some cognitive indices.[25] These observations, together with modern reviews,[11] support systematic longitudinal surveillance for neurodevelopmental, cognitive, behavioral, visual, and motor outcomes, particularly in children who experienced encephalopathy, status epilepticus, PRES, ischemic lesions, or prolonged intensive care.

Rehabilitation should be conceptualized not merely as recovery from observable neurological deficits, but also as the enhancement of higher-order functions. A multidisciplinary model that integrates physiotherapy, occupational therapy, speech and language therapy (as necessary), and neuropsychological assessment is suitable for children with a history of CNS involvement, particularly when school performance, attention, executive function, or fine motor skills are impacted. The objective is to identify subtle impairments at an early stage that could significantly affect educational outcomes and quality of life, even in children considered neurologically "recovered" through standard bedside assessments.

10. Conclusions

Neurological complications in pediatric HUS constitute a critical area that necessitates careful monitoring and systematic evaluation. In STEC-HUS, neurological involvement affects approximately ten percent of children in current cohorts, with seizures being the most common acute manifestation and generally favorable recovery observed when modern intensive care is accessible.[10] In aHUS, neurological involvement occurs more frequently, serves as an indicator of systemic severity, and is closely associated with complement-mediated microangiopathy, thereby highlighting the importance of timely complement blockade in management [22,37,38]. MRI, especially DWI/ADC-based assessment, delineates injury patterns and may offer prognostic insights, whereas EEG aids in the detection and management of epileptic activity and encephalopathy.[32-34, 36] The lack of gross neurological deficit at discharge should not eliminate the need for long-term follow-up, as subtle neurocognitive and neurobehavioral sequelae may persist or develop over time, potentially benefiting from early rehabilitation and educational support.[11,25,42]

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