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CASE SERIES

Neonatal Rotavirus-Associated Leukoencephalopathy: An 11-Case Series Identifying a Stereotyped MRI Diffusion Pattern Independent of Stool Viral Detection

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Abstract

Background: Fifth-day seizures in otherwise well term neonates have been associated with rotavirus-associated leukoencephalopathy, but diagnosis is often limited by stool-based testing, particularly when gastrointestinal symptoms are absent or minimal. Objective: To describe the clinical, laboratory, electroencephalographic, and MRI characteristics of neonates with presumed rotavirus-associated encephalopathy and to assess whether a stereotyped DWI/ADC pattern can support diagnosis when stool tests are negative. Methods: We retrospectively reviewed 11 term neonates who presented with seizures on days 4-6 of life, had no evidence of perinatal asphyxia, and underwent brain MRI with diffusion-weighted imaging and apparent diffusion coefficient mapping after sepsis and metabolic evaluation. Stool rotavirus testing was performed using antigen enzyme immunoassay and/or reverse-transcription PCR. Results: The cohort included 7 female and 4 male neonates. Seizures began on day 4 in 8 infants and day 5 in 3; multifocal clonic seizures predominated (7/11). Gastrointestinal symptoms were present in only 1 infant. Sepsis screens, electrolytes, calcium, glucose, and CSF results were non-contributory. Stool testing confirmed rotavirus in 6/11 infants (5 PCR-positive, 1 antigen-positive), while 5 were stool antigen-negative. All 11 infants demonstrated symmetric restricted diffusion involving the periventricular white matter and corpus callosum, with internal capsule involvement in 3/11. All were seizure-free at discharge; length of stay ranged from 10 to 15 days (median 13). Conclusion: In this homogeneous fifth-day seizure cohort, the MRI diffusion signature was more consistent than stool viral detection and should prompt consideration of rotavirus-associated neonatal leukoencephalopathy even when stool testing is negative.

Key words: rotavirus, neonatal seizures, fifthday fits, diffusionweighted imaging, leukoencephalopathy, corpus callosum, periventricular white matter

1 | INTRODUCTION

Neonatal seizures are a neurological emergency and require rapid evaluation for hypoxic-ischaemic encephalopathy, intracranial haemorrhage, stroke, metabolic derangement, and central nervous system infection. A clinically distinctive subgroup is the term or near-term neonate who is well after birth and then develops clustered seizures around the fourth to sixth day of life. This presentation has historically been described as fifth-day fits and has increasingly been

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linked to neonatal rotavirus-associated leukoencephalopathy (1, 2).

Rotavirus is traditionally regarded as an enteric pathogen; however, extraintestinal neurological manifestations are now well recognised. In neonates, gastrointestinal symptoms may be absent or mild, and the dominant presentation may be seizures with MRI evidence of white-matter injury (3). This creates a diagnostic challenge because a negative stool antigen test does not necessarily exclude a CNS-dominant rotavirus-associated process, particularly if viral shedding is transient or sampling occurs after the enteric phase has peaked.

MRI with diffusion-weighted imaging (DWI) and apparent diffusion coefficient (ADC) mapping has become central to recognising this entity. The pattern described in published series consists of bilateral, symmetric restricted diffusion in the periventricular white matter and corpus callosum, sometimes extending along white-matter tracts such as the internal capsule (4). This pattern differs from the cortical and deep grey-matter injury typical of hypoxic-ischaemic injury and from the posterior predominance often associated with neonatal hypoglycaemic injury.

We present an 11-case single-centre series of neonates with fifth-day seizures and a presumptive diagnosis of rotavirus-associated encephalopathy. The objectives were to characterise the clinical, laboratory, EEG, and neuroimaging features; compare stool-positive and stool-negative cases; and highlight the diagnostic value of a stereotyped MRI diffusion pattern when stool testing is negative.

2 | MATERIALS AND METHODS

2.1 | Study design and setting

This retrospective case series was conducted in a single tertiary-care neonatal intensive care unit. The report was prepared in accordance with the CARE framework for case series reporting. Institutional ethics approval was obtained (Ref:72/IHEC/2025; approval date: 01 December 2025) and written informed consent was obtained from parents or legal guardians for use of anonymised clinical, laboratory, EEG, and neuroimaging data.

2.2 | Eligibility criteria

Inclusion criteria were: term or near-term birth (>=37 weeks gestational age), seizure onset between days 4 and 6 of life, no clinical evidence of perinatal asphyxia, no major congenital anomaly, brain MRI with DWI/ADC sequences, and completion of sepsis and basic metabolic evaluation. Exclusion criteria were: preterm birth, confirmed hypoxic-ischaemic encephalopathy, intracranial haemorrhage, proven bacterial CNS infection, or another specific diagnosis explaining the seizures.

2.3 | Clinical, laboratory, and EEG assessment

Antenatal, obstetric, and perinatal variables included gestational age, birth weight, mode of delivery, antenatal risk factors, Apgar scores, and need for resuscitation. Seizure semiology was recorded as generalised tonic-clonic, focal clonic, or multifocal clonic, and clustering or apnoea was noted when present. Gastrointestinal symptoms were recorded separately. Sepsis evaluation included full blood count, C-reactive protein, and blood culture. Metabolic assessment included serum sodium, potassium, calcium, and random blood glucose. CSF studies included cell count, protein, glucose, culture, and BioFire FilmArray meningitis/encephalitis panel where performed. EEG was interpreted by a paediatric neurologist.

2.4 | Neuroimaging protocol

Brain MRI was performed within 24-48 hours of seizure onset whenever clinically feasible. Standard sequences included T1-weighted, T2-weighted, DWI (b-values 0 and 1000 s/mm2), and corresponding ADC maps. The classical MRI pattern was defined as bilateral symmetric diffusion restriction with high DWI signal and low ADC signal involving the periventricular white matter and corpus callosum. Internal capsule involvement was recorded separately for each case.

2.5 | Rotavirus testing

Stool samples were collected during admission. Rotavirus antigen enzyme immunoassay (EIA) was performed in seven cases and RT-PCR for rotavirus

A in six cases. Cases were classified as laboratory-confirmed when stool antigen or PCR was positive and as stool-test negative when all performed stool assays were negative. Because PCR was not performed in every stool-negative case, negative antigen results were interpreted cautiously.

2.6 | Management and outcome assessment

Initial stabilisation followed standard NICU practice. Phenobarbitone was used as first-line antiseizure therapy, with fosphenytoin and/or levetiracetam added for persistent or clustered seizures. Empiric ampicillin and amikacin were initiated pending culture results and discontinued after 72 hours when cultures remained sterile. Short-term outcomes included seizure freedom at discharge, adequacy of feeding, length of hospital stay, and in-hospital mortality.

3 | RESULTS

3.1 | Cohort characteristics

Eleven neonates fulfilled the eligibility criteria. Based on the case table, the cohort comprised 7 females and 4 males. Gestational age ranged from 37+0 to 38+5 weeks, and birth weight ranged from 2.41 to 3.36 kg. Nine infants were delivered by lower-segment caesarean section (5 elective and 4 emergency), and 2 were delivered vaginally. Apgar scores were 7 or higher at 1 minute and 8 or higher at 5 minutes in all infants, and none required resuscitation or met criteria for perinatal asphyxia (Table 1).

3.2 | Clinical presentation

Seizures began on day 4 of life in 8/11 infants (73%) and on day 5 in 3/11 (27%). Multifocal clonic seizures were the predominant semiology (7/11, 64%), followed by generalised tonic-clonic seizures (2/11, 18%), left focal clonic seizures (1/11, 9%), and right focal clonic seizures (1/11, 9%). Seizures were commonly clustered and were associated with brief apnoeic episodes in the acute phase. Gastrointestinal symptoms were uncommon; only 1 infant had mild loose stools (1/11, 9%) (Figure 3).

3.3 | Laboratory and EEG findings

Sepsis and metabolic evaluations did not identify an alternative cause for seizures. Blood counts were within acceptable neonatal ranges, CRP values were low, and all blood cultures were sterile. Serum sodium, potassium, calcium, and glucose were not explanatory. CSF was acellular with non-contributory chemistry in cases where lumbar puncture was performed; BioFire testing was negative in the two cases tested. EEG showed epileptiform abnormalities in 10/11 infants, most often multifocal or frontocentral discharges; one infant had a normal sleep-wake EEG (Table 2A).

3.4 | Rotavirus status

Rotavirus was confirmed in 6/11 infants (55%): 5 by stool RT-PCR and 1 by stool antigen EIA. The remaining 5 infants (45%) were stool antigen-negative. Stool-positive and stool-negative infants showed no clinically meaningful difference in seizure timing, EEG pattern, MRI distribution, response to antiseizure therapy, or short-term discharge status.

3.5 | MRI findings

All 11 infants (100%) showed the same stereotyped MRI diffusion pattern: bilateral symmetric restriction involving the periventricular white matter and corpus callosum, confirmed on DWI/ADC review. Representative original diffusion images are presented with anatomical annotations in Figure 1. Internal capsule involvement was present in 3/11 infants (27%; Cases 6, 8, and 9). The pattern was present in both stool-positive and stool-negative cases, supporting the diagnostic value of MRI when stool testing is negative. Cranial ultrasound was either normal or showed only mild non-specific periventricular flare, and no infant had haemorrhage.

3.6 | Treatment and short-term outcome

All infants received phenobarbitone as first-line therapy. Fosphenytoin was added in 7 infants, and levetiracetam in 4. Empiric antibiotics were stopped after 72 hours once blood and CSF cultures were sterile. All infants were seizure-free and feeding adequately

at discharge. Length of stay ranged from 10 to 15 days, with a median of 13 days. There were no in-hospital deaths (Table 2B; Figure 4).

Figure 1. Original axial diffusion MRi images in Neonatal rotavirus-associated

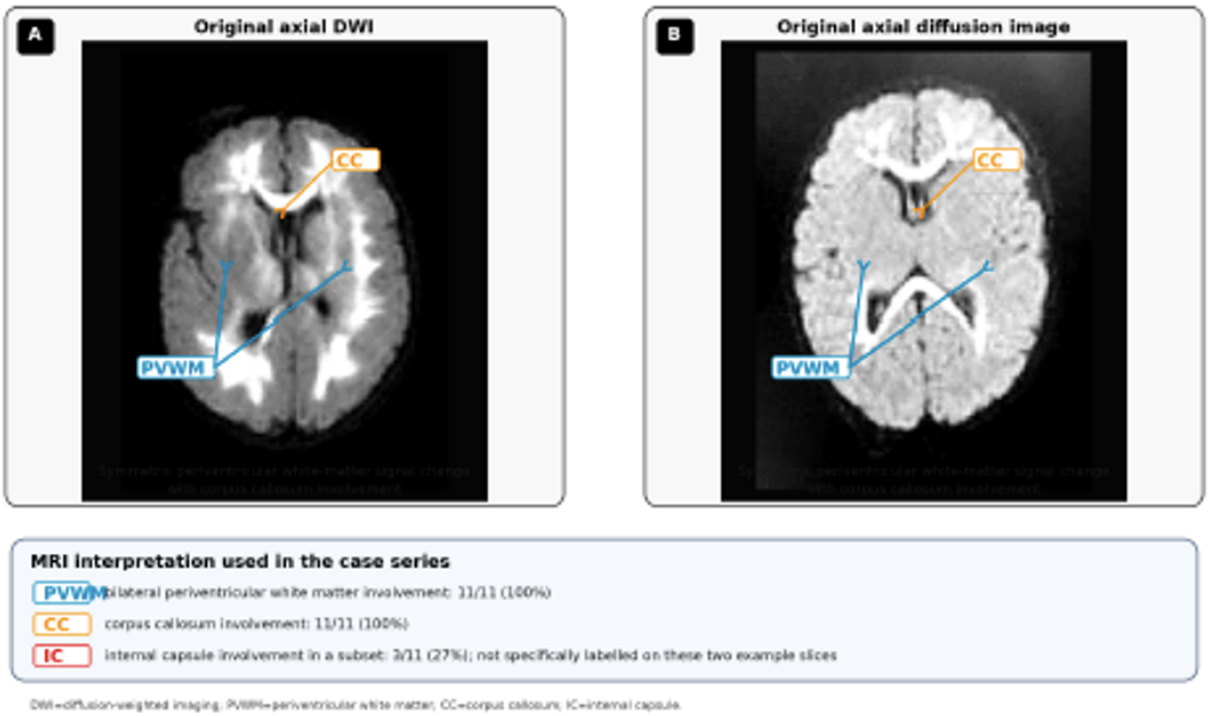

Fig. 1: Original axial MRI diffusion images demonstrating the cohort-defining white-matter pattern. Panel A shows an anonymised original axial diffusion-weighted MRI image with bilateral symmetric signal change in the periventricular white matter and midline callosal region. Panel B shows a second anonymised original axial diffusion image at a comparable level, again demonstrating the same symmetric periventricular-callosal distribution. The coloured annotations identify the key anatomic regions used for interpretation: PVWM (blue) and CC (orange). Across the cohort, PVWM and corpus callosum involvement was present in all 11 infants (100%), while internal capsule involvement was present in 3 of 11 infants (27%). ADC maps were reviewed in the clinical MRI assessment to confirm true restricted diffusion, although the representative images shown here are diffusion panels. DWI=diffusion-weighted imaging; ADC=apparent diffusion coefficient; PVWM=periventricular white matter; CC=corpus callosum; IC=internal capsule.

Table 1. Baseline characteristics and clinical presentation of 11 neonates with rotavirus-associated encephalopathy

Case	Sex	GA (wk+d)	BW (kg)	Delivery / indication	Antenatal risk factors	Apgar 1/5 min	Seizure day	Seizure type	GI symptoms
1	F	38+2	2.920	Em LSCS (fetal distress)	None	8/9	4	GTCS	No
2	F	38+0	2.410	Em LSCS (previous LSCS in labour)	SGA	8/9	4	Multifocal clonic	No
3	M	37+0	2.700	El LSCS (previous LSCS)	None	8/9	5	Multifocal clonic	No
4	M	37+4	2.620	El LSCS (previous LSCS)	GDM (OHA)	8/9	5	Left focal clonic	No
5	M	37+1	3.360	El LSCS (previous LSCS)	None	8/9	4	Multifocal clonic	No
6	F	37+5	2.810	Em LSCS (previous LSCS in labour)	Plummer-Vinson syndrome	7/8	4	GTCS	Yes (loose stools)
7	F	37+4	2.650	El LSCS (previous LSCS)	None	8/9	4	Right focal clonic	No
8	M	37+5	2.720	Em LSCS (pericardial effusion)	Pericardial effusion	8/9	4	Multifocal clonic	No
9	F	38+5	2.880	Vaginal (spontaneous)	GDM (OHA)	7/8	4	Multifocal clonic	No
10	F	38+1	2.990	Vaginal (spontaneous)	None	8/9	5	Multifocal clonic	No
11	F	37+1	2.700	El LSCS (previous LSCS)	Overt DM	8/9	4	Multifocal clonic	No

AN=antenatal; BW=birth weight; El=elective; Em=emergency; GA=gestational age; GDM=gestational diabetes mellitus; GI=gastrointestinal; GTCS=generalised tonic-clonic seizure; LSCS=lower-segment caesarean section; OHA=oral hypoglycaemic agent; SGA=small for gestational age.

Figure 2. CARE-compliant clinical timeline by day of life - rotavirus-associated neonatal encephalopathy

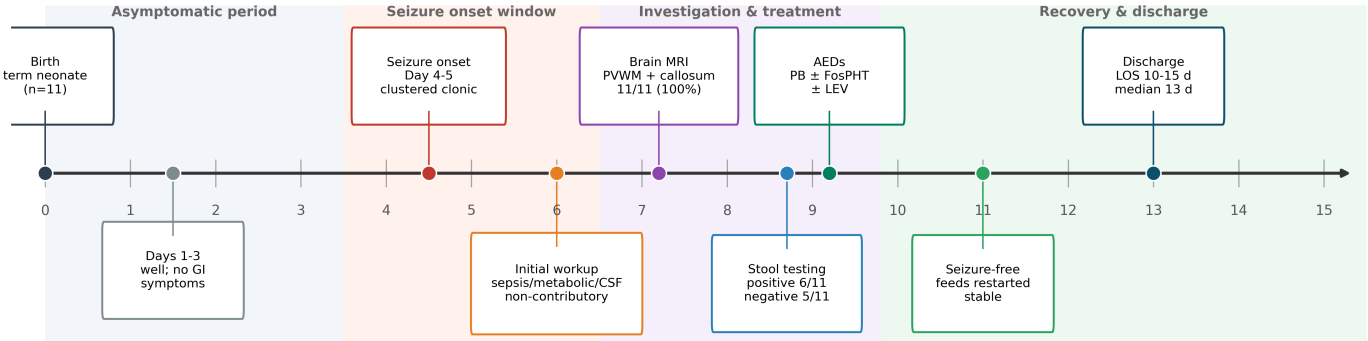

Fig. 2: CARE-compliant clinical timeline by day of life. The cohort was well during days 1-3, developed clustered seizures on days 4-5, underwent sepsis/metabolic/CSF/EEG assessment and MRI during the acute period, and was discharged after clinical stabilisation. Median length of stay was 13 days.

Table 2. Laboratory, EEG, stool, and MRI findings

Case	Hb / TC / Plt	CRP (mg/L)	Na / K / Ca	CSF	EEG	Stool test	Stool result	MRI DWI/ADC pattern	IC on MRI
1	17.4 / 6200 / 1.6 L	1.2	135 / 5.2 / 8.4	Acellular; 0 cells; protein 44; glucose 51	Bilateral FC epileptiform	Antigen	Positive	PVWM + CC (splenium)	No
2	18.3 / 8200 / 2.6 L	2.7	143 / 5.1 / 8.9	Consent refused	Focal left FC-temporal epileptiform	PCR	Positive	PVWM + CC	No
3	17.4 / 10080 / 2.4 L	4.4	138 / 4.8 / 9.5	Not done (PCR positive)	Multifocal bilateral epileptiform	PCR	Positive	PVWM + CC	No
4	17.0 / 10030 / 2.5 L	3.4	139 / 4.9 / 9.2	Acellular; 0 cells; protein 38; glucose 58	Normal sleep-wake EEG	PCR	Positive	PVWM + CC	No
5	17.4 / 8800 / 2.8 L	3.2	140 / 4.6 / 9.4	Acellular; 0 cells; protein 45; glucose 51	Diffuse epileptiform	PCR	Positive	PVWM + CC	No
6	16.4 / 9800 / 1.8L	5.1	137/4.0/8.8	Acellular; normal chemistry	Bilateral FC epileptiform	Antigen	Negative	PVWM + CC (splenium + genu)	Yes
7	17 / 11000 / 2.0 L	4.0	139/4.2/8.9	Normal (BioFire negative)	Focal left frontocentral epileptiform	Antigen	Negative	PVWM + CC	No
8	16.5/ 8500 / 2.5 L	6.0	141/3.9/9.0	Normal (BioFire negative)	Multifocal bilateral epileptiform	Antigen	Negative	PVWM + CC	Yes
9	15.5 / 10830 / 1.9l	4.5	143/4.8/9.3	Acellular; protein 82; glucose 41	Multiple epileptiform foci	Antigen	Negative	PVWM + CC (splenium + genu)	Yes
10	22.1 / 10810/ 2.8 L	1.73	139/5.4/9.2	Acellular; normal chemistry	Diffuse epileptiform	PCR	Positive	PVWM + CC	No
11	20.1 / 5020 / 2.1l	3.9	138.7/5.4/9.1	Acellular; normal chemistry	Bilateral FC (R>L) epileptiform	Antigen	Negative	PVWM + CC	No

ADC=apparent diffusion coefficient; BioFire=BioFire FilmArray meningitis/encephalitis panel; CC=corpus callosum; CRP=C-reactive protein; CSF=cerebrospinal fluid; DWI=diffusion-weighted imaging; EEG=electroencephalography; FC=frontocentral; IC=internal capsule; PVWM=periventricular white matter; WNL=within normal limits.

Table 3. Treatment and discharge outcomes

Case	AED regimen	Antibiotic duration	Discharge outcome	Length of stay (days)
1	PB + FosPHT	72 h	Seizure-free; feeding well	15
2	PB + FosPHT	72 h	Seizure-free; feeding well	13
3	PB + FosPHT	72 h	Seizure-free; feeding well	14
4	PB + FosPHT	72 h	Seizure-free; feeding well	11
5	PB + FosPHT	72 h	Seizure-free; feeding well	10
6	PB + FosPHT + LEV	72 h	Seizure-free; feeding well	15
7	PB + FosPHT	72 h	Seizure-free; feeding well	13
8	PB + FosPHT + LEV	72 h	Seizure-free; feeding well	12
9	PB + FosPHT + LEV	72 h	Seizure-free; feeding well	11
10	PB + LEV	72 h	Seizure-free; feeding well	12
11	PB + LEV	72 h	Seizure-free; feeding well	13

AED=antiepileptic/antiseizure drug; FosPHT=fosphenytoin; LEV=levetiracetam; PB=phenobarbitone.

Figure 3. Clinical and imaging profile of 11 neonates with rotavirus-associated encephalopathy

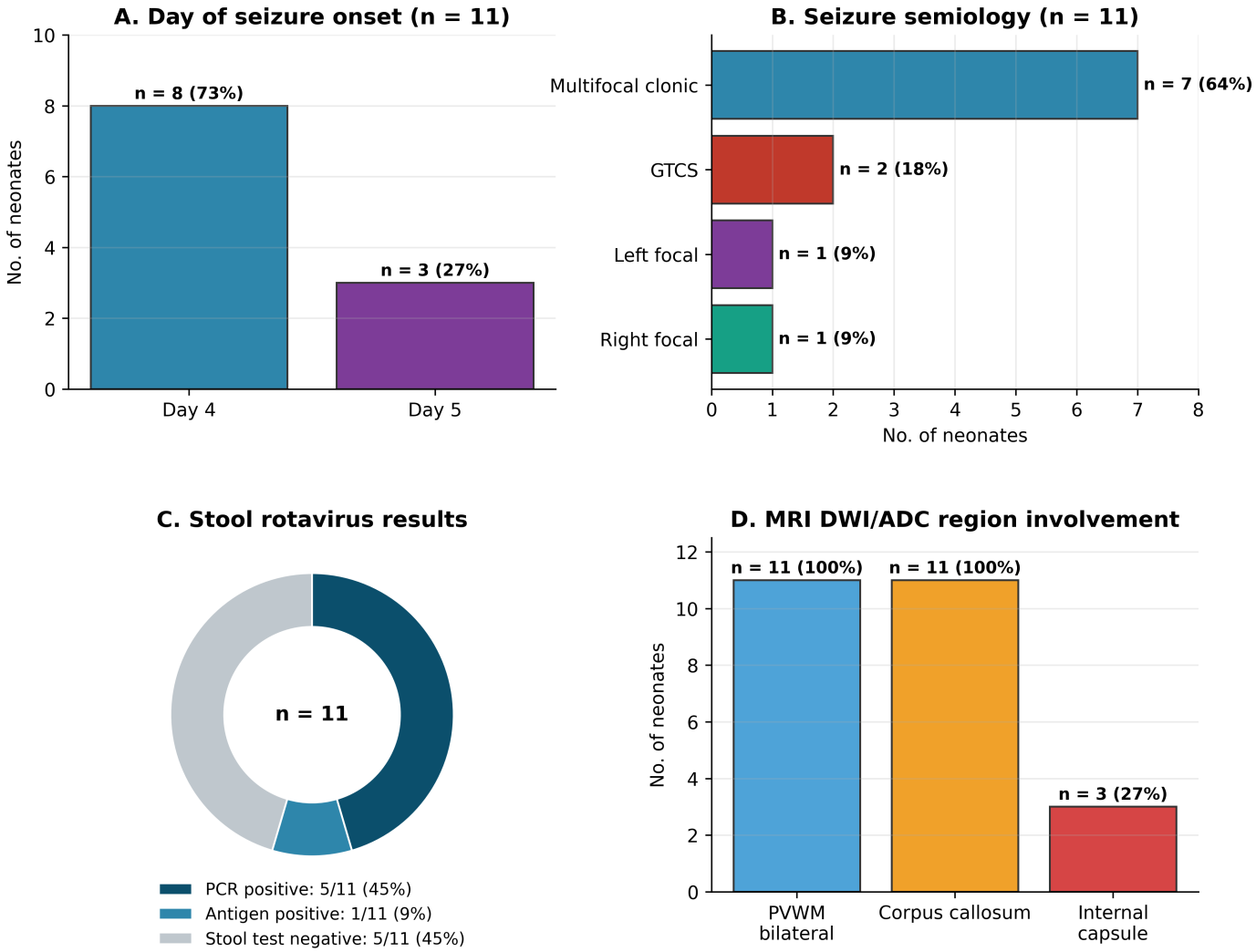

Fig. 3: Cohort clinical and imaging profile. (A) Day of seizure onset: day 4 in 8/11 (73%) and day 5 in 3/11 (27%). (B) Seizure semiology: multifocal clonic 7/11 (64%), GTCS 2/11 (18%), left focal 1/11 (9%), right focal 1/11 (9%). (C) Stool results: PCR-positive 5/11 (45%), antigen-positive 1/11 (9%), stool-test negative 5/11 (45%). (D) MRI involvement: PVWM and corpus callosum 11/11 (100% each), internal capsule 3/11 (27%).

4 | DISCUSSION

4.1 | Principal findings

This 11-case series describes a highly homogeneous phenotype of neonatal seizures occurring around the fifth day of life in otherwise stable term or near-term neonates. The central finding is the universal presence of a stereotyped MRI diffusion pattern, with bilateral periventricular white-matter and corpus callosum restriction in every case; representative original diffusion images are shown in Figure 1. Importantly, this pattern was present regardless of stool rotavirus result, including in the five infants with negative stool antigen testing.

4.2 | Relation to prior literature

The clinical profile aligns with published descriptions of neonatal rotavirus-associated leukoencephalopathy: clustered focal or multifocal clonic seizures around day 5 of life, minimal gastrointestinal symptoms, normal or non-specific CSF findings, and a distinctive symmetric DWI pattern involving cerebral white matter and the corpus callosum. The present series reinforces that this imaging signature

Figure 4. Per-case clinical profile - rotavirus-associated neonatal encephalopathy (n = 11)

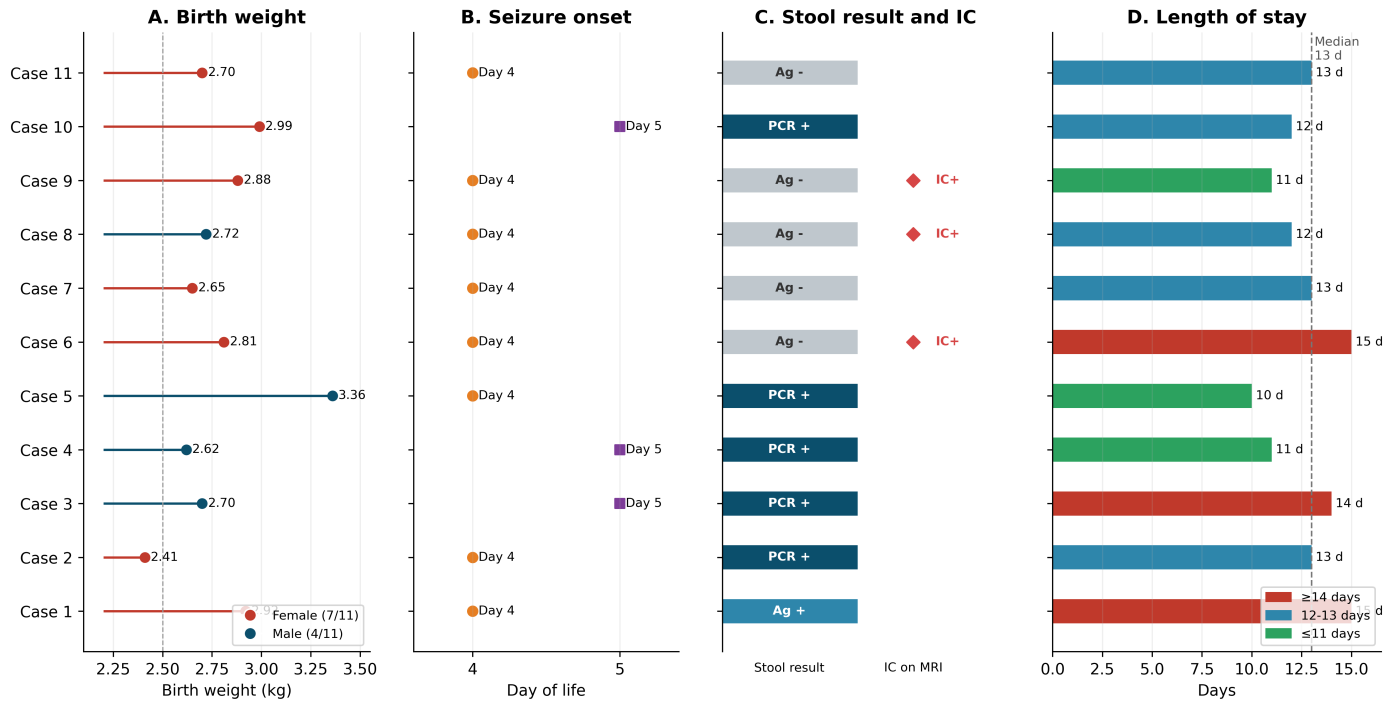

Fig. 4: Per-case clinical profile. Birth weight, day of seizure onset, stool result with internal capsule involvement, and length of stay are displayed for each case. The sex distribution based on the case table was female 7/11 and male 4/11; median hospital stay was 13 days.

can be more consistent than stool detection in clinically selected CNS-dominant cases.

4.3 | Diagnostic implications

The practical implication is that early MRI with DWI/ADC should be prioritised in term neonates with fifth-day seizures when perinatal asphyxia, sepsis, hypoglycaemia, electrolyte disturbance, and haemorrhage are not supported by the clinical or laboratory data. A negative stool antigen test should not lead clinicians to dismiss the diagnosis when the MRI pattern is classical. Cranial ultrasound was insufficiently sensitive for this pattern and should not be relied upon as the definitive neuroimaging test (5).

4.4 | Pathophysiological considerations

The mechanism of rotavirus-associated white-matter injury is incompletely understood. Proposed pathways include systemic inflammatory mediator effects, blood-brain barrier vulnerability in the neonatal period, and viral protein-mediated effects on immature white matter. The relative sparing of cortex and deep grey matter in this series supports a selective white-matter tract vulnerability rather than a non-specific global hypoxic-ischaemic pattern (6, 7).

4.5 | Strengths and limitations

Strengths include the consistent clinical phenotype, systematic MRI review with DWI/ADC correlation, and complete short-term outcome data. Limitations include the retrospective single-centre design, small sample size, lack of CSF rotavirus testing, and absence of long-term neurodevelopmental follow-up. In addition, not all stool-negative cases underwent stool PCR; therefore, antigen-negative cases may include false-negative results rather than true absence of rotavirus infection (8).

4.6 | Future directions

Prospective multicentre studies should standardise stool PCR, CSF viral testing, cytokine profiling,

MRI scoring, and neurodevelopmental follow-up. Serial MRI would clarify whether lesion extent, callosal involvement, or internal capsule extension predicts later motor, cognitive, or epilepsy outcomes. A combined clinical-imaging diagnostic framework may improve recognition and infection-control practice in NICU settings.

5 | CONCLUSION

In neonates presenting with fifth-day seizures, a stereotyped pattern of bilateral periventricular white-matter and corpus callosum diffusion restriction should strongly suggest rotavirus-associated neonatal leukoencephalopathy, even when stool testing is negative. Early MRI with DWI/ADC is therefore central to diagnosis, while stool testing should be interpreted as supportive rather than exclusionary. Long-term neurodevelopmental surveillance is recommended, especially when internal capsule involvement is present.

6 | PATIENT AND PARENT PERSPECTIVE

The sudden onset of seizures in previously well newborns was highly distressing for families. Parents required repeated explanations that negative sepsis screens and normal metabolic results did not exclude a neurological diagnosis, and that MRI provided the key diagnostic evidence. Families also expressed concern about the significance of negative stool tests in some infants. At discharge, parents were counselled regarding seizure recurrence, feeding, medication adherence, and the need for developmental follow-up. This paragraph represents a composite anonymised perspective and does not identify any individual family.

Ethics Statement and Informed Consent:

This study was approved by the Institutional Human Ethics Committee, KMCH Institute of Health Sciences and Research, Coimbatore, India (Study No. 72/IHEC/2025; approval date: 01 December 2025). Written informed consent was obtained from the parents or legal guardians of all included neonates for the use of anonymised clinical, laboratory, EEG, and neuroimaging data for research and publication. All data were handled confidentially, and no personally identifiable information is disclosed. The study was conducted in accordance with the ethical principles of the Declaration of Helsinki and reported in line with CARE reporting standards for case series.

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