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Original Research Article

DIAGNOSTIC YIELD OF INVESTIGATIONS IN INFANTS BEYOND THE NEONATAL PERIOD PRESENTING WITH A FIRST SEIZURE: A PROSPECTIVE OBSERVATIONAL STUDY

Amritha S[1], Harsha T Valoor[2], Shameem A M[3]

[1]*Postgraduate Resident, Department of Paediatrics, Government Medical College, Kozhikode, Kerala, India.*
[2]*Assistant Professor, Department of Neurology, Government Medical College, Manjeri, Kerala, India.*
[3]*Associate Professor, Department of Pediatrics, Government Medical College, Kozhikode, Kozhikode, Kerala, India.*

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Corresponding Author:
Dr. Harsha T Valoor,
Assistant Professor, Department of Neurology, Government Medical College, Manjeri, Kerala, India.
Email: harshavaloor@gmail.com

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ABSTRACT

Background: Infants presenting with a first seizure may undergo electroencephalography, neuroimaging, lumbar puncture and metabolic screening in varying combinations. In resource-constrained settings these investigations compete for limited capacity, and lumbar puncture, sedation for magnetic resonance imaging and the ionising radiation of computed tomography each carry their own costs. The proportion of such investigations that actually returns an abnormal result in Indian infants has not been well described. **Objectives:** To describe the etiological spectrum of first seizure in infants aged 1–12 months and to quantify the diagnostic yield of each investigation as performed under routine clinical indication.

Materials and Methods: Prospective observational study of 90 consecutive infants aged 1–12 months admitted with a first seizure to a tertiary teaching hospital in Kozhikode, Kerala, over 18 months. Neonates and infants with head injury were excluded. Baseline biochemistry was performed in all infants; electroencephalography, magnetic resonance imaging, computed tomography, cerebrospinal fluid analysis and extended metabolic evaluation were performed on clinical indication. Diagnostic yield was calculated as the proportion of abnormal results among infants tested, and the number needed to test to detect one abnormality was derived. Infants were followed for six months.

Results: Febrile seizure accounted for 58 infants (64.4%); the remaining 32 (35.6%) had a serious underlying etiology — epilepsy or epileptic encephalopathy in 16 (17.8%), central nervous system infection in 7 (7.8%) — pyogenic meningitis in 5 and viral meningoencephalitis in 2 — metabolic causes in 6 (6.7%), structural lesions in 2 (2.2%) and cerebral infarct in 1 (1.1%). Diagnostic yield was highest for magnetic resonance imaging (17 of 25; 68.0%; number needed to test 1.5), followed by computed tomography (7 of 14; 50.0%), electroencephalography (19 of 60; 31.7%), cerebrospinal fluid analysis (9 of 31; 29.0%) and extended metabolic evaluation (5 of 22; 22.7%; number needed to test 4.4). Universal baseline biochemistry identified hypocalcaemia as the seizure cause in 4 infants (4.4%). Fever was present in 67 infants (74.4%), of whom 9 (13.4%) had a diagnosis other than febrile seizure. Both deaths (2.2%) and the only new persistent neurological deficit occurred in infants with structural or neurovascular etiology.

Conclusion: Selective, clinically indicated investigation produced a high yield from magnetic resonance imaging and a comparatively low yield from extended metabolic screening. Approximately one in seven febrile infants had a cause other than febrile seizure, indicating that fever should not by itself be

treated as a reassuring finding. These yield figures offer a rational basis for prioritising investigation in similar settings.
Keywords: Infant; Seizures; Febrile Seizures; Diagnostic Tests, Routine; Neuroimaging; Spinal Puncture; India.

INTRODUCTION

Seizures are the commonest neurological emergency of the first year of life, and their incidence in infancy exceeds that of any subsequent period of childhood.[1,2] The immature brain is intrinsically predisposed to hypersynchronous discharge: glutamatergic transmission predominates over inhibition, γ-aminobutyric acid may act as a depolarising rather than a hyperpolarising neurotransmitter owing to the developmental profile of the NKCC1 and KCC2 chloride transporters, and myelination and cortical network organisation remain incomplete.[3,4] Superimposed on this substrate is a high burden of acute precipitants — febrile illness, electrolyte disturbance, hypoglycaemia and central nervous system (CNS) infection — which together account for most first seizures in this age group.[5]

Because the etiological spectrum is broad, the infant who has just convulsed may plausibly require electroencephalography, magnetic resonance imaging, lumbar puncture and metabolic screening. Each carries a cost. Lumbar puncture is invasive and often deferred in the unstable infant; magnetic resonance imaging in infancy usually requires sedation and is frequently rationed by scanner availability; computed tomography delivers ionising radiation to a developing brain, with a demonstrated association between paediatric exposure and subsequent leukaemia and brain tumours,[6,7] and extended metabolic evaluation is expensive, slow to return and frequently equivocal. In a public tertiary hospital these constraints are not theoretical — investigation capacity is finite and must be allocated.

Guidance on evaluating a first seizure in children is derived largely from populations older than one year and from settings in which neuroimaging is readily available.[6,11] Indian hospital-based studies have described the etiological spectrum of first seizure in infancy, and have shown that it varies substantially between regions — metabolic causes predominating in a New Delhi cohort and febrile seizures in a Bengaluru cohort.[9,10] What these studies do not report is how often each investigation, once ordered, actually returned an abnormal result. Yield is the quantity that determines whether an investigation strategy is worth its cost, and it is largely absent from the Indian literature on infantile seizures.

The present study was therefore designed to report, alongside the etiological spectrum, the diagnostic yield of each investigation as it was actually used in routine practice at a tertiary referral centre in northern Kerala. The aim was to provide empirical figures against which local investigation practice can be calibrated.

MATERIALS AND METHODS

Study design and setting

This was a prospective observational cohort study conducted in the Department of Paediatrics, Government Medical College, Kozhikode — a tertiary care teaching hospital and regional referral centre for northern Kerala, India. The study ran over 18 months from June 2024 to December 2025, comprising a 12-month enrolment period followed by six months of scheduled follow-up. Reporting follows the STROBE statement for observational studies.

Participants

Consecutive infants aged 1 to 12 months completed, admitted with a first episode of seizure, were eligible for inclusion. Neonates (aged 28 days or less) were excluded because their etiological spectrum — dominated by perinatal asphyxia, intracranial haemorrhage and transitional metabolic disturbance — differs fundamentally from that of later infancy, and because their investigation pathway is correspondingly different. Infants whose seizure followed head injury were also excluded, as were infants in whom any prior seizure of any type was reported. Written informed consent was obtained from a parent or legal guardian before enrolment. The sample size of 90 was calculated using the formula $4pq/d^2$, taking $p = 68\%$ from a previous Indian study of first seizure in infants and a relative precision of 10%.

Data collection

A pre-designed, pre-tested structured proforma was completed by the treating team at admission. Recorded variables comprised demography; seizure characterisation (semiology classified per the International League Against Epilepsy 2017 operational framework as generalized, focal, focal with secondary generalisation or unknown onset; duration; number of episodes at admission and during the hospital stay); associated symptoms; drug and indigenous-medication history; antenatal, natal and postnatal history including gestation, birth weight, size for gestational age, perinatal asphyxia and neonatal intensive care admission; developmental history; family history of febrile seizure and of epilepsy; immunisation status; and full physical and neurological examination including anthropometry, head circumference, tone, level of consciousness and examination for neurocutaneous stigmata.

Investigation protocol

Baseline investigations were performed in every infant and comprised capillary and venous glucose, serum sodium, potassium, total and ionised calcium, magnesium and phosphate, complete blood count, renal and liver function tests and C-reactive protein.

The following investigations were performed on clinical indication as judged by the treating consultant, rather than by protocol: cerebrospinal fluid (CSF) analysis where CNS infection was suspected; electroencephalography where an epileptic disorder was suspected or seizure classification was uncertain; magnetic resonance imaging (MRI) and computed tomography (CT) of the brain where structural pathology was suspected; and extended metabolic evaluation (plasma ammonia, lactate, pyruvate, arterial blood gas, tandem mass spectrometry and gas chromatography–mass spectrometry) where an inborn error of metabolism was suspected. This reflects real-world practice at the study centre and is the pattern to which the yield figures reported below apply.

Definitions and outcome measures

The primary outcome was the diagnostic yield of each investigation, defined as the number of infants with an abnormal result divided by the number of infants in whom that investigation was performed, expressed as a percentage. The number needed to test — the reciprocal of the yield — was derived to express the same quantity in a form directly usable at the bedside.

Febrile seizure was defined as a seizure accompanying a temperature of 38 °C or higher in an infant without CNS infection, metabolic derangement or prior afebrile seizure. A serious underlying etiology was defined as any final diagnosis other than febrile seizure, comprising CNS infection, structural brain abnormality, neurovascular event, metabolic disturbance or inborn error of metabolism, and epilepsy including epileptic encephalopathy. Final etiological assignment was made at discharge by the treating consultant on the basis of all available clinical, laboratory, electrophysiological and imaging data.

Secondary outcomes were death or persistent neurological deficit at six months, and seizure recurrence within six months. All infants were reviewed at three and six months after discharge.

Statistical analysis

Data were entered into Microsoft Excel and analysed using SPSS. Categorical variables are expressed as frequencies and percentages, and continuous variables as mean and standard deviation. Diagnostic yields are reported as proportions with 95% confidence intervals calculated by the Wilson score method. Associations between categorical variables were examined using the chi-square test, or the Fisher exact test where any expected cell count was below five. A two-sided p value below 0.05 was considered statistically significant.

Ethical considerations

The study was approved by the Institutional Ethics Committee of Government Medical College, Kozhikode, before data collection commenced (reference number GMCKKD/RP 2024/IEC/173), and was conducted in accordance with the Declaration of Helsinki. Written informed consent was obtained from a parent or legal guardian of every participant, and confidentiality was maintained throughout.

RESULTS

Cohort characteristics

Ninety infants were enrolled. Seventy-seven (85.6%) were aged six months or older and 13 (14.4%) were younger than six months; 57 (63.3%) were male. Seventy-two (80.0%) were born at term, 18 (20.0%) had a birth weight below 2.5 kg and nine (10.0%) were small for gestational age. Four infants (4.4%) had a history of severe perinatal asphyxia. Development was appropriate for age in 78 (86.7%), while seven (7.8%) had global developmental delay, three (3.3%) had regression of milestones and two (2.2%) had isolated gross motor delay. Seventy-four (82.2%) were immunised appropriately for age. A family history of febrile seizure was present in 23 (25.6%) and of epilepsy in five (5.6%). Baseline characteristics are shown in Table 1.

Table 1: Baseline characteristics of the cohort (n = 90)

Characteristic	n	%
Age		
<6 months	13	14.4
≥6 months	77	85.6
Sex		
Male	57	63.3
Female	33	36.7
Perinatal history		
Preterm birth	18	20.0
Birth weight <2.5 kg	18	20.0
Small for gestational age	9	10.0
Severe perinatal asphyxia	4	4.4
Development		
Appropriate for age	78	86.7
Global developmental delay	7	7.8
Regression of milestones	3	3.3

Gross motor delay	2	2.2
Family history		
Febrile seizure	23	25.6
Epilepsy	5	5.6
Immunisation		
Complete for age	74	82.2
Partial	16	17.8

Seizure characteristics and clinical status at admission

Generalized seizures accounted for 85 events (94.4%), of which 74 (82.2% of the cohort) were generalized tonic–clonic, nine (10.0%) were infantile spasms and two (2.2%) were generalized myoclonic. Focal seizures occurred in five infants (5.6%). Sixty-one seizures (67.8%) lasted less than five minutes, 23 (25.6%) lasted 5–15 minutes and six (6.7%) exceeded 15 minutes. Forty-seven infants (52.2%) had a single episode, 21 (23.3%) had two and 22 (24.4%) had more than two.

Fever was the commonest associated symptom, present in 67 infants (74.4%), followed by acute respiratory infection in 51 (56.7%), poor feeding and poor activity in eight each (8.9%) and acute diarrhoeal disease in five (5.6%). At admission, 53 infants (58.9%) were neurologically normal, 19 (21.1%) were drowsy and 18 (20.0%) were actively convulsing. On neurological examination 70 (77.8%) were normal, eight (8.9%) had central hypotonia, six (6.7%) hypertonia and six (6.7%) were encephalopathic. Anthropometry was normal in 75 (83.3%), with failure to thrive in 11 (12.2%) and microcephaly in four (4.4%).

Etiological spectrum

Febrile seizure was the single commonest diagnosis, accounting for 58 infants (64.4%). A serious underlying etiology was identified in the remaining 32 (35.6%). Within this group, epilepsy or epileptic encephalopathy was the largest category at 16 infants (17.8% of the cohort), comprising 11 with epilepsy — including two with infantile epileptic spasms syndrome — and five with other unprovoked seizure diagnoses. CNS infection accounted for seven infants (7.8%): five with pyogenic meningitis and two with a cerebrospinal fluid and clinical picture consistent with viral meningoencephalitis. Metabolic causes accounted for six (6.7%): four hypocalcaemic seizures and two inborn errors of metabolism. Structural lesions accounted for two (2.2%) and a subacute cerebral infarct for one (1.1%). The distribution is shown in Table 2.

Table 2: Final etiological diagnosis (n = 90)

Diagnosis	n	%
Febrile seizure	**58**	**64.4**
Serious underlying etiology	**32**	**35.6**
Epilepsy (including 2 with IESS)	11	12.2
Other unprovoked seizure	5	5.6
Pyogenic meningitis	6	6.7
Viral meningoencephalitis	1	1.1
Hypocalcaemic seizure	4	4.4
Inborn error of metabolism	2	2.2
Structural brain abnormality	2	2.2
Subacute cerebral infarct	1	1.1
Total	**90**	**100.0**

IESS = infantile epileptic spasms syndrome.

Diagnostic yield of selectively performed investigations

Investigations were used selectively, with the proportion of the cohort tested ranging from 66.7% for electroencephalography to 15.6% for computed tomography. Yield varied inversely with frequency of use. Magnetic resonance imaging was performed in 25 infants (27.8%) and was abnormal in 17, a yield of 68.0% — approximately three abnormal scans for every two performed, corresponding to a number needed to test of 1.5. Computed tomography was performed in 14 (15.6%) and was abnormal in seven (50.0%). Electroencephalography, the most frequently used investigation, was performed in 60 (66.7%) and was abnormal in 19 (31.7%). CSF analysis was performed in 31 (34.4%) and was abnormal in nine (29.0%). Extended metabolic evaluation had the lowest yield: performed in 22 infants (24.4%), it was abnormal in five (22.7%), a number needed to test of 4.4. Yields are summarised in Table 3.

Table 3: Diagnostic yield of selectively performed investigations (n = 90)

Investigation	Performed, n (%)	Abnormal, n	Yield, % (95% CI)	Number needed to test
Magnetic resonance imaging	25 (27.8)	17	68.0 (48.4–82.8)	1.5
Computed tomography	14 (15.6)	7	50.0 (26.8–73.2)	2.0
Electroencephalography	60 (66.7)	19	31.7 (21.3–44.2)	3.2
Cerebrospinal fluid analysis	31 (34.4)	9	29.0 (16.1–46.6)	3.4
Extended metabolic evaluation	22 (24.4)	5	22.7 (10.1–43.4)	4.4

Yield is the proportion abnormal among infants tested. Number needed to test is the reciprocal of the yield. Confidence intervals calculated by the Wilson score method.

Table 4: Fever status and etiological group (n = 90)

Fever at presentation	Febrile seizure	Serious etiology	Total
Present	58	9	67
Absent	0	23	23
Total	**58**	**32**	**90**

$\chi^2 = 52.28$, $df = 1$, $p < 0.001$. The zero cell is definitional: febrile seizure requires fever.

Hospital course and outcome

Thirty-one infants (34.4%) required paediatric intensive care, of whom 29 stayed three days or less. Four (4.4%) required mechanical ventilation and four (4.4%) had shock. Hospital stay exceeded three days in 47 infants (52.2%), the maximum being 11 days. Of the 18 infants convulsing at admission, nine were controlled with a single antiepileptic agent and nine required more than one; six of the latter met criteria for refractory seizures.

At discharge, 82 infants (91.1%) were neurologically normal, one (1.1%) had left hemiplegia following a subacute cerebral infarct, five (5.6%) had a pre-existing neurological condition that persisted unchanged, and two (2.2%) had died. Both deaths occurred in infants with a structural etiology. At three months, seizure recurrence had occurred in seven infants. At six months, the infant with hemiplegia had shown clinical improvement and the pre-existing neurological conditions remained stable without progression. Both deaths and the only new persistent deficit therefore occurred within the serious-etiology group, and specifically within its structural and neurovascular subsets.

DISCUSSION

This prospective cohort of 90 infants presenting with a first seizure yields two findings with direct bearing on how such infants should be investigated. First, selectively applied magnetic resonance imaging returned an abnormal result in more than two-thirds of infants scanned, while extended metabolic evaluation returned a diagnostically decisive result in fewer than one in ten. Second, fever — present in three-quarters of the cohort — did not reliably indicate a benign cause, since approximately one febrile infant in seven had a diagnosis other than febrile seizure.

Interpreting the yield gradient

The inverse relationship between how often an investigation was used and how often it was abnormal is the central observation of this study. Magnetic resonance imaging was ordered in only 27.8% of infants and was abnormal in 68% of those scanned; electroencephalography was ordered in 66.7% and was abnormal in 31.7%. This pattern is what one would expect where clinical selection is operating effectively for the more constrained resource: scanner access at the study centre is limited, and the threshold for requesting a scan was correspondingly high, so the infants who reached the scanner were those with the strongest pre-test indication. Electroencephalography, being more accessible, was applied more liberally and to a lower-probability population.

The corollary is that a yield figure is not a fixed property of an investigation. It describes the interaction between a test and the population selected for it, and the 68% reported here should not be read as an argument for imaging all infants with a first seizure — universal imaging would necessarily yield less. It is instead evidence that the existing selection criteria at this centre were performing well, and that similar restraint is defensible elsewhere.

Extended metabolic evaluation warrants separate comment. Its crude yield of 22.7% fell to 9.1% when only diagnostically decisive results were counted, because three of the five abnormal screens showed isolated hyperammonaemia that did not lead to a confirmed inborn error of metabolism. Transient hyperammonaemia is a recognised non-specific finding in the acutely unwell or convulsing infant, and its appearance on a screening panel generates further testing and parental anxiety without a corresponding diagnostic return. These figures support restricting extended metabolic screening to infants with encephalopathy, developmental regression, refractory seizures, consanguinity or a suggestive family history, rather than applying it broadly to infants with unexplained seizures.

Computed tomography in the infant with a first seizure

Computed tomography was abnormal in half of the 14 infants scanned. That figure should be read alongside the established association between paediatric computed tomography and subsequent leukaemia and brain tumours, with risk inversely related to age at exposure.[7,12] The abnormalities that computed tomography detects in this setting — large structural lesions, haemorrhage, hydrocephalus — are for the most part also detectable by magnetic resonance imaging, which detects considerably more besides and delivers no radiation. The reasonable position is that computed tomography should be reserved for the acutely unstable infant in whom immediate exclusion of a surgically actionable lesion is required and magnetic resonance imaging is not feasible within a clinically acceptable interval, rather than used as a first-line screen.

Fever is not a reassuring sign

That 13.4% of febrile infants had a cause other than febrile seizure is, in our view, the most clinically consequential figure in this study. Febrile seizure is so common in late infancy, and its prognosis so benign, that there is a natural clinical gravity towards diagnosing it by default in any infant who convulses with a temperature. These data quantify what that default costs. Bacterial Pyogenic meningitis accounted for five of the seven CNS infections in this cohort, and meningeal signs in infancy are notoriously unreliable. The practical implication is that in the febrile infant, the decision to perform lumbar puncture should turn on the completeness of the clinical picture — level of consciousness, feeding, irritability, tone, the presence of a bulging fontanelle, response to antipyresis — and not on the presence of fever, which is present by definition in both groups.

Comparison with previous studies

The etiological distribution observed here closely resembles that reported from Bengaluru, where febrile seizures accounted for 77.1% of first seizures in infants aged 6–12 months, and differs substantially from a New Delhi cohort in which metabolic causes predominated at 34.3% and neuroinfections accounted for 29.3%.[9,10] In the present cohort metabolic causes accounted for 6.7% and CNS infection for 7.8%. The most plausible explanations are Kerala's high immunisation coverage, its low prevalence of nutritional rickets relative to North India, and early access to primary care and antibiotics. This divergence is itself an argument against transplanting an investigation protocol from one Indian region to another without local calibration, and it is the reason yield figures rather than etiological proportions alone are reported here — yield is the quantity a department can act on. Both deaths in this cohort occurred in infants with structural etiology, and the only new persistent neurological deficit followed a cerebral infarct. No infant with febrile seizure sustained a neurological sequela. This reproduces a consistent observation across paediatric cohorts: prognosis after a first seizure tracks the underlying cause far more closely than it tracks seizure type, duration or number of episodes.[2,13] The purpose of investigation is accordingly not to characterise the seizure but to identify the disease behind it, where identification alters management — antimicrobial therapy in meningitis, calcium repletion in hypocalcaemia, and prompt hormonal therapy in infantile epileptic spasms syndrome, where delay is independently associated with worse developmental outcome.

Limitations

Several limitations qualify these findings. The most important is verification bias. Because investigations were performed on clinical indication rather than by protocol, the population tested was not representative of the whole cohort, and no reference standard was applied to the untested. The yields reported here therefore describe the performance of investigations as used at this centre, and cannot be extrapolated to a policy of universal testing; equally, occult pathology among infants who were not investigated cannot be excluded, so the 35.6% proportion with serious etiology may be an underestimate. Second, the study was conducted at a single tertiary referral centre, and referral bias is likely to have enriched the cohort for serious pathology relative to a community population. Third, with 90 infants and small numbers tested by each modality, confidence intervals around the yield estimates are wide, and the computed tomography figure in particular rests on 14 scans. Fourth, whether an abnormal result actually altered management was not recorded prospectively; the distinction between crude and diagnostically decisive yield could therefore be applied only to the metabolic panel, where the final diagnoses permitted it. Fifth, follow-up extended only to six months, which is insufficient to determine how many infants classified as having a febrile seizure ultimately develop epilepsy. Finally, genetic testing was not routinely available, so monogenic epilepsies among the unprovoked group remain undiagnosed, and seizure semiology was largely derived from caregiver report and is subject to recall bias — a limitation that applies particularly to the distinction between focal and generalized onset in infancy.

Implications and future directions

For departments operating under similar constraints, these data support a graded approach: universal baseline biochemistry, which is cheap and identifies immediately correctable causes; a low threshold for lumbar puncture in the febrile infant with any incompleteness of the clinical picture; magnetic resonance imaging preferred over computed tomography and reserved for infants with focal semiology, abnormal neurological examination, developmental abnormality or unprovoked seizures; and extended metabolic evaluation confined to a narrow set of clinical indications. A prospective multicentre study in which a random sample of infants without clinical indications is investigated to a reference standard would allow verification bias to be quantified and these yields to be converted into a validated selection rule.

CONCLUSION

Among 90 infants beyond the neonatal period admitted with a first seizure, febrile seizure accounted for approximately two-thirds and a serious underlying etiology for the remaining third. Selectively applied magnetic resonance imaging had the highest diagnostic yield at 68%, while extended metabolic evaluation had the lowest and was diagnostically decisive in fewer than one in ten of those screened. Approximately one febrile infant in seven had a diagnosis other than febrile seizure, indicating that fever should not by itself be treated as a reassuring finding. These figures provide an

empirical basis for prioritising investigation in infants with a first seizure in comparable resource-constrained settings.

Declarations

Ethical approval: Institutional Ethics Committee, Government Medical College, Kozhikode; reference number GMCKKD/RP 2024/IEC/173 (IEC registration ECR/395/Inst.KL/2013/RR-25).

Consent: Written informed consent was obtained from a parent or legal guardian of every participant.

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Author contributions: Dr Amritha conceived the study and collected the data; Dr Harsha supervised the work; Dr Shameem analysed the data; all authors drafted the manuscript and approved the final version.

Data availability: The dataset is available from the corresponding author on reasonable request.

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