Section: Paediatrics

Original Research Article

CLINICAL, ETIOLOGICAL CHARACTERISTICS OF CHILDHOOD ARTERIAL ISCHEMIC STROKE AND PROGNOSTIC FACTORS INFLUENCING ITS OUTCOME USING STROKE SCALES

Harshitha C[1], Aksshita S[2], Saranya Muthu[1], Stalin S[3]

[1]Assistant Professor, Department of Paediatrics, Institute of Child Health and Hospital for Children, EGMORE, Tamilnadu, India
[2]Assistant Professor, Department of Paediatrics Institute of Social Obstetrics and Government Kasturba Gandhi Hospital for Women and Children, Chennai, Tamilnadu, India
[3]Professor, Department of Paediatrics, Government Kilpauk Medical College Hospital, Chennai, Tamilnadu, India

Received : 12/04/2026
Received in revised form : 21/05/2026
Accepted : 06/06/2026

Keywords:
Paediatric arterial ischaemic stroke, PedNIHSS, PSOM, aetiology, prognostic factors, neuroimaging.

Corresponding Author:
Dr. Stalin S,
Email: stalin1027@gmail.com

DOI: 10.47009/jamp.2026.8.3.221

Source of Support: Nil,
Conflict of Interest: None declared

Int J Acad Med Pharm
2026; 8 (3); 1240-1247

ABSTRACT

Background: Paediatric arterial ischaemic stroke is a serious neurological condition with high morbidity and mortality. This study evaluated the clinical and etiological characteristics of arterial ischaemic stroke in children aged 2 months to 12 years and analysed outcomes using standardised stroke scales. **Materials and Methods:** This prospective observational study included 50 patients from a tertiary care centre between January 2023 and July 2024. Demographic data, clinical features, and risk factors were recorded. Laboratory investigations, genetic testing, and neuroimaging (MRI/CT) were conducted. Stroke severity was assessed using the PedNIHSS. Outcomes were evaluated using survival status and PSOM at discharge and 6 month follow up. Prognostic factors were systematically analysed. **Result:** Most patients were ≤3 years old (46.0%), male (52.0%), and urban (58.0%). Motor involvement (96.0%) and cranial nerve deficits (60.0%) were the most common symptoms. Term births were 84.0%, and normal development was 92.0%. Neurocutaneous markers were observed in 52.0% of the patients. Infarcts were observed in 94.0% of patients. CBC abnormalities, vasculitis, and cardiac disease were observed in 62.0%, 56.0%, and 20.0% of patients, respectively. Cardiac evaluation showed abnormality in 20.0% (acyanotic heart disease 16.0%, cyanotic heart disease 4.0%), while a clinical history of heart disease was present in 36.0% of patients. L MCA involvement was highest (38.0%). The main aetiologies were mineralising angiopathy and idiopathic (14.0% each). Survival was 96.0%. PedNIHSS correlated with PSOM0 (r=0.609) and PSOM6 (r=0.642). The mean PedNIHSS score was 16.26±1.81, and the PSOM score improved from 2.96±0.77 to 1.54±0.75. **Conclusion:** Pediatric AIS shows diverse clinical and etiological patterns with significant effects. Early diagnosis and stroke scale-based assessment are essential for prognostication and improving neurological outcomes.

INTRODUCTION

Paediatric arterial ischaemic stroke (AIS), although relatively less frequent than in adults, is an important neurological condition associated with high mortality and lasting effects.[1,2] The worldwide occurrence ranges from 1 to 13 per 100,000 per year in children, with about half being ischaemic stroke.[3-5] Although low in frequency, AIS is one of the common causes of neurological disability and death during childhood.[1] This condition is associated with hemiparesis, seizures, problems with speech and vision, and altered levels of consciousness, with age-related signs making the diagnosis difficult.[5,6] The time taken for diagnosis usually surpasses the six-hour therapeutic window, limiting treatments and affecting outcomes.[5] This delay occurs due to variations in signs and limited access to neuroimaging tests. Late effects may consist of mortality ranging from 10% to 25%, along with recurrence of stroke in 25% of children.[1]

Over 50% of children who survive suffer from disabilities, including movement difficulties, learning disorders, epilepsy, and behavioural problems.[5,7] These sequelae burden individuals, families, and healthcare systems, particularly in resource-limited settings. Prognosis is affected by recurrent stroke occurrence, brain involvement, and

ISSN (O): 2687-5365; ISSN (P): 2753-6556

aetiology. Recurrent strokes are associated with a poor prognosis.[7] The aetiology, risk factors, and pathogenesis of paediatric AIS differ from those of adult stroke and require a unique approach. Where adult stroke is associated with atherosclerosis, paediatric AIS has etiological factors such as arteriopathies, congenital heart disease, infections, and haematological disorders.[5,8] Arteriopathies include moyamoya disease and focal cerebral arteriopathy, which are common among the Asian population. In 6 cases congenital heart diseases was found to play a big role in cardioembolic causes. Infections and inflammation play a key role in the pathogenesis of paediatric AIS through vascular damage and a prothrombotic state.[8]

Hematologic disorders such as hereditary thrombophilia as well as other diseases, like iron-deficiency anaemia, add another risk factor in paediatric patients with AIS, especially in underdeveloped countries.[5] Such an array of causative agents emphasizes the intricacy of paediatric AIS and calls for assessment methods that take into account various risk factors. The clinical presentation of paediatric AIS depends on age, site of infarction, and cause. Hemiparesis occurs mostly in AIS cases, while seizures can be seen among younger children in whom cortical infarction occurs.[6] Speech disturbances and visual deficits complicate the diagnosis. Posterior circulation strokes are rare, and ataxia or headache makes diagnosing these cases difficult.[9] These factors demand systematic assessment for better diagnosis and management. The evaluation of outcomes among paediatric patients with AIS is very important for recovery. The use of standardised scales quantifies stroke severity, predicts outcomes, and facilitates research comparisons.

The Pediatric National Institutes of Health Stroke Scale (PedNIHSS) assesses stroke severity and its outcomes. The Modified Rankin Scale (mRS) evaluates AIS-related functional disabilities but ignores cognitive or behavioural issues in paediatric patients. The Paediatric Stroke Outcome Measure (PSOM) reliably assesses sensorimotor, language, and cognitive domains for long-term outcomes.[10] Despite its increased use, its prognostic utility in paediatrics is unclear. Age, developmental level, aetiology subtype, and healthcare facilities may affect reliability. Differences in outcomes and follow-up durations complicate these comparisons. Most studies from developed nations have small sample sizes or retrospective designs, limiting their applicability. There is insufficient region-specific evidence from developing countries, where infections and nutritional deficiencies may affect paediatric AIS presentation and aetiology. Studies have focused on clinical features, aetiology, or outcomes, complicating the identification of prognostic indicators. The combined evaluation of clinical presentation, etiological factors, and standardised outcomes is underexplored, and studies are needed to integrate these domains in defined paediatric populations.

Aim: This study aimed to evaluate the clinical and etiological characteristics of arterial ischaemic stroke in children aged 2 months to 12 years and analyse the associated outcomes using standardised stroke scales.

MATERIALS AND METHODS

This prospective observational study was conducted in paediatric patients attending the Institute of Child Health & Hospital for Children, Egmore, Chennai, from January 2023 to July 2024. Ethical approval was obtained from the Institutional Ethical Committee, and written informed consent was obtained from the parents or legal guardians before the initiation of the study.

Inclusion and Exclusion criteria

This study included children aged 2 months and less than 12 years who presented with acute neurological symptoms suggestive of arterial ischaemic stroke with confirmation of focal brain infarction on neuroimaging (MRI or CT scan). Children with intracranial haemorrhage, space-occupying lesions such as brain tumours, encephalitis, hemiplegic cerebral palsy, and a history of transient ischaemic attack were excluded from the study.

Materials

The materials used in the study included structured data collection forms, laboratory investigations including complete blood count, lipid profile, coagulation profile, inflammatory markers, genetic testing where indicated, neuroimaging modalities such as MRI and CT scan, and standardised stroke assessment scales including the Pediatric NIH Stroke Scale (PedNIHSS) and Paediatric Stroke Outcome Measure (PSOM).

Methods

Eligible children (n=50) were enrolled and evaluated after consent. Demographic details like age, sex, residence, and socioeconomic status were recorded. Clinical history, including symptom onset, pre-existing conditions, and risk factors like infections, trauma, and family stroke history, was documented. The comprehensive assessment included clinical evaluation and laboratory investigations. Blood tests included blood count, lipid, coagulation profiles, and inflammatory markers. Genetic screening was performed in suspected hereditary cases. Neuroimaging with MRI or CT confirmed ischemic lesions' presence, location, and extent.

Stroke severity was assessed at admission using the Pediatric NIH Stroke Scale (PedNIHSS). Outcome assessment focused on survival. Neurological outcomes among survivors were assessed at discharge and at the 6-month follow-up using the Paediatric Stroke Outcome Measure (PSOM), which evaluates sensorimotor, language, and cognitive/behavioural domains. Prognostic factors such as age at onset, stroke severity (PedNIHSS score), aetiology, and comorbid conditions were

ISSN (O): 2687-5365; ISSN (P): 2753-6556

analysed in relation to outcomes. All clinical, etiological, and outcome data were systematically recorded, and follow-up information was documented for recovery and recurrence.

Statistical analysis

Data were presented as mean, standard deviation, frequency, and percentage. Variables were compared using the chi-square test or Fisher's exact test for categorical data and the unpaired t-test for continuous data. Logistic regression analysis was performed. The normality of the data was assessed using the Shapiro-Wilk test, and non-parametric tests were applied where appropriate. Significance was defined as $P<0.05$, and analysis was performed using IBM-SPSS version 23.

RESULTS

The age distribution showed that most patients were ≤3 years (23 [46.0%]), followed by 6.1–9 years (14 [28.0%]), >9 years (8 [16.0%]), and 3.1–6 years (5 [10.0%]). The gender distribution was nearly equal, with 26 (52.0%) males and 24 (48.0 %) females. More patients were from urban (29 [58.0%]) than rural (21 [42.0 %]) areas. Most were lower middle class (21 [42.0%]), followed by upper middle (15 [30.0%]), upper lower (12 [24.0%]), and lower classes (2 [4.0%]). Right-sided limb weakness was more common (31 [62.0%]) than left-sided weakness (19 [38.0%]).

Motor system involvement was the most frequent (48 [96.0%]), followed by cranial nerve involvement (30 [60.0%]), altered sensorium (24 [48.0%]), and seizures (23 [46.0%]). Heart disease was present in 18 (36.0%) patients, with fever and trauma in 16 (32.0%) and 15 (30.0%) patients, respectively. Bleeding manifestations were observed in 11 (22.0%) patients, autonomic disturbances in 10 (20.0%), and sleep disturbances and diarrhoea in 9 (18.0 %) each. Increased intracranial tension and sensory involvement were each seen in 7 (14.0%), and involuntary movements in 6 (12.0%). Similar illnesses were observed in five (10.0%) patients, and cerebellar dysfunction was the least common, observed in three (6.0%) patients [Table 1].

Table 1: Demographic Characteristics, Clinical Presentation, and History of Patients

		N (%)
Age distribution	≤3 yrs	23 (46.0%)
	3.1–6 yrs	5 (10.0%)
	6.1–9 yrs	14 (28.0%)
	>9 yrs	8 (16.0%)
Gender	Male	26 (52.0%)
	Female	24 (48.0%)
Residence	Urban	29 (58.0%)
	Rural	21 (42.0%)
Socioeconomic status	Upper middle	15 (30.0%)
	Lower middle	21 (42.0%)
	Upper lower	12 (24.0%)
	Lower	2 (4.0%)
Side of limb weakness	Right	31 (62.0%)
	Left	19 (38.0%)
Presenting features	Altered sensorium	24 (48.0%)
	Motor system involvement	48 (96.0%)
	Bleeding manifestation	11 (22.0%)
	Involuntary movement	6 (12.0%)
	Seizures	23 (46.0%)
	Increased ICT	7 (14.0%)
	Cranial nerve involvement	30 (60.0%)
	Sensory system involvement	7 (14.0%)
	Cerebellar dysfunction	3 (6.0%)
	Autonomic disturbances	10 (20.0%)
	Sleep disturbances	9 (18.0%)
	Heart disease	18 (36.0%)
	Diarrhoea	9 (18.0%)
	Trauma	15 (30.0%)
	Fever	16 (32.0%)
	Similar illness	5 (10.0%)

All patients had an uneventful antenatal history. Most were term (42 [84.0%]), and 8 (16.0%) were preterm. Delivery was split between vaginal and LSCS, each with 24 (48.0%), and 2 (4.0%) had assisted delivery. The birth weight was mostly in between 2.1–3 kg (22 [44.0%]) and 1.5–2 kg (20 [40.0%]), followed by >3 kg (7 [14.0%]) and <1.5 kg (1 [2.0%]). Development was normal in most (46 [92.0%]) patients and delayed in four (8.0%). Immunisation was complete in 49 (98.0%) patients, with 1 (2.0%) not immunised. Family history was absent in 44 (88.0%) and present in 6 (12.0%) patients. Contact history was absent in 48 (96.0%) patients and present in 2 (4.0%) patients. NICU admission was not needed for 42 (84.0%) patients, and 8 (16.0%) patients had a stay.

The general examination was normal in 45 [90.0%]) and abnormal 5 (10.0%) patients. The nutritional status was normal in 32 (64.0%) and abnormal in 18

ISSN (O): 2687-5365; ISSN (P): 2753-6556

(36.0%) patients. Neurocutaneous markers were present in 26 (52.0%) patients and absent in 24 (48.0%) patients. Higher functions were preserved in 47 (94.0%) patients, and abnormalities were observed in three (6.0%) patients. Cranial nerve examination showed normal and abnormal findings in 25 (50.0%) patients. The sensory system was normal in 49 (98.0%) patients, with 1 (2.0%) abnormality. Cerebellar signs were absent in 46 (92.0%) and present in 4 (8.0%) patients. Spine and cranium examinations were normal in 48 (96.0%) and abnormal in 2 (4.0%) patients. Meningeal signs were absent in 46 (92.0%) and present in 4 (8.0%) patients.

Motor examination showed normal tone in 34 (68.0%) patients and hypertonia and hypotonia in eight (16.0%) patients each. Muscle power was mostly grade 2 (24 [48.0%]), followed by grade 1 (11 [22.0%]), grade 3 (9 [18.0%]), grade 4 (5 [10.0%]), and grade 0 1 (2.0%). Deep tendon reflexes were present in 40 (80.0%), brisk in 7 (14.0%), exaggerated in 1 (2.0%), and absent in 2 (4.0%). Plantar reflex was extensor in 38 (76.0%), flexor in 12 (24.0%). Systemic examination showed normal cardiovascular findings in 39 (78.0%) and abnormal findings in 11 (22.0%) patients. Respiratory and abdominal examinations were normal in all patients (50 [100.0%]).

Table 2: Perinatal History, Developmental Profile, and Clinical Examination Findings of Patients

		N (%)
Antenatal history	Uneventful	50 (100.0%)
Gestational age	Term	42 (84.0%)
	Preterm	8 (16.0%)
Mode of delivery	Normal	24 (48.0%)
	LSCS	24 (48.0%)
	Assisted	2 (4.0%)
Birth weight	<1.5 kg	1 (2.0%)
	1.5–2 kg	20 (40.0%)
	2.1–3 kg	22 (44.0%)
	>3 kg	7 (14.0%)
Development	Normal	46 (92.0%)
	Delay	4 (8.0%)
Immunisation	Given	49 (98.0%)
	Not given	1 (2.0%)
Family history	Present	6 (12.0%)
	Absent	44 (88.0%)
Contact history	Present	2 (4.0%)
	Absent	48 (96.0%)
NICU admission	Present	8 (16.0%)
	Absent	42 (84.0%)
General examination	Normal	45 (90.0%)
	Abnormal	45 (90.0%)
Nutritional status	Normal	32 (64.0%)
	Abnormal	18 (36.0%)
Neurocutaneous markers	Present	26 (52.0%)
	Absent	24 (48.0%)
Higher functions	Normal	47 (94.0%)
	Abnormal	3 (6.0%)
Cranial nerve exam	Normal	25 (50.0%)
	Abnormal	25 (50.0%)
Sensory system	Normal	49 (98.0%)
	Abnormal	1 (2.0%)
Cerebellar signs	Normal	46 (92.0%)
	Abnormal	4 (8.0%)
Spine & cranium	Normal	48 (96.0%)
	Abnormal	2 (4.0%)
Meningeal signs	Normal	46 (92.0%)
	Abnormal	4 (8.0%)
Tone	Normal	34 (68.0%)
	Hypertonia	8 (16.0%)
	Hypotonia	8 (16.0%)
Power (MRC)	0	1 (2.0%)
	1	11 (22.0%)
	2	24 (48.0%)
	3	9 (18.0%)
	4	5 (10.0%)
Deep tendon reflex	Absent	2 (4.0%)
	Present	40 (80.0%)
	Brisk	7 (14.0%)
	Exaggerated	1 (2.0%)
Plantar reflex	Flexor	12 (24.0%)
	Extensor	38 (76.0%)
CVS	Normal	39 (78.0%)

ISSN (O): 2687-5365; ISSN (P): 2753-6556

	Abnormal	11 (22.0%)
RS	Normal	50 (100.0%)
P/A	Normal	50 (100.0%)

CT brain findings showed infarction in 47 (94.0%) patients; porencephalic cyst, tuberculoma, and other findings each in 1 (2.0%). Abnormal complete blood counts were observed in 31 (62.0%) patients, with 19 (38.0%) having normal counts. Peripheral smear abnormalities were observed in 11 (22.0%) patients, and 39 (78.0%) patients had normal results. ESR was elevated in 7 (14.0%) and normal in 43 (86.0 %) patients. Glucose and renal and liver function tests were normal in 49 (98.0%) patients and abnormal in 1 (2.0%) patient each. Urine examination was normal in 48 (96.0%) and abnormal in 2 (4.0%) patients. Viral markers (HIV) were positive in 5 (10.0%) and negative in 45 (90.0%) patients. Sickling test and lipid profile abnormalities were observed in 1 (2.0%) patient each, and 49 (98.0%) patients had normal results.

Infective profile abnormalities were observed in 7 (14.0%) and 43 (86.0%) patients. The coagulation profile was abnormal in 5 (10.0%) and normal in 45 (90.0%) patients. TB screening was positive in 1 (2.0%) and negative in 49 (98.0%) patients. Serum lactate and ammonia levels were elevated in 4 (8.0%) patients each and normal in 46 (92.0%) patients. TMS was normal in all patients (50 [100.0%]). Chest X-ray abnormalities were observed in 6 (12.0%) and 44 (88.0%) patients. CSF analysis was abnormal in 2 (4.0%) and normal in 48 (96.0%) patients. Prothrombotic workup abnormalities were observed in 3 (6.0%) patients, and 47 (94.0%) patients had normal results. Vasculitis profile abnormalities were observed in 28 (56.0%) and 22 (44.0%) patients. Carotid Doppler was performed in 56 patients; thrombus was detected in 7 (12.5%) and absent in 43 (87.5%). 14 patients did not undergo Doppler assessment. Cardiac evaluation was normal in 40 (80.0%) patients, with acyanotic heart disease in 8 (16.0%) and cyanotic heart disease in 2 (4.0%) [Table 3].

Table 3: Neuroimaging and Laboratory Investigation Findings of Patients

		N (%)
CT Brain	Infarct	47 (94.0%)
	Porencephalic cyst	1 (2.0%)
	Tuberculoma	1 (2.0%)
	Others	1 (2.0%)
CBC	Abnormal	31 (62.0%)
	Normal	19 (38.0%)
Peripheral smear	Abnormal	11 (22.0%)
	Normal	39 (78.0%)
ESR	Abnormal	7 (14.0%)
	Normal	43 (86.0%)
Glucose	Abnormal	1 (2.0%)
	Normal	49 (98.0%)
RFT	Abnormal	1 (2.0%)
	Normal	49 (98.0%)
LFT	Abnormal	1 (2.0%)
	Normal	49 (98.0%)
Urine routine	Abnormal	2 (4.0%)
	Normal	48 (96.0%)
Viral markers (HIV)	Abnormal	5 (10.0%)
	Normal	45 (90.0%)
Sickling	Abnormal	1 (2.0%)
	Normal	49 (98.0%)
Lipid profile	Abnormal	1 (2.0%)
	Normal	49 (98.0%)
Infective profile	Abnormal	7 (14.0%)
	Normal	43 (86.0%)
Coagulation profile	Abnormal	5 (10.0%)
	Normal	45 (90.0%)
TB screening	Abnormal	1 (2.0%)
	Normal	49 (98.0%)
Serum lactate	Abnormal	4 (8.0%)
	Normal	46 (92.0%)
Ammonia	Abnormal	4 (8.0%)
	Normal	46 (92.0%)
TMS	Normal	50 (100.0%)
CXR	Abnormal	6 (12.0%)
	Normal	44 (88.0%)
CSF analysis	Abnormal	2 (4.0%)
	Normal	48 (96.0%)
Prothrombotic workup	Abnormal	3 (6.0%)
	Normal	47 (94.0%)
Vasculitis profile	Abnormal	28 (56.0%)

ISSN (O): 2687-5365; ISSN (P): 2753-6556

	Normal	22 (44.0%)
Carotid Doppler thrombus	Present	7 (14.0%)
	Absent	43 (86.0%)
Cardiac evaluation	Normal	40 (80.0%)
	Acyanotic CHD	8 (16.0%)
	Cyanotic CHD	2 (4.0%)

In MRI territories, the left middle cerebral artery (L MCA) was affected in 19 (38.0%) patients, followed by the right MCA in 12 (24.0%) and the left internal carotid artery (L ICA) in 9 (18.0%). Combined involvement of the LMCA + ACA, L PCA, and PICA occurred in two (4.0%) patients. Less frequent were bilateral MCA, bilateral PCA, right ICA, and RMCA + ACA, each in one (2.0%) patient. Mineralising angiopathy and idiopathic causes were the most common, each in seven (14.0%) patients, followed by moyamoya disease in six (12.0%) patients.

Infective causes and ventricular septal defect (VSD) were observed in five (10.0%) patients each. Homocystinemia, iron deficiency anaemia (IDA), protein C and S deficiency, and rheumatic heart disease (RHD) were observed in two (4.0%) patients each. Less common aetiologies (n = 1, 2.0% each) included APLA, Ebstein anomaly, MTHFR mutation, nephrotic syndrome, NF1, post-varicella, protein S deficiency, thrombocytopenia, tetralogy of Fallot (TOF), trisomy 21, tuberculosis, and vasculitis. Most patients survived (48 [96.0%]), with mortality in two (4.0%) [Table 4].

Table 4: MRI Distribution, Etiological Spectrum, and Outcome of Patients

		N (%)
MRI territories	L MCA	19 (38.0%)
	R MCA	12 (24.0%)
	L ICA	9 (18.0%)
	LMCA + ACA	2 (4.0%)
	L PCA	2 (4.0%)
	PICA	2 (4.0%)
	B/L MCA	1 (2.0%)
	B/L PCA	1 (2.0%)
	R ICA	1 (2.0%)
	RMCA + ACA	1 (2.0%)
Etiology	APLA	1 (2.0%)
	Ebstein anomaly	1 (2.0%)
	Homocystinemia	2 (4.0%)
	IDA	2 (4.0%)
	Idiopathic	7 (14.0%)
	Infective	5 (10.0%)
	Mineralising angiopathy	7 (14.0%)
	Moyamoya	6 (12.0%)
	MTHFR mutation	1 (2.0%)
	Nephrotic syndrome	1 (2.0%)
	NF1	1 (2.0%)
	Post varicella	1 (2.0%)
	Protein C & S deficiency	2 (4.0%)
	Protein S deficiency	1 (2.0%)
	RHD	2 (4.0%)
	Thrombocytopenia	1 (2.0%)
	TOF	1 (2.0%)
	Trisomy 21	1 (2.0%)
	Tuberculosis	1 (2.0%)
	Vasculitis	1 (2.0%)
	VSD	5 (10.0%)
Outcome	Death	2 (4.0%)
	Survived	48 (96.0%)

Correlation analysis showed a significant positive association between the PedNIHSS and PSOM scores. The PedNIHSS correlated with PSOM at admission (PSOM0) ($r = 0.609$, $p = 0.0001$) and was stronger at 6 months (PSOM6) ($r = 0.642$, $p = 0.0001$), indicating that higher initial stroke severity was linked to poorer outcomes at baseline and follow-up [Table 5].

Table 5: Correlation Between Stroke Severity and Outcome Scores

	Correlation Value	P value
PedNIHSS vs PSOM0	0.609	0.0001
PedNIHSS vs PSOM6	0.642	0.0001

The mean age of the patients was 5.03 ± 3.77 years. The mean PedNIHSS score was 16.26 ± 1.81, indicating a moderate-to-severe stroke. The mean PSOM score at admission (PSOM0) was 2.96 ± 0.77, which decreased to 1.54 ± 0.75 at 6 months (PSOM6), showing improvement [Table 6].

ISSN (O): 2687-5365; ISSN (P): 2753-6556

Table 6: Descriptive Statistics of Clinical and Outcome Measures

	N	Mean ± SD
Age	50	5.03 ± 3.77
PedNIHSS	49	16.26 ± 1.81
PSOM0	50	2.96 ± 0.77
PSOM6	50	1.54 ± 0.75

DISCUSSION

Our study evaluated paediatric arterial ischaemic stroke (AIS) characteristics, which aligned with the existing literature and regional variations. 46% of patients were ≤3 years, with a mean age of 5.03 ± 3.77 years, similar to Sood et al.'s median age of 24 months, indicating early childhood stroke predominance.[11] Rafay et al. reported a higher median age of 7.45 years in children with arteriopathy, showing etiological variations.[12] DeVeber et al. noted a broader age range affecting neonates and older children.[13] Male predominance was mild (52%), consistent with that reported by deVeber et al. (55%) and Sood et al. (57%).[11,13]

The clinical presentation in our study was mainly motor, with motor system involvement in 96% of patients, cranial nerve involvement in 60%, seizures in 46%, and altered sensorium in 48%. Cooper et al. reported that hemiplegia is common in childhood AIS and DeVeber et al. noted focal deficits in 77% of older children and seizures in 88% of neonates, showing an age-dependent variation.[13,14] Sood et al. linked seizures, encephalopathy, and low Glasgow Coma Scale scores with severe disability, supporting the 48% altered sensorium in our study.[11]

Our study's etiological profile showed mineralising angiopathy and idiopathic causes (14% each), followed by moyamoya (12%) and infective causes (10%). Khileri et al. identified mineralising angiopathy as common, while Sood et al. reported arteriopathy in 77% of patients, with 42% infection-associated and 13% mineralising angiopathy.[11,15] Rafay et al. reported arteriopathy in 34% of patients, with subtypes including dissection (27%), moyamoya disease (24.5%), focal cerebral arteriopathy (15%), and vasculitis (15%).[12] These findings confirm arteriopathy as the primary mechanism of paediatric AIS.

Kamate et al. highlighted the association of mineralising angiopathy with early life factors, noting a 68.2% perinatal infection rate in affected children.[16] Our study found an uneventful antenatal history in 100% of patients, suggesting that postnatal factors play a larger role. Gorodetsky et al. described mineralising angiopathy affecting young children with basal ganglia infarcts, often after minor trauma, with hemiparesis common in 12 of 14 cases.[17]

Our neuroimaging findings showed left MCA involvement in 38.0%, right MCA involvement in 24.0%, and left ICA involvement in 18.0% of patients, aligning with Rafay et al.'s anterior circulation findings.[12] Laboratory results revealed CBC abnormalities (62.0%), vasculitis profile (56.0%), coagulation profile (10.0%), and cardiac issues (20.0%), similar to the study by deVeber et al.[13] The higher prevalence of vasculitis and infection markers reflects regional differences in developing areas. We showed 96.0% survival and 4.0% mortality, comparable to deVeber et al.'s 5% stroke-specific mortality rate.[13] Long-term neurological impairment remains significant, with Fearn et al. reporting poor outcomes in 43% of patients and recurrence in 15% of patients.[18]

Sood et al. found that 61% of survivors had sensory-motor deficits and 39% had severe disability.[11] The mean PedNIHSS was 16.26 ± 1.81, indicating moderate-to-severe stroke severity, with PSOM scores improving from 2.96 ± 0.77 at admission to 1.54 ± 0.75 at 6 months, showing partial recovery. The strong correlation between the PedNIHSS and PSOM scores suggests that the initial stroke severity predicts outcomes, consistent with the validation of the PSOM by Cooper et al. and Slim et al.[14,19] Khileri et al. reported higher PSOM scores linked to age 2-5 years, impaired consciousness, and CNS tuberculosis, reinforcing early severity indicators' importance.[15]

Limitations: This study is limited by a small sample size, restricting subgroup analysis and generalisability. As a single-centre study from a tertiary hospital in Chennai, findings may not reflect wider epidemiology. The 18-month duration limited long-term outcome assessment. Larger multicentre studies with genetic profiling are needed.

CONCLUSION

This study provides insights into the clinical and etiological spectrum of AIS in children, highlighting the importance of early diagnosis, individualised management, and comprehensive rehabilitation. Given the challenges of paediatric AIS, a multidisciplinary approach is vital to optimise outcomes and improve the quality of life. Integrating the latest research underscores the need for advancements in paediatric AIS care, including tailored interventions, socioeconomic support, and targeted rehabilitation strategies. Ongoing research is crucial to deepen the understanding of paediatric AIS pathophysiology and improve prognostic and therapeutic protocols tailored to the developmental needs of young stroke patients.

REFERENCES

1. Sultan T. Pediatric stroke: a review. Pakistan Journal of Neurological Sciences 2023;17. https://doi.org/10.56310/pjns.v17i03.215.
2. Amlie-Lefond C. Evaluation and acute management of ischemic stroke in infants and children. Continuum (Minneap

ISSN (O): 2687-5365; ISSN (P): 2753-6556

Minn) 2018; 24:150–70. https://doi.org/10.1212/CON.0000000000000559.
3. Klucka J, Stourac P, Stoudek R, Toukalkova M, Harazim H, Kosinova M, et al. Ischemic stroke in paediatrics - narrative review of the literature and two cases. Biomed Pap Med Fac Univ Palacky Olomouc Czech Repub 2017; 161:24–30. https://doi.org/10.5507/bp.2016.053.
4. Ferriero DM, Fullerton HJ, Bernard TJ, Billinghurst L, Daniels SR, DeBaun MR, et al. Management of stroke in neonates and children: A scientific statement from the American heart association/American stroke association. Stroke 2019;50: e51–96. https://doi.org/10.1161/STR.0000000000000183.
5. Pangprasertkul S, Borisoot W, Buawangpong N, Sirikul W, Wiwattanadittakul N, Katanyuwong K, et al. Comparison of arterial ischemic and hemorrhagic pediatric stroke in etiology, risk factors, clinical manifestations, and prognosis. Pediatr Emerg Care 2022;38: e1569–73. https://doi.org/10.1097/PEC.0000000000002614.
6. Akter N, Anwar S, Samshad IA, Akter S. Clinical spectrum and neuroimaging characteristics of arterial ischemic stroke in childhood: Experience in a tertiary care hospital. J Enam Med Coll 2024; 12:36–42. https://doi.org/10.3329/jemc.v12i1.71692.
7. Aprasidze T, Tatishvili N, Shatirishvili T, Lomidze G. Predictors of neurological outcome of arterial ischemic stroke in children. J Pediatr Neurol 2021; 19:161–5. https://doi.org/10.1055/s-0040-1701204.
8. Bartoszek L, Kapłan W, Ostański J, Zalewa K, Orłowska D, Świdziński R, et al. Etiology and risk factors for strokes in the pediatric population. J Educ Health Sport 2024; 52:181–96. https://doi.org/10.12775/jehs.2024.52.013.
9. Fink M, Slavova N, Grunt S, Perret E, Regényi M, Steinlin M, et al. Posterior arterial ischemic stroke in childhood: Clinical features and neuroimaging characteristics. Stroke 2019; 50:2329–35. https://doi.org/10.1161/STROKEAHA.119.025154.
10. Elbers J, deVeber G, Pontigon A-M, Moharir M. Long-term outcomes of pediatric ischemic stroke in adulthood. J Child Neurol 2014; 29:782–8. https://doi.org/10.1177/0883073813484358.
11. Sood A, Suthar R, Sahu JK, K Baranwal A, Saini AG, Saini L, et al. Etiologic profile of childhood stroke from north India: Is it different from developed world? J Child Neurol 2021; 36:655–63. https://doi.org/10.1177/0883073821991291.
12. Rafay MF, Shapiro KA, Surmava A-M, deVeber GA, Kirton A, Fullerton HJ, et al. Spectrum of cerebral arteriopathies in children with arterial ischemic stroke. Neurology 2020;94: e2479–90. https://doi.org/10.1212/WNL.0000000000009557.
13. deVeber GA, Kirton A, Booth FA, Yager JY, Wirrell EC, Wood E, et al. Epidemiology and outcomes of arterial ischemic stroke in children: The Canadian Pediatric Ischemic Stroke Registry. Pediatr Neurol 2017; 69:58–70. https://doi.org/10.1016/j.pediatrneurol.2017.01.016.
14. Cooper AN, Anderson V, Hearps S, Greenham M, Hunt RW, Mackay MT, et al. The Pediatric Stroke Outcome Measure: A predictor of outcome following arterial ischemic stroke: A predictor of outcome following arterial ischemic stroke. Neurology 2018;90: e365–72. https://doi.org/10.1212/WNL.0000000000004906.
15. Khileri B, Choudhary PK, Meena AK, Parakh E, Rathore B, Parakh M. Pediatric stroke and predictors of neurological outcome: A prospective observational study. J Neurosci Rural Pract 2025; 16:192–8. https://doi.org/10.25259/jnrp_413_2024.
16. Kamate M, Reddy NA, Detroja M. Perinatal infections: An important etiological risk factor for mineralizing angiopathy. Indian J Pediatr 2021; 88:58–60. https://doi.org/10.1007/s12098-020-03332-w.
17. Gorodetsky C, Pulcine E, Krishnan P, Singh J, Moharir M, MacGregor D, et al. Childhood arterial ischemic stroke due to mineralizing angiopathy: an 18-year single-center experience. Dev Med Child Neurol 2021; 63:1123–6. https://doi.org/10.1111/dmcn.14903.
18. Fearn ND, Yau ML, Gajera J, Monagle P, Grobler A, Stojanovski B, et al. Factors influencing long-term outcomes in childhood arterial ischemic stroke. Pediatr Neurol 2025; 169:123–30. https://doi.org/10.1016/j.pediatrneurol.2025.05.015.
19. Slim M, Fox CK, Friefeld S, Dlamini N, Westmacott R, Moharir M, et al. Validation of the pediatric stroke outcome measure for classifying overall neurological deficit. Pediatr Res 2020; 88:234–42. https://doi.org/10.1038/s41390-020-0842-5.

ISSN (O): 2687-5365; ISSN (P): 2753-6556