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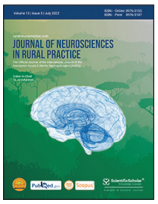

Original article

Pediatric stroke and predictors of neurological outcome: A prospective observational study

Birbal Khileri[1], Puneet Kumar Choudhary[2], Ankit Kumar Meena[3], Esha Parakh[4], Bhanupratap Rathore[1], Manish Parakh[1]

[1]Department of Pediatrics, Dr Sampurnanand Medical College, Jodhpur, Rajasthan, [2]Department of Pediatrics, All India Institute of Medical Sciences, Patna, Bihar, [3]Department of Pediatrics, All India Institute of Medical Sciences, Bathinda, Punjab, India, [4]MBChB, Aston Medical School, Aston University, United Kingdom, Birmingham, United Kingdom.

ABSTRACT

Objectives: Stroke in children is a significant cause of mortality and neurological disability in the long term. There is a paucity of data regarding the factors that affect neurological outcomes in childhood stroke patients.

Materials and Methods: This prospective cohort study aimed to explore the clinical profile of children with stroke at a tertiary care hospital in western India and the factors responsible for neurological disability. The study population consisted of children up to 18 years of age. Clinical, radiological, and laboratory data at the time of stroke onset were collected. Neurological disability was assessed through the pediatric stroke outcome measure (PSOM) at the time of stroke and 6 months post-stroke. The PSOM is a validated structured neurological tool for outcome assessment in pediatric stroke patients.

Results: Fifty-five children were enrolled in the study over 2 years. Arterial-ischemic stroke was the most common (75%) type of stroke, followed by hemorrhagic stroke (13%). Overall, mineralizing angiopathy was the most common cause of stroke. The factors associated with higher PSOM scores at the time of stroke were age between 2 and 5 years, central nervous system tuberculosis, bilateral hemispheric involvement, impaired consciousness, and the presence of anemia. Forty-four children completed follow-up at 6 months after stroke onset. The factors associated with significantly higher PSOM scores were age between 2 and 5 years, central nervous system tuberculosis, and impaired consciousness at presentation ($P < 0.05$). The mean PSOM score was lowest at onset (1.53, standard deviation [SD] = 1.17) and 6 months post-stroke (0.5, SD = 0.42) in children with mineralizing angiopathy.

Conclusion: At 6 months post-stroke, children who present with stroke onset between 2 and 5 years of age, impaired consciousness at the time of stroke, and neurotuberculosis have slower recovery with greater disability. Children with mineralizing angiopathy have relatively better recovery with fewer disabilities, as assessed by the PSOM.

Keywords: Childhood stroke, Pediatric stroke outcome measure, Pediatric stroke

INTRODUCTION

Pediatric stroke is a major cause of mortality and long-term neurological disability in children. The prevalence of childhood stroke varies from 2 to 13/100,000 children.[1,2] While there has been a decline in the overall prevalence of stroke and mortality over the last few decades, the absolute number of stroke cases is increasing. In addition, the incidence rates of ischemic stroke in children from developing countries may not have declined; thus, pediatric stroke remains a significant health concern in children.[3]

Although pediatric stroke is rare, stroke in children exhibits age-related variations in risk factors, etiopathogenesis, and clinical presentation. In contrast to adults, where arteriosclerosis is the primary cause, pediatric stroke is associated with multiple risk factors, including cardiac disorders, infections, prothrombotic conditions, moyamoya disease, and others. Pediatric strokes have a subtle non-specific presentation as compared to adults who show clear focal deficits at the onset.[4] Arteriopathies secondary to central nervous system infection are the most common cause of arterial ischemic stroke in India and other low- to middle-income countries. Stroke secondary to mineralizing angiopathy is common in India and is now being increasingly recognized. In developed countries, non-infectious arteriopathies, cardiac disorders, and prothrombotic states are the predominant causes.[5,6] The notion that children exhibit superior recovery after a stroke compared with

***Corresponding author:** Manish Parakh, Department of Pediatrics, Dr Sampurnanand Medical College, Jodhpur, Rajasthan. manparakh@gmail.com

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adults is probably a common misconception. The long-term neurological outcomes following a stroke in children are akin to those reported in adults.[7] The neurological sequelae of stroke vary from death to long-term disabilities such as motor and speech impairments, epilepsy, intellectual disability, and an overall impaired quality of life.[8] In a study by Sood *et al*. from North India, the pediatric stroke outcome measure (PSOM) score was >1, suggesting a moderate deficit in 83% of children at the 3-month follow-up.[6] There is a paucity of studies evaluating neurological outcomes over a longer period from developing countries and the factors that impact disability. In view of their different etiological profiles and long-term outcomes in low- to middle-income countries, children are likely to follow a different trajectory than those in developed countries. In this prospective cohort study, we examined the clinical, laboratory, and radiological profiles of children with stroke, as well as the neurological outcomes and factors responsible for long-term disability.

MATERIALS AND METHODS

This single-center cohort study was conducted at a tertiary care and research hospital in western India using the principles of Good Clinical Practice and the Declaration of Helsinki, over a period of 2 years. The study population included consecutive children and adolescents (up to the age of 18 years) with stroke who had acute neurological deficits of vascular origin confirmed by neuroimaging. Children with acute-onset focal deficits of nonvascular origin, such as channelopathies and metabolic diseases, or those with incomplete clinical, laboratory, and radiological details were excluded. We defined the type of stroke as ischemic, cerebral venous thrombosis, or hemorrhagic as per the American Heart Association guidelines. All the children were treated as per the standard management guidelines.[9] Rehabilitation was initiated, and the children were followed up regularly by a team of pediatric neurologists, physiotherapists, speech therapists, and occupational therapists. Neuroimaging was performed on the basis of clinical assessment, with magnetic resonance angiography or venography conducted as indicated. The baseline laboratory parameters included a hemogram, serum C-reactive protein level, serum iron level, serum ferritin level, and total iron-binding capacity. Anemia and iron deficiency were defined according to the World Health Organization definitions.[10,11] Details of the prothrombotic workup, including prothrombin time and index, antithrombin III levels, protein C functional assay, protein S functional assay, homocysteine levels, antiphospholipid antibodies, factor V Leiden mutation, and methylenetetrahydrofolate reductase (MTHFR) mutation, were performed as clinically indicated and documented.

In addition, hemoglobin electrophoresis, rheumatological tests, and other relevant tests were performed and recorded on the basis of the patient's clinical profile to determine the etiology of the stroke. Children were enrolled after written informed consent was obtained from the parents or guardians. The institutional ethics committee approved the study.

The primary aim of this study was to assess neurological disability in children at the time of stroke and 6 months after stroke as well as to identify the factors affecting disability. The secondary aim was to delineate the clinical, laboratory, and radiological profiles of the study cohort. For the assessment of neurological disability, the PSOM was used. The PSOM is a structured neurological assessment tool comprising 115 items across the following domains: Language, cognition, motor skills, cranial nerves, sensory perception, cerebellar function, and gait. For children younger than 2 years, the PSOM consists of developmental milestones and reflexes. The total PSOM score ranges from 0 to 10, encompassing five subscales: Right sensorimotor, left sensorimotor, language production, language comprehension, and cognitive/behavior. A score of 10 represents maximum impairment, and 0 suggests no deficit. It is a validated tool in pediatric stroke and is appropriate for retrospective and prospective studies.[12] PSOM scoring was performed at the time of acute stroke and 6 months post-stroke. The grading of neurological deficits according to the PSOM score is as follows: 0 – No deficit, 0.5–1 – Mild–moderate deficit, and 2 – Severe deficit with missing function. A detailed clinical evaluation was performed during the follow-up visits, and information was systematically documented on a predefined data form. Appropriate rehabilitation in the form of physiotherapy, occupational therapy, and speech therapy was started early, and compliance was ensured during the follow-up visits.

Statistical methods

Considering a mean PSOM change of 0.5 over 6 months, a standard deviation (SD) of 1.3, a power of 80%, and a significance level of 0.05, the expected sample size was 44.[13] Considering a 20% attrition rate, we planned to enroll 55 children. For continuous variables, the means ± SDs or medians and interquartile ranges were used depending on the type of distribution. For categorical variables, proportions or percentages with 95% CIs were used. The data distribution was assessed through the Kolmogorov–Smirnov test and the Shapiro–Wilk test. Continuous variables were compared between groups through the Student's *t*-test or the Mann–Whitney test. Categorical variables were compared between groups through the Chi-square test, Fisher's exact test, or the Kruskal–Wallis test, as applicable. Statistical significance was established at the 5% significance level.

RESULTS

A total of 55 consecutive children (33 males) with stroke were enrolled in the study over 2 years. The mean age of the cohort

at the time of enrollment was 35.52 ± 43.03 months. Twenty-six children (47.27%) were aged 6–24 months, nine children (16.36%) were under 6 months, eight children (14.54%) were aged 24–60 months, eight children (14.54%) were aged 60–120 months, and four children (7.27%) were over 120 months. Seventy percent of the children in the study were from rural areas. At the time of presentation, seizures were noted in 34 (62%) children, and impaired consciousness was noted in 28 (51%) children. Hemiparesis was observed in 39 children (71%) and was the most common pattern of motor weakness. Other less common neurological deficits observed included quadriparesis in 5 (9%) children, ataxia in 2 children (3.6%), facial palsy (31%), and movement disorders (14.5%). Thirty-three (60%) children were brought to the hospital within 24 h of stroke symptom onset, and only 5 (9%) presented within 6 h of stroke onset. Most of the children (32.72%) presented between 12 and 24 h, 10 (18%) presented within 12 h, and 12 (21.81%) presented 3 days after stroke symptom onset. Laboratory investigations revealed anemia in 75% of the children. The serum ferritin level was assessed in 31 out of 55 children and was found to be low in 7 (23%) patients, all of whom had arterial ischemic stroke. All the children underwent a magnetic resonance imaging of the brain or a computed tomography (CT) scan of the head to confirm stroke. Non-contrast CT of the head is diagnostic for mineralizing angiopathy, and no additional imaging was performed for these children.

Arterial ischemic stroke was the most common type of stroke observed in 41 (75%) children, followed by hemorrhagic stroke in 7 (13%), cerebral sinovenous thrombosis (CSVT) with hemorrhagic transformation in 4 (7%), and CSVT alone in 3 children (5%). Magnetic resonance or CT angiography was performed in 30/55 children and was found to be abnormal in 18/30 (60%). Mineralizing angiopathy was the most significant cause of stroke, accounting for 27% of the total cases. All the children with mineralizing angiopathy had a history of hemiparesis following trivial trauma, and non-contrast CT of the head revealed punctate calcifications in the basal ganglia region. The mean age of the children with mineralizing angiopathy was 12 months (SD-2.44). The circulation involvement was anterior in 32 (60%) children, posterior in 3 (6%) children, and combined in 18 (34%) children. The etiology of the stroke in the cohort is depicted in Table 1.

The mean PSOM score at the time of stroke was 2.45 (2.3). Forty-four children (80%) were available at follow-up for PSOM scoring at 6 months and had a mean (SD) PSOM score of 1.13 (1.4). The PSOM scores at baseline and the 6-month follow-up, in relation to factors affecting outcomes, are summarized in Table 2.

DISCUSSION

The current study was designed to study pediatric stroke outcomes at 6 months post-stroke and determine factors affecting outcomes. Mineralizing angiopathy and central nervous system tuberculosis were the two most common causes of stroke in our cohort, accounting for 44% of the cases. Overall, infections constituted approximately a quarter of the stroke cases in our study. In low- to middle-income countries, infections continue to be a major etiological factor for stroke in children, contributing to approximately 40% of cases. In developed countries, non-infectious causes are predominant, and infections contribute to only approximately 15% of cases.[5,14] Mineralizing angiopathy, a common cause of stroke in infants, manifests as acute-onset stroke following a trivial fall and is associated with calcification of the lenticulostriate arteries.[15] A prothrombotic state was detected in six children (11%). Two patients had MTHFR mutations and hyperhomocysteinemia (with homocysteine levels exceeding 15 μmol/L). One patient, each with protein C and protein S deficiency, was identified, with a diagnosis confirmed by repeat testing performed after 3 months. The PSOM score was not different between the ischemic, hemorrhagic, CSVT, or CSVT with hemorrhagic transformation groups at the onset of stroke ($P = 0.422$) or the 6-month follow-up ($P = 0.135$). When PSOM scores were analyzed according to the underlying etiology of stroke, the PSOM score at the onset of stroke was significantly higher in central nervous system tuberculosis patients than in those with mineralizing angiopathy and other etiologies combined ($P < 0.05$). Both conditions have different etiologies but have lenticulostriate regions as the most common stroke location. Similarly, the PSOM score at 6 months post-stroke was significantly greater in children with central nervous system tuberculosis and lowest in children with mineralizing angiopathy ($P < 0.05$).

In this study, 60% of the children with acute stroke reached the hospital within 24 h of stroke, and only 5 (9%) presented to the hospital before 6 h of stroke onset. Other studies have estimated that the average delay in presentation is up to 28.5 h, and only a quarter are present within the first 6 h.[16,17] Although data concerning the efficacy of reperfusion therapy for ischemic stroke in children are scarce due to the lack of randomized controlled trials, the American Heart Association and American Stroke Association recommend considering perfusion therapy in select cases with persisting neurological deficits, radiologically confirmed cerebral artery occlusion, and expertise available at the center.[9] Thus, it is vital that children with stroke, particularly those with arterial ischemic stroke, present to the hospital within time for acute perfusion therapies and neuroprotective measurements. Our study revealed that, at the time of acute stroke, the PSOM score was higher in children with late presentation. Similarly, the PSOM score was higher at 6 months post-stroke in those who presented late. However, these differences in the PSOM score were not statistically significant. None of the children in this study underwent acute reperfusion therapy.

Table 1: Baseline characteristics of the cohort, *n*=55.

	Overall cohort (*n*-55)	Acute ischemic stroke (*n*-41)	Non-ischemic stroke (*n*-14)
Mean age at presentation in months (SD)	35.5 (43)	31.9 (36.1)	46 (59.4)
Male (%)	33 (60)	24 (58)	9 (64)
Etiology			
1. Arterial ischemic stroke - 41 (75%) • Mineralizing angiopathy - 15 • Central nervous system tuberculosis - 07 • Cyanotic congenital heart disease - 02 • Non-tubercular meningoencephalitis - 02 • Orbital cellulitis and mastoiditis - 02 • Takayasu arteritis - 02 • Prothrombotic states - 06 (MFTHR mutation: 02, Hyperhomocysteinemia: 02, Protein C and S deficiency: 02) • Moyamoya disease - 02 • Post-congenital heart disease corrective surgery - 02 • Neck manipulation - 01 2. Hemorrhagic - 7 (13%) • Arteriovenous malformation - 05 • Intracranial aneurysm - 01 • Collagen synthesis defect (COL4A1 mutation) - 01 3. CSVT - 3 (5%) • Orbital cellulitis - 02 • Mastoiditis - 01 4. CSVT with hemorrhagic transformation - 4 (7%)			

CSVT: Cerebral sinovenous thrombosis, MFTHR: Methylenetetrahydrofolate reductase, SD: Standard deviation

At the time of the acute stroke, disability was greater in the 2- to 5-year age group (mean PSOM score 5.5, SD-3.02) than in the other age groups, and this difference was statistically significant. Bilateral hemispheric involvement, impaired consciousness, and severe anemia were other factors associated with higher PSOM scores at the time of stroke. The International Pediatric Stroke Study also identified bilateral ischemia and impaired consciousness as predictors of early poor outcomes in pediatric stroke patients.[18] In addition, children with stroke are more likely to have IDA than those without stroke.[19] There is evidence in the adult population that anemia is a risk factor for poor outcomes and increases hospital stay duration and mortality.[20]

The factors associated with higher PSOM scores 6 months after stroke were age between 2 and 5 years, central nervous system tuberculosis, and impaired consciousness. There are conflicting data regarding the age at the onset of stroke and long-term outcomes in pediatric stroke patients.[8] In a large observational study of 587 children, Felling *et al.* reported that age between 28 days and 1 year and between 1 and 4 years were associated with increased odds of a poor outcome 2 years after stroke, according to univariate analysis.[21] The higher PSOM score in our study in the 2- to 5-year age group than in the other age groups is likely due to the underlying etiology. All the children with mineralizing angiopathy were younger than 2 years (range 9–16 months), which likely contributed to overall better outcomes in children younger than 2 years. Children with central nervous system (CNS) tuberculosis, in addition to stroke, experience greater disability due to additional neurological damage caused by factors such as hydrocephalus, tuberculomas, and arachnoiditis. Impaired consciousness at the time of stroke signifies more extensive or bilateral involvement and, understandably, is associated with greater disability, which is concordant with the findings of previous studies (18). Similarly, bilateral hemispheric involvement was associated with a higher PSOM score at 6 months, but the difference was not significant. A significantly higher PSOM score in children with anemia at the onset of stroke was not true at 6 months after stroke (0.149).

Stroke is generally not included as a diagnostic or management priority in children with neurotuberculosis or other infectious central nervous system diseases, and guidelines primarily focus on treating infection. However, as apparent from the observations in this study, the outcome is also influenced by the incidence of stroke in these infectious etiologies, as highlighted by higher PSOM scores in patients with tuberculosis. This clearly emphasizes that the management of acute stroke should be a part of the guidelines for the diagnosis and treatment of infective etiologies, such as neurotuberculosis, to improve long-term neurologic outcomes and reduce disability. There is evidence that antiplatelet therapy in the form of aspirin has a role in reducing new infarcts in CNS tuberculosis.[22] Given

Table 2: Comparison of the PSOM score at the time of acute stroke and at 6 months between the variables affecting the outcome.

Variables	Mean total PSOM score (SD) at the time of stroke, *n*=55	*P*-value*	Mean total PSOM score (SD) 6 months after stroke, *n*=44	*P*-value*
Age of onset of stroke	< 6 months (*n*-9): 1.38 (1.24)	0.001	< 6 months (*n*-9): 1.05 (1.13)	0.005
	6–24 months (*n*-26): 2.21 (1.99)		6–24 months (*n*-17): 1.0 (1.42)	
	2–5 years (*n*-8): 5.5 (3.02)		2–5 years (*n*-7): 2.5 (1.89)	
	5–10 years (*n*-8): 2 (1.60)		5–10 years (*n*-7): 0.57 (0.44)	
	>10 years (*n*-4): 1.37 (1.70)		>10 years (*n*-4): 0.50 (0.57)	
Etiology	Mineralizing angiopathy (*n*-15): 1.53 (1.17)	<0.001	Mineralizing angiopathy (*n*-12): 0.5 (0.42)	0.007
	Central nervous system tuberculosis (*n*-7):5.5 (2.6)		Central nervous system tuberculosis (*n*-3): 2.33 (2.3)	
	Other etiologies (*n*-33): 2.2 (2.6)		Other etiologies (*n*-29): 1.26 (0.91)	
Gender	Male (*n*-33): 2.04 (1.80)	0.2	Male (*n*-27): 0.94 (1.31)	0.249
	Female (*n*-22): 3.09 (2.88)		Female (*n*-17): 1.44 (1.49)	
Type of stroke	Acute ischemic stroke (*n*-41): 2.71 (2.50)	0.422	Acute ischemic stroke (*n*-31): 1.11 (1.38)	0.135
	Hemorrhagic (*n*-7): 1.28 (1.38)		Hemorrhagic (*n*-7): 0.64 (0.55)	
	CSVT (*n*-3): 2.83 (2.46)		CSVT (*n*-3): 2.83 (2.46)	
	CSVT with hemorrhagic transformation (*n*-4): 1.62 (1.10)		CSVT with hemorrhagic transformation (*n*-3): 0.83 (0.76)	
Time of presentation to hospital	Before 6 h (*n*-5): 1.7 (1.09)	0.29	Before 6 h (*n*-4): 0.87 (0.85)	0.08
	6–24 h (*n*-28): 2.57 (2.13)		6–24 h (*n*-23): 0.73 (0.79)	
	After 24 h (*n*-22): 3.38 (2.94)		After 24 h (*n*-17): 1.7 (1.89)	
Circulation involved	Anterior (*n*-32): 1.89 (1.81)	0.04	Anterior (*n*-25): 0.98 (1.41)	0.6
	Posterior (*n*-3): 1.16 (2.02)		Posterior (*n*-3): 0.33 (0.57)	
	Both anterior and posterior (*n*-18): 3.50 (2.90)		Both anterior and posterior (*n*-14): 1.14 (1.0)	
Hemisphere involved	Left (*n*-14): 1.96 (2.25)	0.003	Left (*n*-11): 1.45 (1.70)	0.18
	Right (*n*-20): 1.45 (1.19)		Right (*n*-17): 0.64 (0.60)	
	Both (*n*-21): 3.76 (2.66)		Both (*n*-16): 1.43 (1.67)	
Seizure	Generalized (*n*-25): 2.94 (2.53)	0.134	Generalized (*n*-18): 1.33 (1.44)	0.30
	Focal (*n*-9): 3 (3.18)		Focal (*n*-9): 1.50 (1.78)	
	No seizure (*n*-21): 1.66 (1.36)		No seizure (*n*-17): 0.73 (1.06)	
Level of consciousness	Intact (*n*-27): 1.46 (1.27)	0.001	Intact (*n*-22): 0.70 (0.99)	0.03
	Impaired (*n*-28): 3.42 (2.71)		Impaired (*n*-22): 1.56 (1.61)	
Anemia	No anemia (*n*-13): 2.07 (1.46)	0.03	No anemia (*n*-11): 0.68 (0.60)	0.149
	Mild-moderate anemia (*n*-33): 2.46 (2.40)		Mild-moderate anemia (*n*-26): 1.19 (1.47)	
	Severe anemia (*n*-4): 5.50 (3.24)		Severe anemia (*n*-2): 2.75 (3.18)	
Ferritin level	Normal (*n*-24): 2.27 (2.36)	0.715	Normal (*n*-20): 1.15 (1.61)	0.55
	Low ferritin (*n*-7): 1.92 (1.51)		Low ferritin (*n*-6): 0.75 (0.27)	

*Independent samples *t*-test was used for the comparison of means between two groups and one-way ANOVA was used comparison of means between more than two groups. PSOM: Pediatric stroke outcome measure, SD: Standard deviation, CSVT: Cerebral sinovenous thrombosis

that tuberculosis leads to greater disability, the addition of antiplatelet therapy to standard CNS tuberculosis treatment should be considered and further studied in clinical trials. The risk factors for greater disability identified from our cohort can help anticipate and subsequently identify children who will need more intensive rehabilitation.

Seventy percent of the children enrolled in the current study were from rural areas of western Rajasthan, which is difficult terrain, and financial constraints likely led to the loss of follow-up of 11 children out of 55. Another limitation is the limited follow-up of 6 months. The observations made in this study would have been more significant if a longer follow-

up (2 years and above) could have been made. In resource-limited settings, longer follow-up is challenging because of continuing disabilities, financial hurdles, lack of awareness, and difficulty in transportation.[23] However, there is evidence that the PSOM score does not change significantly 6 months after stroke onset.[24] Compared with those in adults, there are few cases in specific subgroups, which is likely due to the rarity of stroke in children. The findings of this study need confirmation in a more extensive cohort study. The PSOM focuses mainly on sensorimotor impairments.[12] Thus, the use of the PSOM as a measure of disability likely results in overlooking other important domains, such as emotion and behavior, which are also impaired in children who have suffered stroke. In addition, children with poorer outcomes are more likely to be lost to follow-up; thus, the actual disability may be underestimated in this study.

CONCLUSION

Children with stroke aged 2–5 years, underlying neurotuberculosis, extensive bilateral involvement, impaired consciousness at the time of presentation, and severe anemia are slower to recover and have greater neurologic disability, as determined by the PSOM at 6 months. Mineralizing angiopathy is a major contributor to stroke in young children and has a good prognosis. The presence of these risk factors associated with greater disability may improve patient prognosis and should prompt the initiation of extensive rehabilitation to achieve optimal outcomes.

Ethical approval: The research/study was approved by the Institutional Review Board at Institutional Ethics Committee, Dr. Sampurnanad Medical College, Jodhpur, number SNMC/IEC/IIP/2024/Plan/084, dated June 14, 2024.

Declaration of patient consent: The authors certify that they have obtained all appropriate patient consent.

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