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Seizures as a presenting symptom of acute arterial ischemic stroke in childhood

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Abstract

Objectives—To define incidence of seizures as a presenting symptom of acute arterial ischemic stroke (AIS) in children and to determine whether younger age, infarct location, or AIS etiology were risk factors for seizure at AIS presentation.

Study design—Children aged 2 months to 18 years presenting with AIS from January 2005 to December 2008 were identified from a single center prospective pediatric stroke registry. Clinical data were abstracted, and a neuroradiologist reviewed imaging studies.

Results—Among 60 children who met inclusion criteria, seizures occurred at stroke presentation in 13 (22%). Median age was significantly younger in children who presented with seizures than in those who did not (1.1 versus 10 years, p=0.0009). Seizures were accompanied by hemiparesis in all patients. Three of four children with clinically overt seizures at presentation also had non-convulsive seizures on continuous EEG monitoring.

Conclusions—About one-fifth of children with acute AIS present with seizures. Seizures were always accompanied by focal neurologic deficits. Younger age was a risk factor for seizures at presentation. Seizure at presentation was not associated with infarct location or etiology. Non-convulsive seizures may occur during the acute period.

Keywords

EEG; non-convulsive seizures; hemiparesis

Recent reports have elucidated many mechanisms and risk factors for arterial ischemic stroke (AIS) in children (1–3), but few have investigated the presenting features (4). Several recent series have reported that there is often delayed recognition of pediatric stroke (5–8). Improved understanding of the presenting symptoms and signs of AIS may reduce the time from symptom onset to the time of definitive diagnosis (5), which, in turn, may improve the

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physician's ability to intervene acutely and thereby to improve outcome. Previous studies have reported that 19–44% of children with AIS present with seizures (9–12). However, no detailed studies in children with acute AIS have investigated the clinical and radiological risk factors associated with seizures at stroke onset. In a retrospective analysis of a prospectively identified cohort of children with AIS, we aimed to define the incidence of seizures as a presenting symptom of acute AIS in children and identify risk factors for seizures at stroke onset.

METHODS

Children age 2 months to 18 years with AIS receiving acute stroke care at a single tertiary care children's hospital between January 2005 and December 2008 were prospectively enrolled in a stroke registry. Acute AIS was defined as an acute neurological deficit of any duration consistent with focal brain ischemia conforming to an arterial distribution, with neuroimaging confirmation of acute infarction in an arterial territory corresponding to the clinical deficit. Infants age < 2 months, children with infarction secondary to cerebral sinus venous thrombosis, and children with ischemic stroke secondary to spontaneous intracerebral hemorrhage were excluded. Seizure at presentation was defined as any clinically evident seizure observed by a parent/guardian or medical provider occurring as the heralding sign of the stroke or within 1 hour of the onset of other stroke symptoms. Patients who had seizures at presentation of their stroke, or had seizures during the acute hospitalization were identified from our stroke registry database. Additional retrospective review of clinical charts confirmed seizure occurrence, timing, and semiology.

Diagnostic evaluations including routine and continuous electroencephalograms (EEGs) were performed as clinically indicated. At the discretion of the clinical team, patients with seizures generally received a standard EEG, and those with persistently altered mental status underwent continuous EEG monitoring. All neurophysiology reports were reviewed to identify EEG abnormalities and non-convulsive seizures. Per institutional protocol most patients with suspected AIS undergo brain and vascular imaging with magnetic resonance imaging (MRI), echocardiogram, and thrombophilia studies.

A board-certified pediatric neuroradiologist (AV) blinded to clinical information and initial radiological interpretation reviewed all neuroimaging and determined the infarct location according to the following categories: 1) vascular distribution - anterior circulation, posterior circulation or both, 2) supratentorial, infratentorial or both, and 3) cortical, subcortical or both, for supratentorial infarcts. Stroke risk factors included arteriopathy, cardiac conditions, tumor-related, meningitis, isolated thrombophilia (without additional risk factors), sickle cell anemia, or cryptogenic. Arteriopathy was classified according to the method of Sebire et al, and included arterial dissection, intracranial arteriopathy, moyamoya disease, and autoimmune vasculitis (13,14). When no stroke risk factor was identified after comprehensive evaluation, cases were classified as cryptogenic.

Statistical analysis was performed using STATA 10.0 (Stata Corp, College Station, TX). Differences in demographic characteristics (age, sex, race) between patients presenting with and without seizures were compared using Fisher exact test for dichotomous variables and Mann Whitney rank sum tests for non-normally distributed continuous variables. Univariate logistic regression was used to compare the independent associations of age, cortical involvement of stroke, and supratentorial location with seizure at presentation. Mann Whitney rank sum tests were used to compare length of intensive care unit stay and length of hospital stay between children with seizures and children without seizures at presentation. A p-value of less than 0.05 was considered statistically significant. This study was reviewed and approved by the Institutional Review Board.

RESULTS

Sixty infants and children (40 males) with acute AIS were enrolled. The median age at presentation was 5.7 years [interquartile range (IQR) 1.4–13.1 years]. Forty-three children where white [72% (39 non-Hispanic, 4 Hispanic)], 14 were black (23%), and 3 (5%) were classified as other. Seizures occurred at initial presentation in 13 children [22%; 95% binomial exact confidence interval (CI) 12–34%]. All children with a seizure on presentation had additional focal neurologic deficits. Clinical signs at presentation in the 13 children with seizures included hemiparesis in 13 (100%), mental status change in 5 (38%), visual deficit in 3 (23%), dysarthria or aphasia in 2 (15%), and ataxia in 1 (8%). In all children with seizures at presentation, the seizure was the heralding sign of stroke onset, preceding the appearance of other focal neurologic symptoms and signs. In all children with seizures, the hemiparesis persisted for >24 hrs after onset of the seizure. Additionally, no child presenting with seizure had a history of prior seizures.

The Table summarizes the analysis of association between seizures at presentation and patient demographics, stroke risk factors, and infarct location. Children with seizure at initial presentation were younger (median 1.1 years; IQR 0.7–2.8 years) than children without seizure at initial presentation (median 10 years; IQR 2.3–13.8 years; $p = 0.0009$). Using logistic regression, for every additional year of age, the odds of presenting with seizure was 0.78 (95% CI 0.65–0.94, $p=0.007$). Because the upper limit of the interquartile range for age in the subjects who seized was 2.8 years, we used 3 years of age as a cutoff for binary comparison. The relative risk of AIS presenting with a seizure in children younger than 3 years compared with those older than 3 years was 7.7 (95% CI 1.9–31.7; $p=0.0008$). There were no statistically significant differences in sex, race, stroke risk factor, or infarct location in those with seizures compared with those without seizures.

Seizures had clear focal features in 10/13 children (lateralized tonic-clonic in 7, eye and head version without tonic-clonic movement in 3) and had no focal features in 3/13 children. The semiology of seizures with lateralizing signs was concordant with the infarcted hemisphere. Multiple seizures in close succession occurred in 8 children, and single seizures occurred in 5 children. One patient had focal status epilepticus (45 minute focal clonic seizure).

Eleven of the 13 children presenting with seizure had an EEG within the initial 48 hours of presentation, including continuous EEG monitoring in 4 children. Of the 4 children who had EEG monitoring, 3 (23% of the children presenting with seizures) had subsequent electrographic seizures recorded. These electrographic seizures were detected on routine EEG in 1 child and on continuous EEG in 2 children. The child with electrographic seizure on routine EEG subsequently underwent continuous EEG monitoring. Among the 3 children with electrographic seizures, one had both clinical and non-convulsive seizures and two had only non-convulsive seizures. The seizures were frequent in two of these children constituting focal electrographic status epilepticus. Of the 3 children with non-convulsive seizures, only 1 was receiving paralytics (for underlying medical condition and not specifically for stroke management). Seizure localization and infarct locations were concordant in all three children. Four children had focal inter-ictal epileptiform discharges recorded, including 2 with electrographic seizures and 2 without recorded electrographic seizures, and these were concordant with infarct location. Non-specific background abnormalities including slowing, attenuation, and disorganization were severe in 8 children. Of the 47 children who did not present with seizures, 13 had routine EEGs and 4 had continuous EEG monitoring, and no non-convulsive seizures were identified. Two children had focal inter-ictal epileptiform discharges recorded, including periodic lateralized epileptiform discharges in one, and these were concordant with infarct location. EEGs were

normal in 3. Non-specific background abnormalities were present in 14 and included focal slowing in 9, diffuse slowing in 3, and focal and diffuse slowing in 2. Two subjects without seizures on presentation had clinically evident seizures during hospitalization. Both had a history of epilepsy including one with cryptogenic infantile spasms and one with partial epilepsy related to a brain tumor.

The median length of hospitalization was 13 days (IQR 7–33 days) in those with seizure at presentation and 8 days (IQR 5–15 days) in those without seizure at presentation (p=0.09). The median length of intensive care unit stay was 13 days (IQR 4–20 days) in those with seizure at presentation compared with 5 days (IQR 2–9 days) in those without seizure at presentation (p=0.07). Three children died during the acute stroke hospitalization, none of whom presented with seizures. Two deaths were attributable to complex congenital heart disease complications. One death occurred in a child with a brain tumor, who developed extensive swelling leading to progressive infarction and eventual herniation.

DISCUSSION

Twenty-two percent of children with acute AIS presented with acute symptomatic seizures. All of these children presented with focal neurologic deficits in addition to seizure. These findings indicate that stroke should be considered in children presenting with new-onset seizures in combination with focal neurological deficits. Additionally, even without the utilization of standardized continuous EEG monitoring, 23% of those with clinically evident seizures also had non-convulsive seizures detected on EEG. For patients presenting with altered mental status and acute ischemic stroke, EEG monitoring to evaluate for non-convulsive seizures may be indicated in some children.

Our finding that seizures occur at stroke presentation in 22% of children with AIS is consistent with prior reports. Prior prospective (12) and retrospective (9–11) studies that included only AIS or allowed separation of children with AIS from the full cohort reported seizures in 19–44% of children at presentation (9–12) or within the initial one day to two weeks (15). All patients with seizures in our study had associated hemiplegia, consistent with a prior study (15). No child in our study had an isolated seizure without associated focal neurologic deficits. Studies that have included children with AIS, intracerebral hemorrhage, and sinovenous thrombosis have reported seizures in 27–58% at presentation (16–19) or within the initial several weeks (20,21). This is consistent with a large number of adult stroke studies that report early seizures (22), variably defined as within the first 24 hours or several weeks, as occurring in 2–33% (23–29), with the majority occurring within the first 24 hours (30–32).

This was an exploratory study designed to identify possible risk factors for seizures on stroke presentation including age, infarct location and AIS etiology, which could be evaluated in larger multi-center studies, thereby elucidating which children presenting with seizures require a stroke evaluation. Although the present study was not powered to detect small differences in the occurrence of risk factors, we did find that the risk of seizures at presentation of AIS was 7.7 times greater in children under the age of 3 compared with older children in this study. Prior studies have had inconsistent results with respect to age as a risk factor for seizure at AIS presentation. One study of 76 children with ischemic stroke aged 44 weeks to 19 years reported that children less than 1 year of age were significantly more likely to present with seizure (33). In contrast, a study of 48 children with ischemic and hemorrhagic stroke reported that children older than 1 year of age were more likely to present with seizures (19). Developmentally determined differences in neurotransmitter receptor composition, number, and distribution favor a state of enhanced excitability in immature versus mature brain (34). These differences may facilitate seizure propagation in

younger versus older children or adults in the setting of acute brain injury such as ischemic infarction. This may explain why the rate of acute symptomatic seizures was higher in the youngest children in our cohort, and why children are more likely to have seizures at stroke onset than adults.

Our data show no association between seizures at presentation and infarct location. Although seizures are often considered cortically based, and cortical infarcts have been identified as a risk factor for recurrent seizures (21), in our cohort several children with isolated subcortical infarcts had seizure on presentation. This observation is consistent with prior reports in children (21) and adults (19,24,35). Prior studies have reported that presence of seizures at presentation predicted involvement of the anterior circulation and cortical structures (9,15,17,35). Studies in adults have demonstrated that additional lesions may be present as well as the most prominent subcortical lesion, and this more widespread disease might be responsible for seizures (36,37). Similarly, in our study seizures at presentation were not associated with stroke etiology. This is consistent with a prior pediatric study that demonstrated no difference in presentation (including seizures) in children with arteriopathic and non-arteriopathic stroke (2). In contrast, one study reported an association between infectious etiology and early seizures (15). However, because the etiologies of pediatric AIS are heterogeneous, our study did not have had adequate power to detect a difference in seizure presentation amongst the various stroke etiologies.

If seizures did not occur as part of the initial presentation, they were rare later during the acute hospitalization in our study. Only two of 47 children who did not present with seizures had clinically evident seizures later during the acute hospitalization, and both had a history of epilepsy. Thus, if seizures do not occur on presentation in children with AIS, prophylactic anticonvulsants may not be warranted. Adult stroke patients tend to have early seizures in the initial several hours after stroke onset, leading to speculation that progressive ischemia of the penumbra may reduce epileptogenic activity and thus reduce seizures after the initial several hours (30).

A recent study of children (including neonates) with acute neurological syndromes that mimicked stroke demonstrated that all children presenting with seizure had a serious neurological disorder such as brain tumor, acute demyelinating encephalomyelitis, or intracranial infection (4). This finding, coupled with our data that seizures may be a presenting symptom of AIS, suggests that children with new onset seizures and focal neurologic deficits (especially hemiparesis) may require prompt neuroimaging as part of their evaluation. This is consistent with the American Academy of Neurology practice recommendation focused on evaluating a first nonfebrile seizure in children, which includes that “emergent neuroimaging should be performed in a child of any age who exhibits a post-ictal focal deficit not resolving quickly” (38). The recognition that nearly one quarter of children with acute AIS present with seizures accompanied by focal deficits could facilitate more rapid diagnosis of stroke. More rapid institution of early management strategies such as initiation of intravenous fluids and antithrombotics, as well as identification and management of underlying risk factors, could potentially improve outcome and reduce the risk of recurrent stroke. Additional studies are needed to determine what proportion of children presenting with seizure and paralysis have an acute lesion requiring urgent management such as stroke, and to define clinical features that distinguish children with stroke from those with other causes of new onset seizure with focal deficit.

The effect of seizures at presentation on the clinical course of patients with AIS is unclear. Our data demonstrate a trend toward longer durations of ICU stay and hospitalization, but an assessment of other measures of short-term and long-term outcome was beyond the scope of this study. Higher mortality has been reported in adults with early seizures versus without

(33% versus 14%) (28), and persistent motor seizures are associated with worsening neurologic deficits (39). Studies of neonatal (40) and pediatric stroke (9,18) have reported an association between convulsions at presentation and more severe outcome.

Although the primary aim of this study was to investigate seizures on presentation, we noted that continuous EEG monitoring was performed in some patients due to persistent altered mental status and clinical suspicion of ongoing seizures. Four children with clinically evident seizures underwent subsequent EEG monitoring and 3 had non-convulsive seizures identified, including 2 with non-convulsive status epilepticus. Stroke is a known cause for non-convulsive seizures (41) and non-convulsive status epilepticus in children (42,43) and adults (44–46). Non-convulsive seizures have been associated with elevations in intracranial pressure and metabolic stress in adults with acute brain injury (47), and our observations suggest that non-convulsive seizures may represent an under-recognized and potentially important cause of both short-term and long-term neurologic consequences in children with acute ischemic stroke. Recent stroke management guidelines for neonates and children suggest that continuous EEG monitoring be considered for individuals with cerebral sinovenous thrombosis who are unconscious (48). Our data suggest that continuous EEG monitoring also should be considered in children with AIS with impairment of consciousness. Although it is conceivable that improved detection and treatment of non-convulsive seizures could lead to improved stroke outcome, our study did not assess this as the sample size was small and the institution of EEG monitoring was not protocol driven. The incidence of non-convulsive seizures in children with stroke needs to be evaluated in well-designed prospective clinical trials.

Optimizing care of children with AIS involves specifically addressing acute and chronic comorbidities, such as seizures and epilepsy. Thus, additional studies are needed to define the risk of seizure recurrence and development of epilepsy, to determine the optimal use of anticonvulsant medications, and to determine the role of continuous EEG monitoring to detect non-convulsive seizures in children with AIS. In addition, further characterization of the relationship between AIS, acute seizures, and epilepsy may yield useful insights into the process of epileptogenesis after acquired brain injury in children.

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Table 1

Risk Factors for Seizures

Variable (N)	Seizure N (%)	RR (95% exact CI)	p-value
Age			
≤ 3 years (25)	11 (44%)	7.7 (1.9–31.7)	0.0009
>3 years (35)	2 (6%)	(reference)	
Sex			
Male (40)	7 (16%)	0.6 (0.2–1.5)	0.33
Female (20)	6 (30%)	(reference)	
Race/Ethnicity			
White non-Hispanic (39)	8 (21%)	0.9 (0.3–2.3)	0.94
Other (21)	5 (24%)	(reference)	
Primary Stroke Risk Factor			
Cardiac (15)	5 (33%)	1.9 (0.7–4.9)	0.28
Arteriopathy (18)*	4 (22%)	1.0 (0.4–2.9)	1.00
Meningitis (4)	2 (50%)	2.6 (0.8–7.8)	0.20
Brain tumor (8)	0 (0%)	0 (…)	0.18
Sickle cell anemia (2)	0 (0%)	0 (…)	1.00
Isolated thrombophilia (3)	0 (0%)	0 (…)	1.00
Cryptogenic (10)	2 (20%)	0.9 (0.2–3.5)	1.00
Infarct Location			
Supratentorial (54)	13 (24%)	--	0.32
Infratentorial (6)	0 (0%)	(reference)	
Infarct Arterial Distribution			
Anterior circulation (46)	12 (26%)	3.7 (0.5–25.7)	0.26
Posterior circulation (14)	1 (7%)	(reference)	
Infarct Location			
Cortical involvement (42)	10 (24%)	1.4 (0.4–4.6)	0.74
No cortical involvement (18)	3 (17%)	(reference)	

N, number of subjects; RR, relative risk; CI confidence interval

± Relative risk reflects comparison of each individual mechanism to all others mechanisms

*Arteriopathy: 6 moyamoya, 6 intracranial arteriopathy, 4 vertebral dissection, 1 carotid dissection, 1 vasculitis