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Mimics of Childhood Stroke: Characteristics of a Prospective Cohort

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ABSTRACT

BACKGROUND. Little is known about the clinical features and spectrum of diagnoses in children with "stroke mimics," those with acute neurologic deficits but without cerebrovascular diseases.

OBJECTIVES. Our goal was to describe patients with stroke mimics and to determine if clinical features predict benign diagnoses.

METHODS. Our stroke consult team registered a prospective consecutive cohort of 143 patients with acute presentations suspicious for cerebrovascular disease from November 2003 to November 2004. Cases in which stroke was ruled out (stroke mimics) were reviewed for clinical features and diagnostic test results and were classified "benign" if there was no structural brain lesion and there was an expectation of complete recovery.

RESULTS. Of the 143 cases evaluated for suspected stroke, 30 (21%) had stroke mimics. Presenting signs included seizure (n = 11), headache (n = 9), mental status change (n = 6), focal weakness (n = 14), and focal sensory change (n = 7). Eleven patients had "benign" diagnoses (3 migraine, 3 psychogenic diagnoses, 3 musculoskeletal abnormalities, 1 delirium, and 1 episodic vital sign changes). Nineteen patients had "not-benign" diagnoses (3 reversible posterior leukoencephalopathy syndrome, 3 neonatal seizures, 2 vascular anomalies, 2 inflammatory disease, 2 intracranial infection, 2 epilepsy, 2 metabolic stroke, 1 tumor, 1 drug toxicity, and 1 idiopathic intracranial hypertension). Except for the presence of seizures, there were no significant differences in presentation or risk factors between benign and not-benign cases.

CONCLUSIONS. Many disorders mimic childhood stroke. History and clinical presentation often do not distinguish the one third of patients with benign disorders from the two thirds with more serious problems, necessitating timely comprehensive investigations, especially brain MRI.

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Key Words
stroke, differential diagnosis, neuroimaging, hemiparesis

Abbreviations
AIS—arterial ischemic stroke
SVT—sinovenous thrombosis
ICH—intracranial hemorrhage
TIA—transient ischemic attack
ADEM—acute disseminated encephalomyelitis
CT— computed tomography
RPLS—reversible posterior leukoencephalopathy syndrome
AVM—arteriovenous malformation
t-PA—tissue plasminogen activator

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A CHILD WHO presents to the pediatrician's office or emergency department with an acute neurologic deficit poses a diagnostic challenge. Although stroke is one cause of acute neurologic deficits, it is a relatively rare condition, with a reported incidence of 2.1 to 13.1 per 100 000 children per year.[1–5] Acute neurologic symptoms are frequently attributed to more common alternative diagnoses such as seizure and migraine. Although clinical teaching is that disorders that resemble stroke ("stroke mimics") are more common than strokes in children, the presenting signs and symptoms and differential diagnosis of pediatric stroke mimics have not been described in the literature.

Our purpose with this study was to characterize the clinical and radiographic characteristics of a referral-based prospective cohort of children with acute neurologic symptoms mimicking stroke. We hoped to identify features of the history or physical examination that were associated with a benign diagnosis and to determine the role of neuroimaging in making a definitive clinical diagnosis.

METHODS

Patients

Subjects were a consecutive cohort of patients referred for evaluation of suspected acute stroke syndromes to a dedicated stroke team at a single tertiary care children's hospital (Children's Hospital of Philadelphia) between November 1, 2003, and November 1, 2004. Patients with acute neurologic deficits for whom no stroke-team consultation was requested could not be included in the cohort. Cases were identified from a log of consultation requests maintained by the stroke team. Referrals to the stroke team are made directly from the primary medical/surgical teams and indirectly from the general child neurology consult service after triage by the on-call neurology resident. The stroke team provides specific guidelines for telephone triage to the neurology residents. The stroke team consists of 3 attending child neurologists (S.E.S., D.J.L., R.I.) who are available by pager at all times and who coordinate the services of a multidisciplinary team through consensus-based treatment protocols for all major stroke subtypes in children. The protocols specify prompt neuroimaging, comprehensive stroke risk-factor evaluation, and clinical and laboratory assessment.[6,7] The protocols define arterial ischemic stroke (AIS) as an acute clinical syndrome with a neurologic deficit referable to a cerebral arterial territory and a brain MRI showing a corresponding area of acute infarct. Diagnoses of cerebral sinovenous thrombosis (SVT) and intracranial hemorrhage (ICH) are confirmed by MRI and/or magnetic resonance venogram in children with acute neurologic syndromes attributable to their radiologic lesion. We define a transient ischemic attack (TIA) as a transient neurologic deficit referable to a cerebral arterial territory in a patient whose MRI shows no acute ischemia but whose history and other workup strongly suggest cerebrovascular disease (eg, a child with transient focal weakness conforming to the middle cerebral artery distribution whose imaging shows a carotid dissection but no infarction).

The following criteria were used for inclusion in this study: (1) aged newborn to 18 years; (2) acute neurologic syndrome prompting a request by the primary providers for consultation from the stroke team for suspected AIS, SVT, TIA, or ICH; and (3) final diagnosis for the neurologic complaint being a disorder other than AIS, SVT, ICH, or TIA.

Data Collection

Review of the medical chart was conducted by one of the investigators (R.A.S.) using a standardized data-collection instrument to record gender, age at the time of consultation, reason for stroke-team consultation, preexisting risk factors for stroke, precipitating factors for a neurologic event, medications, reason for hospitalization, clinical history, nature of acute neurologic event, physical examination findings, results of neuroimaging and other diagnostic studies, clinical diagnosis, and treatment. Final diagnosis was extracted from the medical chart as determined by members of the stroke team in consultation with the primary clinical service caring for the patient during the hospital admission. This study was approved by the institutional review board at the Children's Hospital of Philadelphia.

Analysis

After review of clinical and diagnostic data, the final diagnosis for each subject was classified as "benign" or "not benign." The diagnosis was considered benign if there was no clinically significant structural abnormality on neuroimaging and there was an expectation of complete recovery. Diagnoses were considered not benign if there was a clinically significant structural abnormality on neuroimaging or the disorder required long-term treatment or was associated with a risk of adverse long-term functional outcome. Continuous variables in the benign versus not-benign groups were compared by using Student's t test (Excel; Microsoft, Redmond, WA). Categorical variables in the benign versus not-benign groups were compared by using Fisher's exact test (SigmaStat 3.01).[8]

RESULTS

Demographics

Of 143 children evaluated by the stroke team during the enrollment period, 30 patients (21%; 16 boys, 14 girls) were classified as stroke mimics. Of these 30 patients, 11 (37%) had benign diagnoses, and 19 patients (63%) had nonbenign diagnoses (Table 1). The mean age of the

TABLE 1 **Final Diagnoses**

Diagnosis	No. of Patients
Benign	11
Migraine	3
Psychogenic	3
Musculoskeletal abnormalities	3
Delirium	1
Periodic hypertensive episodes	1
Not benign	19
Neonatal seizure	3
RPLS	3
Metabolic stroke	2
Epilepsy (new diagnosis)	1
Postictal paralysis	1
ADEM	1
Tumor	1
Cerebellitis	1
Drug toxicity	1
Idiopathic intracranial hypertension	1
Subdural empyema	1
AVM	1
Moyamoya	1
Intracranial abscess	1

stroke-mimic cohort was 10.5 ± 1.1 years. Mean age of the patients with benign diagnoses was not different from those with nonbenign diagnoses. More girls ($n = 9$) than boys ($n = 2$) had benign diagnoses ($P = .046$).

Signs and Symptoms at Presentation

The presenting signs and symptoms are described in Table 2. The number of patients presenting with a single symptom (4 benign, 7 not benign), compared with those with >1 neurologic symptom (7 benign, 12 not benign), was not different between the 2 groups. Presentation with exclusively subjective complaints of numbness/tingling, dizziness without ataxia, headache, or nonspecific blurred vision was equal in the 2 groups (2 benign, 2 not benign). The presence of focal, objective, neurologic signs on initial examination was not different between the 2 groups (9 benign, 13 not benign).

Seizure at presentation always indicated a nonbenign diagnosis and was common in this group, affecting 11 (58%) of 19 cases (vs 0 of 11 patients with benign diagnoses; $P = .002$). Eleven patients (37%) had seizures as part of their presentation, only 2 of whom had previously been diagnosed with epilepsy. One patient with known epilepsy and Sturge-Weber syndrome presented with prolonged (>9 hours) postictal paralysis, and 1 patient had known epilepsy but a new diagnosis of moyamoya disease. The 9 patients who presented with new-onset seizure were either neonates (3) or had additional presenting signs or symptoms (3 weakness and/or numbness, 4 headache, 1 change in mental status).

Headache was a presenting symptom for 9 patients (30%) and the sole symptom of 1 patient. This patient developed altered mental status and was ultimately diagnosed with acute disseminated encephalomyelitis (ADEM). Headache was associated with additional signs or symptoms in the remaining 8 patients (4 altered mental status, 3 focal numbness and/or weakness, 2 seizure, and 2 ataxia).

TABLE 2 **Presenting Signs and Symptoms**

Sign/Symptom	Total No.	Benign	Not Benign
Focal weakness	14	6	8
Seizure	11	0	11
Headache	9	3	6
Focal numbness or tingling	7	6	1
Altered mental status	6	2	4
Vision changes	5	2	3
Ataxia	2	0	2
Isolated change in vital signs (episodic hypertension)	1	1	0

Stroke Risk Factors

Request for stroke-team consultation was sometimes motivated by the presence of a known stroke risk factor in the patient or a family history suggestive of stroke or vascular disease. Of the 30 patients, 13 (43%) had preexisting risk factors or strong family histories for stroke or vascular disease, including congenital heart disease, premorbid hypertension, coagulopathy, rheumatologic disease, or genetic or metabolic syndromes or family history of early stroke (not necessarily in childhood), myocardial infarction, or coagulopathy.

Neuroimaging

All patients underwent neuroimaging with computed tomography (CT) and/or MRI. The initial study was CT in 18 patients (60%), of which 11 were abnormal. Among the 7 patients with normal CT, 3 had benign diagnoses (2 musculoskeletal, 1 psychogenic). Three patients with normal CT underwent brain MRI for clinically suspected conditions, which was abnormal in 1 case, showing a Chiari 1 anomaly in a patient with migraine.

Among the 11 patients with abnormal initial head CT, 1 patient had findings consistent with a known diagnosis of Sturge-Weber syndrome, and 10 underwent MRI, all of which were abnormal. Five of these patients had hypodensities on CT that were insufficiently specific to distinguish AIS from other diagnoses. MRI in these cases defined diagnoses requiring specific treatment, including reversible posterior leukoencephalopathy syndrome (RPLS) in 2, metabolic disorder in 2, and tumor in 1 case. In 2 subjects whose initial CT was suspicious for SVT, MRI ruled out SVT and confirmed other diagnoses (cerebellitis and hypoxic-ischemic encephalopathy).

Brain MRI was the initial study performed in 11 patients, of which only 3 were normal. Among all 25

patients who underwent brain MRI, 7 (28%) were normal and 18 (72%) were abnormal. Specific management changes resulted from 11 (61%) of the 18 abnormal MRIs. Interventions included management of hypertension and identification of renal disease in patients with RPLS ($n = 3$), steroid pulse for ADEM ($n = 1$), medical management of metabolic abnormalities for metabolic disorders ($n = 2$), and neurosurgical intervention ($n = 5$) for left parietal tumor, left temporal arteriovenous malformation (AVM), subdural empyema, bifrontal abscesses, and moyamoya disease. In 1 patient, the stroke team's clinical evaluation suggested a lumbar spine localization. She therefore underwent spine MRI, which was normal, and her diagnosis was psychogenic weakness.

Final Diagnoses

Eleven patients were given benign diagnoses, and 19 had nonbenign diagnoses (Table 1). Seizure at presentation always indicated a nonbenign diagnosis, as discussed previously. Three patients with seizures had a final diagnosis of acute symptomatic neonatal seizure, including 1 with hypoplastic left heart syndrome, 1 with hypoxic ischemic encephalopathy, and 1 with presumed sepsis. One patient had a new diagnosis of unclassified epilepsy, and 1 had known partial epilepsy and a new diagnosis of moyamoya disease. One patient had known Sturge-Weber syndrome and a prolonged postictal paralysis. The other patients' seizures were provoked by their underlying diagnoses (3 RPLS, 1 tumor, 1 intracranial abscess).

Nine patients presented with headache, 3 of whom had migraine, a benign diagnosis. The other 6 were classified as not benign (2 RPLS, 1 idiopathic intracranial hypertension, 1 tumor, 1 cerebellitis, 1 ADEM).

One patient each had AVM and moyamoya as their final diagnoses. Although these cerebrovascular abnormalities can precipitate AIS or ICH, these patients had no evidence of ischemia or hemorrhage on MRI, and their presenting signs and symptoms were not referable to a specific cerebrovascular territory, so they were classified as stroke mimics rather than TIA.

Two patients had presumed metabolic strokes. One was a 40-month-old boy with cytochrome c oxidase deficiency who presented with seizures in the setting of respiratory failure resulting from RSV infection. MRI showed right parietal-occipital infarction, which did not conform to a cerebrovascular territory, and magnetic resonance arteriogram was normal. The second patient was a 20-month-old girl with left hemiparesis after a minor head trauma. Her head CT demonstrated symmetric basal ganglia calcifications, and her brain MRI showed right putamen and caudate ischemia. She developed a persisting nongap acidosis, with an abnormal acylcarnitine profile, consistent with mitochondrial disease.

Three patients' final diagnosis was "psychogenic." One was a 15-year-old girl with a family history of stroke who presented with right face and arm weakness and right arm and leg numbness. The second patient was a 16-year-old girl with a history of SVT and idiopathic intracranial hypertension who presented with blurred peripheral vision, nausea, and vomiting. The third patient was a 13-year-old girl with a family history of migraine who complained of right leg weakness and numbness.

Three patients were diagnosed with musculoskeletal abnormalities. The first was a 15-year-old girl with factor V Leiden mutation who complained of tingling and numbness of the left arm and leg. The second patient was a 14-year-old boy who was thought by the pediatrics service to have focal weakness after a prolonged PICU admission; he was ultimately diagnosed with deconditioning resulting from critical illness. The third patient was a 22-month-old girl with a limp whom the referring physician felt had right hemiparesis but whose formal neurologic examination was normal.

DISCUSSION

This is the first published study of a consecutive cohort of children with signs and symptoms of suspected stroke syndromes that were proven to be caused by conditions other than stroke (stroke mimics) after prospectively defined, systematic, protocol-driven diagnostic evaluation. Our study found that most patients (79%) referred to our pediatric stroke team were ultimately diagnosed with stroke syndromes. However, of the 21% with stroke mimics (conditions other than cerebrovascular disorders in which signs and symptoms resembled acute stroke syndromes), the majority (63%) were found to have serious neurologic disorders for which timely and specific management depended on prompt comprehensive diagnostic evaluation, including neuroimaging with MRI.

A comparison of the diagnostic and acute management challenges of stroke mimics in children with those of adults provides interesting similarities, as well as some important differences. Most studies of adult populations are focused on the accuracy of stroke diagnosis by emergency department physicians and/or first responders.[9–13] In several studies comparing admission diagnoses to discharge diagnoses, 4% to 19% of adult patients admitted to the hospital with a diagnosis of stroke were ultimately found to have an alternate diagnosis.[10–12]

Our rate of stroke mimics (21%) was slightly higher than that described in the adult literature. Of note, our stroke team is consulted for suspicion of SVT as well as AIS. In addition, only those patients for whom the diagnosis is difficult or the referring physician's suspicion for cerebrovascular disease is very high are seen by the stroke team. Therefore, the proportion of mimics in our cohort may not be directly comparable to the above-

mentioned studies of adults, which only included patients with suspected AIS.

Studies of adults with stroke mimics have demonstrated a variety of diagnoses, although characteristics of their presenting signs and symptoms are seldom detailed.[9–13] The final diagnoses in our cohort partially overlap but are not identical to those described in the adult literature for stroke mimics. The most commonly described diagnoses for stroke mimics in adults are seizure and postictal deficits, migraine, systemic infection, psychiatric disorders, brain tumor, toxic-metabolic abnormalities, cranial neuropathy, and syncope/presyncope. Our pediatric patients had a range of clinical presentations and final diagnoses, representing many of the conditions that should be considered in the differential diagnosis for children with acute neurologic deficits and/or suspected cerebrovascular disease and that are rarely reported in adult series. These include ADEM, RPLS, acute postinfectious cerebellitis, metabolic stroke, idiopathic intracranial hypertension, empyema, and intracranial abscess. Encephalitis, such as herpes simplex virus, could also be included in the differential diagnosis but did not occur in our series. Likewise, some stroke mimics observed in the adult literature were not seen in our series. These mimics include vertigo (peripheral or central), syncope or presyncope, autoimmune central nervous system disease other than ADEM (eg, sarcoid, systemic lupus erythematosus, central nervous system vasculitis), meningitis, and subdural hematoma. Children can certainly develop any of these diagnoses. It may be that the other pediatricians or neurologists involved in these patients' care correctly identified those diagnoses without the involvement of the stroke team. In addition, several of these conditions are rare enough that we would not anticipate seeing them in a relatively small cohort of patients. Our data suggest that just as risk factors for stroke in adults differ from those in children, the differential diagnosis for acute stroke is also different, with a greater representation of infectious diseases and a different spectrum of immune-mediated inflammatory conditions in children.

There is a need to quickly and accurately diagnose stroke in adult patients so that acute therapy such as intravenous tissue plasminogen activator (t-PA) can be administered.[14–17] The utility of t-PA in childhood stroke is limited, because it is extremely rare for a child with a stroke to be diagnosed within the 3-hour window for treatment with this medication and because the safety of t-PA has not been established in childhood stroke. The importance of timely and accurate diagnosis for children with suspected stroke is, nonetheless, illustrated by the results of our study. In patients who do have stroke syndromes, specific acute treatment such as antithrombotic agents for arterial infarction of thromboembolic origin, anticoagulation for SVT, and optimized cerebral perfusion using intravascular volume expansion or pressors may be considered. Institution of the appropriate, specific therapy in patients with stroke mimics depends on accurate diagnosis of a variety of conditions, many of which are time sensitive.

When Norris and Hachinski[18] first described adults with stroke mimics in 1982, they argued that the diagnosis can and should be made clinically. This article was published before the era of MRI. With new technology, our ability to make precise diagnoses is much enhanced. Although head CT was abnormal in almost all patients whose diagnoses were not benign, the CT alone was generally not specific enough for precise diagnosis. Therefore, brain MRI was usually required. Normal results were most helpful in diagnoses of exclusion such as complicated migraine, whereas abnormal results were important in directing appropriate medical and surgical management. Forty-four percent of our patients' brain MRIs had abnormalities that directly altered treatment, which argues strongly for expedited definitive neuroimaging for children with clinical presentations suspicious for stroke syndromes.

Our study has several limitations. Not all children with acute neurologic abnormalities are referred to our stroke team. Some patients are evaluated independently by their general pediatricians, and others are cared for by the general child neurology consultation service. Therefore, we cannot comment on the absolute incidence of childhood stroke mimics. Although our patients were enrolled prospectively, the charts were reviewed retrospectively. Therefore, the extent of the patients' evaluations was somewhat variable. For example, all 30 patients had neuroimaging, but 5 did not have a brain MRI. Finally, the number of patients in our study is relatively small. With a larger cohort, it may be possible to identify other features that distinguish patients with stroke mimics that are benign and not benign.

CONCLUSIONS

This study is the first to describe the clinical and imaging characteristics of a prospectively defined cohort of children with conditions that mimic stroke syndromes. Their presentations and diagnoses were diverse and distinctly different from those previously described in adults with stroke mimics. Except for the presence of seizures, history and clinical presentation did not distinguish the one third of patients with benign disorders from the two thirds with more serious problems. Therefore, complete evaluations, including brain MRI, are indicated for children with acute neurologic deficits. The prevalence of medically significant diagnoses in our patients with stroke mimics suggests that evaluation of children for suspected cerebrovascular disease should be comprehensive and timely.

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REFERENCES

1. Fullerton HJ, Wu YW, Zhao S, Johnston CS. Risk of stroke in children: ethnic and gender disparities. *Neurology.* 2003;61: 189–194
2. Giroud M, Lemesle M, Gouyon JB, Nivelon JL, Milan C, Dumas R. Cerebrovascular disease in children under 16 years of age in the city of Dijon, France: a study of incidence and clinical features from 1985 to 1993. *J Clin Epidemiol.* 1995;48:1343–1348
3. Steinlin M, Pfister I, Pavlovic J, et al. The first three years of the Swiss Neuropaediatric Stroke Registry (SNPSR): a population-based study of incidence, symptoms, and risk factors. *Neuropediatrics.* 2005;36:90–97
4. deVeber G; Canadian Paediatric Ischemic Stroke Study Group. Canadian Paediatric Ischemic Stroke Registry: analysis of children with arterial ischemic stroke [abstract]. *Ann Neurol.* 2000; 48:514
5. Lynch JK, Hirtz DG, deVeber G, Nelson KB. Report of the National Institute of Neurologic Disorders and Stroke workshop on perinatal and childhood stroke. *Pediatrics.* 2002;109: 116–123
6. Hutchison JS, Ichord R, Guerguerian AM, deVeber G. Cerebrovascular disorders. *Semin Pediatr Neurol.* 2004;11:139–146
7. Pediatric Stroke Working Group. Stroke in childhood: clinical guidelines for diagnosis, management and rehabilitation. Available at: www.rcplondon.ac.uk/pubs/books/childstroke. Accessed June 5, 2006
8. *SigmaStat for Windows* [computer program]. Version 3.0.1. Chicago, IL: SPSS Inc; 2003
9. Ay H, Buonanno FS, Rordorf G, Schaefer PW, Schwamm LH, Wu O. Normal diffusion-weighted MRI during stroke-like deficits. *Neurology.* 1999;52:1784–1792
10. Harbison J, Hossain O, Jenkinson D, Davis J, Louw SJ, Ford GA. Diagnostic accuracy of stroke referrals from primary care, emergency room physicians, and ambulance staff using the face arm speech test. *Stroke.* 2003;34:71–76
11. Kothari RU, Brott T, Broderick JP, Hamilton CA. Emergency physicians: accuracy in the diagnosis of stroke. *Stroke.* 1995;26: 2238–2241
12. Scott PA, Silbergleit R. Misdiagnosis of stroke in tissue plasminogen activator-treated patients: characteristics and outcomes. *Ann Emerg Med.* 2003;42:611–618
13. Libman RB, Wirkowski E, Alvir J, Rao TH. Conditions that mimic stroke in the emergency department: implications for acute stroke trials. *Arch Neurol.* 1995;52:1119–1122
14. National Institute of Neurologic Disorders and Stroke rt-PA Stroke Study Group. Tissue plasminogen activator for acute ischemic stroke. *N Engl J Med.* 1995;333:1581–1587
15. Cannon BC, Kertesz NJ, Friedman RA, Fenrich AL. Use of tissue plasminogen activator in a stroke after radiofrequency ablation of a left-sided accessory pathway. *J Cardiovasc Electrophysiol.* 2001;12:723–725
16. Thirumalai SS, Shubin RA. Successful treatment for stroke in a child using recombinant tissue plasminogen activator. *J Child Neurol.* 2000;15:558
17. Carlson MD, Leber S, Deveikis J, Silverstein FS. Successful use of rt-PA in pediatric stroke. *Neurology.* 2001;57:157–158
18. Norris JW, Hachinski VC. Misdiagnosis of stroke. *Lancet.* 1982; 319:328–331

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