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# Acute Neurological Deficit in a 13 Month Old Female

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**Abstract**

A 13-month-old female presented to the emergency department with acute neurological deficit. Her parents stated that she had awoken that morning at approximately 6:30 am only looking to the right, with left-sided mouth droop, and weakness on the left side. They reported that the child hit her head earlier that same evening after falling backwards against the wall. She did not lose consciousness and was acting normal after the incident, until bed-time, around 8:30 PM. She was recovering from an upper respiratory infection (URI) and had appeared very pale over the previous couple of weeks.

**Keywords:** Acute neurological deficit; Noncontrast computed tomography; Magnetic resonance; Imaging; Transient ischemic attack

## Case Description

The child's past medical history was only significant for operative release of craniosynostosis approximately 9 months previously, with no complications. She did not take any medications and had no possible exposure to medications or toxins. Her family history was non-contributory. Physical exam was significant for deviated gaze to the right, left-sided facial droop, and left hemiparesis, with decreased sensation on the left side. On admission, her Pediatric -NIH Stroke Scale was 8.

Noncontrast Computed Tomography (CT) scan was negative for any intracranial bleed or skull fracture. Magnetic Resonance Imaging (MRI) and Magnetic Resonance Angiography (MRA) and were negative for any intraparenchymal or vascular anomaly. Electrocardiogram and echocardiography were unremarkable. Hypercoaguability workup revealed no predisposition to thrombosis. Complete Blood Count (CBC) and differential was significant for a hemoglobin of 6.2 g/dL (62 g/L) and hematocrit of 18.4%, and absolute reticulocyte count was 3.83 (% reticulocyte count = 0.163%). Total serum ferritin and iron were normal as was the basic metabolic panel.

The patient was transfused with 10 cc/kg of packed red blood cells, and hemoglobin increased to 8.7 g/dL. She recovered to baseline within 24 hours (Ped-NIHSS =0), and no further treatment was necessary. The child has progressed well since the incident, without recurrence of neurological deficits.

## Diagnosis

Transient Ischemic Attach (TIA) and Transient Erythroblastopenia of Childhood (TEC); the severe anemia, which developed sub-acutely after the recent URI, was felt to be the cause of the TIA.

## Discussion

Incidence of pediatric stroke (including ischemic and hemorrhagic) is reported to be between 2.3 and 13 per 100,000 children per year Roach et al. [1]. Approximately 55% of pediatric strokes are ischemic with the remaining 45% being hemorrhagic. The most common conditions associated with ischemic events in children include sickle cell disease (SCD), congenital or acquired heart disease, infections, anemia, prothrombotic disorders, and arterial malformation. Ganesan et al. [2]. Boys are at higher risk than girls and African-American children are at higher risk than Asian or Caucasian children independent of predisposing conditions or events Roach et al. [1].

Transient Ischemic Attack (TIA) is defined as a transient episode of neurological dysfunction caused by focal ischemia, without acute infarction. TIA and stroke in pediatric patients can often mimic other conditions that are more common in this population such as complicated migraines or postictal neurological deficits Tsze and Valente [3], Transient Erythroblastopenia of Childhood (TEC), defined by a diminished number of erythroblasts in the bone marrow, has been recently associated with TIA occurrence. Neurological deficits have been reported in one out of seven patients with TEC, however no lesion was identified by brain CT or CT angiography in these patients Chan et al. [4]. Causes of TEC include nutritional anemia, gastroenteritis, and respiratory tract infections Ganesan et al. [5]. The mechanism of the neurological involvement remains unknown, but it is postulated that erythroblastopenia leads to anemic hypoxia which is responsible for the neurologic symptoms Young et al. [6]. As neurological complications have been reported in patients with TEC and hemoglobin as high as 9.1 g/dL, this theory is under dispute. Additionally, studies reporting a tendency for temporal clustering of TEC in the spring or fall have suggested a purely infectious etiology or an immune-mediated process causing a vasculitis or neuropathy Chan et al. [4].

To the best our knowledge, while silent infarction and TIAs have been extensively described in the setting of Moya-Moya, sickle cell disease Ohene-Frempong et al. [7] or presence of prothrombotic factors Roach et al. [1] no cases of TIA followed transient erythroblastopenia of childhood have been reported.

Minor head or neck injuries, including falls from minor heights, have been reported to precipitate cerebral infarction in children. The pathophysiology of this association is not completely understood, however it is postulated that either thrombus or spasm in the lenticulostriate branches of the middle cerebral artery (MCA) with consequent decrease in local blood flow is responsible. These vessels, more so in infancy and gradually less so as one ages, emerge at an acute angle from the MCA. In children these vessels have a larger mobile extracerebral segment which might make them more susceptible to

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injury following minor head trauma leading to ischemic changes in the lentiform nucleus and the head of the caudate nucleus Nabika et al. [8].

Timely recognition and diagnosis of pediatric cerebrovascular events including TIAs is of paramount importance as the consequences of brain ischemia can be devastating. Residual sequelae including motor or cognitive impairment, behavioral problems, and epilepsy are reported to occur in as many as 75% of children after ischemic stroke Ganesan et al. [9]. As many as 10-25% of children with stroke will experience a recurrence and up to 25% of pediatric stroke victims will die with increased mortality in case of recurrence Lanthier et al. [10]. Therefore, it is essential for the primary care provider to consider this diagnosis when appropriate, as early detection and management is crucial for optimal outcome.

The goal of treatment in childhood stroke is to preserve neurological function in the acute setting as well as to prevent recurrence. Acute supportive care includes correction of hypoxemia, control of fever and hypertension, and normalization of serum glucose levels. It is also recommended to treat dehydration and anemia Roach et al. [1]. Secondary prevention of ischemic stroke in children can be accomplished by use of antiaggregation or anticoagulation with low molecular weight heparin (LMWH) or warfarin as appropriate, in accordance to recently published guidelines Roach et al. [1]. Thrombolytic therapy with alteplase (t-PA) is currently not recommended for children with ischemic stroke until clear safety and efficacy data date will be available Roach et al. [1]. A clinical trial regarding safety of t-PA in children is underway Amlie-Lefond et al. [11].

## Conclusion

TIA in children is a rare occurrence that primary care physicians should consider in their differential diagnosis due to the potential progression to ischemic stroke with resulting severe morbidity. TIA as possible complication of transient erythroblastopenia of childhood should prompt early recognition and consideration for treatment of this condition by the primary care physician.

All potential causes of stroke should be excluded in a child presenting with TIA. Early diagnosis and management of identified risk factors is key to improved clinical outcome.

### References

1. Roach ES (2008) Management of stroke in infants and children: a scientific statement from a special writing group of the American heart association stroke council and the council on cardiovascular disease in the young. Stroke 39: 2644-2691.
2. Ganesan V, Prengler M, McShane MA, Wade AM, Kirkham FJ (2003) Investigation of risk factors in children with arterial ischemic stroke. Ann Neurol 53: 167-173.
3. Tsze DS, Valente JH (2011) Pediatric stroke: a review. Emerg Med Int 2011: 734506.
4. Chan GC, Kanwar VS, Wilimas J (1998) Transient erythroblastopenia of childhood associated with transient neurologic deficit: report of a case and review of the literature. J Paediatr Child Health 34: 299-301.
5. Ganesan V, Hogan A, Shack N, Gordon A, Isaacs E, et al. (2000) Outcome after ischaemic stroke in childhood. Dev Med Child Neurol 42: 455-461.
6. Young RS, Rannels DE, Hilmo A, Gerson JM, Goodrich D (1983) Severe anemia in childhood presenting as transient ischemic attacks. Stroke 14: 622-623.
7. Ohene-Frempong K, Weiner SJ, Sleeper LA, Miller ST, Embury S, et al. (1998) Cerebrovascular accidents in sickle cell disease: rates and risk factors. Blood 91: 288-294.
8. Nabika S, Kiya K, Satoh H, Mizoue T, Oshita J, et al. (2007) Ischemia of the internal capsule due to mild head injury in a child. Pediatr Neurosurg 43: 312-315.
9. Ganesan V, Hogan A, Shack N, Gordon A, Isaacs E, et al. (2000) Outcome after ischaemic stroke in childhood. Dev Med Child Neurol 42: 455-461.
10. Lanthier S, Carmant L, David M, Larbrisseau A, de Veber G (2000) Stroke in children: the coexistence of multiple risk factors predicts poor outcome. Neurology 54: 371-378.
11. Amlie-Lefond C, Chan AK, Kirton A, deVeber G, Hovinga CA, et al. (2009) Thrombolysis in acute childhood stroke: design and challenges of the thrombolysis in pediatric stroke clinical trial. Neuroepidemiology 32: 279-286.