

Pediatric Ischemic Stroke Presenting with Altered Consciousness and Brainstem Signs

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ABSTRACT

While ischemic stroke is rare in children, with an incidence rate of 1/3500 live births in newborns and 1–2 per 100,000 annually during childhood—peaking during the perinatal period, under the age of 5, and in adolescence—it ranks among the top 10 causes of death. It poses significant diagnostic and treatment challenges because of its multiple causes and often subtle, variable symptoms. The variability in symptoms based on age can delay diagnosis and worsen outcomes. Early management should focus on prompt recognition, a standardized approach (ABCDE), and an early neurological examination with neuroimaging—preferably magnetic resonance imaging (MRI). This case involves a healthy school-aged child who suddenly developed neurological symptoms at home and was later diagnosed with a brainstem stroke caused by thrombosis of the right posterior cerebral artery (PCA). Although the computed tomography (CT) scan appeared normal, the MRI confirmed the diagnosis. Laboratory tests showed elevated Lipoprotein (Lp) levels, a recently recognized independent risk factor for pediatric arterial ischemic stroke. Overall, this case underscores the importance of early detection, advanced neuroimaging, and thorough etiologic evaluation, including thrombophilia screening, in the diagnosis and management of pediatric stroke.

Keywords: Initial approach; ischemic stroke; management; neuroimaging.

INTRODUCTION

Pediatric stroke is among the top 10 causes of death in children.¹ However, it remains poorly understood, often leading to significant delays in diagnosis, despite being a rare but increasingly recognized cause of acute neurologic dysfunction in children. The variety of clinical presentations and the lack of randomized clinical trials on acute stroke treatment in children contribute to delayed diagnosis and misdiagnosis.² As a result, multiple causes and age-specific differences in clinical symptoms make prompt diagnosis difficult, often resulting in delayed treatment, worse outcomes, and a long-term financial burden for families.³

According to the World Health Organization (WHO), pediatric ischemic stroke is characterized by an acute neurological sign or symptom caused by focal brain infarction that results in severe short- and long-term disabilities, such as neuropsychological impairment and deficits in motor, cognitive, or behavioral functions. Furthermore, the causes and clinical features of AIS differ between neonates (<29 days old) and older children (29 days to 18 years old).⁴ It presents with symptoms lasting more than 24 hours or until death, along with neuroimaging, pathological, or other objective evidence of focal ischemic injury, with an estimated incidence of 2 to 13 per 100,000 children per year.⁵ Unlike adult stroke, which is usually associated with modifiable cardiovascular risk factors like hypertension, diabetes, smoking, and atherosclerosis, pediatric stroke often results from various

underlying issues such as arteriopathies, chronic inflammatory diseases, sickle cell anemia, cardiac problems, hypercoagulable states, metabolic disorders, trauma, infection, dehydration, cancer, and inherited thrombophilias.⁵ In children, AIS is a multifactorial disease, and the combined effect of multiple risk factors increases the risk of thrombosis. A significant proportion of children with AIS have no identifiable risk factors. These factors can cause pediatric ischemic stroke, which often presents with subtle or atypical symptoms. It is important to note that data on children with stroke cases positive for SARS-CoV-2 are limited.⁶ Recent literature indicates that lipoprotein(a) (Lp(a)) may serve as a critical, previously unrecognized risk factor for pediatric AIS. Different pediatric guidelines and consensus papers now highlight increased Lp(a) levels as a potential risk factor for pediatric stroke and recommend testing for Lp(a) in children with an ischemic stroke of unknown cause.^{6,7} Notably, strokes in the posterior circulation—accounting for about 10–20% of all pediatric strokes—may present with nonspecific signs like headache, vomiting, vertigo, confusion, syncope, nausea, seizures, or altered mental status rather than apparent focal deficits.⁸ Although less common, brainstem strokes are severe because this area contains vital autonomic, motor, and cranial nerve pathways.⁸

Neuroimaging is essential for early detection. While computed tomography (CT) is commonly used initially in



emergency settings, it has limited sensitivity for detecting acute strokes in the posterior fossa. Magnetic resonance imaging (MRI), especially diffusion-weighted imaging (DWI), offers much higher sensitivity and is considered the gold standard for diagnosing early ischemic injuries in children.⁹ However, practical delays in diagnosis can occur due to limited access to MRI. Additionally, echocardiography should be performed for all children with AIS to exclude possible cardio-embolic causes; this should not delay urgent treatments like thrombolysis or thrombectomy. It is worth noting that this guideline is based on adult data, and our understanding of its application in children remains limited. It is also important to note that another key aspect in pediatric stroke is screening for thrombophilic disorders.^{9,10}

It is important to recognize that recently, elevated lipoprotein Lp(a) levels have been identified as an independent risk factor for arterial ischemic stroke in children, especially when no other clear causes are present. Lp(a) plays a role in promoting blood clotting and inflammation, potentially increasing the risk of thrombotic vascular events even when traditional risk factors are absent.^{7,11}

This report describes a previously healthy school-aged child who developed acute neurological symptoms and was later diagnosed with a brainstem infarction caused by thrombosis of the right posterior cerebral artery. The case highlights the importance of early recognition, appropriate neuroimaging, and a thorough etiologic workup—including evaluation of Lp(a)—in pediatric stroke.

CASE

A previously healthy 11-year-old boy woke up acting normally and was getting ready for school. However, the mother reported that the child suddenly developed a severe headache, double vision, dizziness, and vomited once. Soon after, the child showed eye deviation, became very drowsy, and lost the ability to speak, which led the family to call emergency services (112). When they arrived, they measured a blood glucose level of 250 mg/dL, and the child was taken to the pediatric emergency department with a preliminary diagnosis of hyperglycemia.

Past Medical History does not include known chronic illnesses, trauma, intoxication, or medication use. There is also no family history of stroke or prior syncopal or seizure episodes, so prenatal, natal, and postnatal history were normal.

During admission, the patient was critically ill and drowsy, with a Glasgow Coma Scale (GCS) score of 11. The neurological assessment revealed intermittent responsiveness, delayed

reactions, bilateral miosis, sluggish light reflex, normal muscle tone, and symmetrical deep tendon reflexes. No abnormal reflexes were found. Other system evaluations showed pale skin; the heart produced clear sounds; lung auscultation detected vesicular breath sounds; the abdomen was soft and non-tender; and there was no hepatosplenomegaly. Over several hours, the GCS dropped to 10, and the child developed left-sided ptosis, facial nerve paresis, and hemiparesis on the same side. The child had no active movement on the left side.

Initially, the CT scan of the brain did not detect any focal abnormalities. Blood tests, including a coagulation profile, CRP, liver and kidney function tests, electrolyte levels, and glycosylated hemoglobin, all fell within normal ranges; however, the patient's clinical condition worsened. An MRI of the brain was performed and revealed an acute ischemic lesion in the brainstem and a thrombus in the proximal right posterior basilar artery (Fig.1, 2, and 3).

FIGURE 1. A. Restriction of diffusion in the brainstem T2 dark fluid; B. T2 dark-fluid

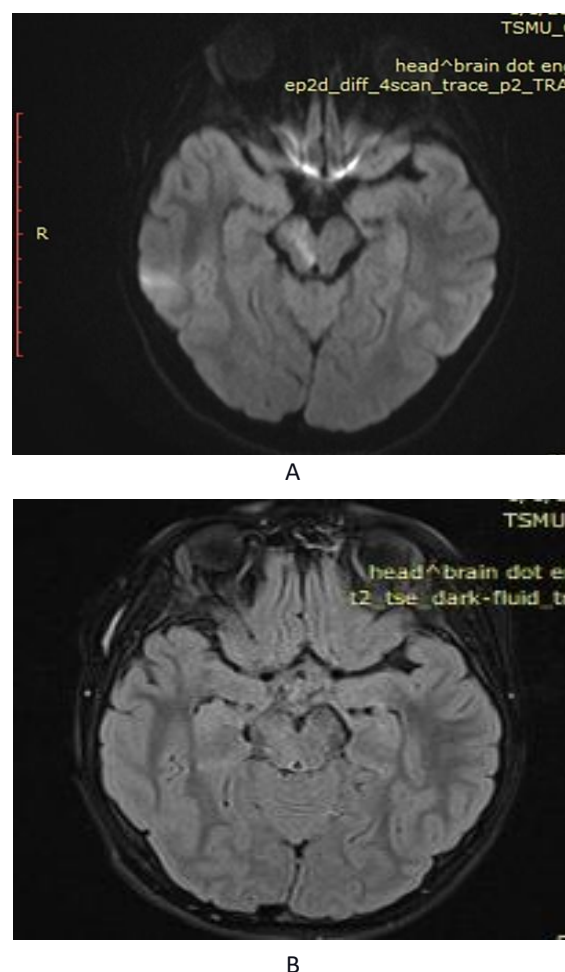


FIGURE 2. Filling defect on the projection of the basilar artery (axial projection)

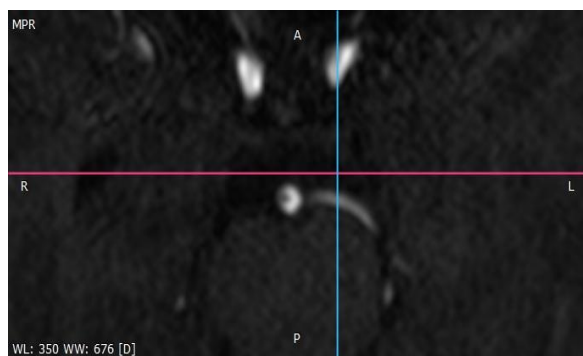


FIGURE 3. Excavation and filling defect on the projection of the basilar artery to the right posterior cerebral artery (frontal projection)



Further workup for the etiology of ischemic stroke included a thrombophilia panel, which tested for anticardiolipin antibodies (IgG/IgM), lupus anticoagulant, proteins C and S, Factor II, V, VII, and X, antithrombin, and Lipoprotein(a). The Lipoprotein(a) level was elevated at 1.03 g/L (normal < 0.3 g/L). Additional tests for homocysteine, TAT complex, ferritin, organic acids, plasma amino acids, and ammonia were all within reference ranges.

Based on clinical evaluations and examinations, the definitive diagnosis was Cerebral infarction caused by thrombosis of cerebral arteries - ICD-10 Code: I63.3.

Management involved initiating aspirin therapy at 2 mg per kg, starting from an initial dose of 1 mg per kg, consulting neurosurgery, and emphasizing that no surgical intervention was necessary. Supportive care was provided in the pediatric emergency department and later in the neurology ward. After treatment, the patient gradually improved and was discharged home after two weeks, neurologically stable, with a prescribed aspirin 100 mg per day (2 mg per kg).

DISCUSSION

Pediatric ischemic stroke is rare but serious, with challenging difficulties in diagnosis and treatment. Unlike adult strokes, which are often linked to modifiable risk factors like hypertension and diabetes, strokes in children usually result from various congenital or acquired conditions.¹⁻¹² These include arteriopathies, heart anomalies, trauma, infections, metabolic and blood disorders, and inherited or acquired thrombophilias. The wide range of causes makes quick diagnosis and standard treatment more complicated, often leading to delays and poorer outcomes.^{12,13}

In this case, a previously healthy 11-year-old boy developed acute neurological symptoms—severe headache, dizziness, double vision, vomiting, and drowsiness—that quickly progressed to altered consciousness, eye deviation, and unilateral cranial nerve involvement. These signs are consistent with a posterior circulation stroke, which accounts for 10–20% of pediatric arterial ischemic strokes. Such strokes often present with nonspecific symptoms like nausea, vertigo, confusion, and vomiting, which can be mistaken for more benign conditions such as viral illness, migraine, or intoxication—leading to diagnostic delays.

Brainstem strokes are critical and demand swift recognition and imaging, as delays can cause permanent neurological damage. CT is the most frequently used imaging tool for suspected stroke cases due to its availability, speed, simplicity, and lower cost compared to MRI.⁹ Nonetheless, while CT effectively detects acute intracranial hemorrhage, it is less sensitive to acute ischemic stroke. In this case, the initial CT scan did not reveal ischemic events, making MRI vital for identifying acute infarction and vascular issues.¹³ The early non-contrast CT was uninformative, a common finding during early ischemic stages, especially in the posterior fossa. As highlighted earlier, MRI with diffusion-weighted imaging (DWI) is far superior for detecting early ischemic signs and should be the preferred imaging when clinical suspicion is high. Here, MRI confirmed a brainstem infarction and revealed thrombus in the right posterior cerebral artery (PCA), directly linked to the patient's neurological symptoms.^{9,12,13}

Additionally, it should be understood that Thrombophilia screening is a vital part of the cause investigation in pediatric stroke, especially in children without obvious risk factors. Although most cases are multifactorial or idiopathic, identifying an underlying prothrombotic condition can help guide secondary prevention strategies.¹² In this patient, a significantly elevated Lipoprotein(a) Lp(a) level was found (1.03 g/L; normal < 0.3 g/L). Lp(a) is increasingly recognized as

an independent risk factor for arterial ischemic stroke in children due to its role in promoting both blood clots and vascular inflammation. Its presence in otherwise healthy children with stroke highlights the need for increased awareness and possibly routine measurement of Lp(a) during pediatric stroke evaluations.

The absence of other significant findings on thrombophilia testing, cardiac evaluation, and metabolic panels suggests that elevated Lp(a) may have been the primary contributing factor in this case. This aligns with growing evidence that Lp(a) may independently predispose individuals to thrombotic events even in the absence of conventional risk factors.

Treatment options for pediatric stroke remain limited and are mainly extrapolated from adult protocols due to the lack of randomized controlled trials in children.^{2,14} Although thrombolysis and mechanical thrombectomy have shown promise in select pediatric cases, their use remains off-label, institution-dependent, and often constrained by narrow time windows and logistical issues.^{1,12} In this case, conservative management with aspirin was started, in line with current guidelines for non-cardioembolic arterial ischemic stroke in children, and the neurosurgeon did not consider surgical intervention.^{14,15} The patient responded well to antiplatelet therapy and supportive care, with gradual neurological improvement and a stable condition after two weeks.

CONCLUSIONS

This case underscores several practical lessons for clinicians:

- Pediatric strokes can present with vague or nonspecific symptoms, making early diagnosis particularly challenging. A high index of suspicion is crucial.
- When posterior circulation involvement is suspected, MRI remains the imaging modality of choice, offering greater sensitivity than CT in the acute phase. Magnetic resonance imaging (MRI) is considered the gold standard for emergency evaluation of patients with acute stroke, including both ischemic and hemorrhagic types.
- The role of lipoprotein(a) in stroke in children may be underappreciated. Its inclusion in thrombophilia testing may help identify less obvious risk factors. Our case highlights the importance of measuring Lp(a) levels in all children with AIS, mainly when no other cause is found.
- A prompt and thorough diagnostic workup is key to uncovering rare underlying causes and tailoring appropriate management.

Because childhood stroke is rare, diagnosing it can be challenging and delay treatment decisions. Early detection and

prompt intervention greatly enhance outcomes, emphasizing the crucial role of a multidisciplinary team approach.

INFORMED CONSENT

Informed consent was obtained from the subject's parents.

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