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CASE REPORT **OPEN ACCESS**

UNKNOWN RISK FACTOR IN PEDIATRIC ISCHEMIC STROKE: CHALLENGES IN A DEVELOPING COUNTRY

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ABSTRACT

Background: Stroke is a sudden neurological disturbance with high mortality rates in both children and older people. Pediatric stroke is rare but can lead to lifelong disability due to the immaturity of the brain, and its management can be challenging.
Case: A 23-year-old female presented with a history of right-sided weakness and spasticity that began 11 years ago and improved over the past two years. Risk factors that could be excluded were ruled out, while congenital disorders could not be investigated due to financial constraints. Diagnosed with non-hemorrhagic stroke at age 12, an MRI revealed an old infarct in the left internal capsule. The absence of known risk factors underlying her ischemic stroke made a congenital disorder a plausible diagnosis. She was given botulinum toxin type A (BoNT-A) injections to manage spasticity, and her symptoms improved. In the past year, she developed mood disturbances and sleep difficulties, which were treated with quetiapine.
Discussion: The pediatric brain is metabolically more active with higher cerebral blood flow demands, making it more susceptible to focal neurological injuries. Most pediatric strokes are ischemic, with numerous potential risk factors including cardiac issues, vasculopathy, coagulopathies, infections, and congenital disorders with vascular complications. Treatment for pediatric ischemic stroke is similar to that in adult cases. In this patient, BoNT-A injections reduced acetylcholine release, effectively treating the patient's spasticity.
Conclusion: Stroke can occur at any age, with pediatric strokes potentially resulting in worse outcomes. Therefore, thoroughly evaluating diverse potential etiologies, including congenital disorders, is crucial.

Keywords: botulinum toxin, ischemic stroke, pediatric stroke, unknown risk factor

Introduction

According to the WHO, stroke is defined as a sudden neurological or functional brain disturbance that lasts for more than 24 hours and can result in death. Stroke is a non-communicable disease caused by a disruption of blood flow to a specific part of the brain, leading to focal symptoms or affecting the entire brain with global symptoms.[1] Stroke can occur at any age, with high morbidity and mortality rates in both older people and children, with mortality rates in children ranging from 10 to 25%. Although it is more commonly found in older people, strokes in pediatric populations can lead to a longer duration of disability, potentially lasting throughout the lifespan. The incidence of stroke in children from birth to 28 days is 10.2 per 100,000 live births, while from 29 days to 18 years, it is 1.72 per 100,000 children per year.[2]

Stroke is one of the top 10 causes of death in children, with a higher mortality rate in hemorrhagic stroke than in ischemic stroke.[2,3] The prevalence of ischemic stroke can be 4-5 times higher in developing countries compared to developed countries.[2] Pediatric stroke survivors often live with disabilities for decades. About 10-20% experience recurrent strokes, and more than half have long-term neurological impairments.[3] Even up to 20 years after a pediatric ischemic stroke, there is a risk of epilepsy.[4]

Based on age, pediatric strokes can be divided into perinatal and childhood. A perinatal or neonatal stroke occurs between 28 weeks of gestation and 28 postnatal days (the first month of life for term babies), while childhood stroke occurs after 28 days up to 18 years of age. Perinatal stroke can be further divided into acute perinatal stroke, which typically occurs following focal seizures or encephalopathy in newborns or near birth, and presumed perinatal stroke, which consists of chronic infarcts in the perinatal period, often presenting with seizures or pathological early handedness. Asymptomatic infarcts occurring in older infants and children can be classified as silent strokes or silent infarcts. If undetected, they may lead to vascular cognitive impairment in adulthood. Perinatal stroke results from disruptions in either arteries or veins, manifesting as both ischemic and hemorrhagic types. Approximately 80% of these cases are acute ischemic stroke (AIS), often presenting with focal motor seizures involving one extremity. The left cerebral hemisphere is most commonly affected, with the majority of lesions occurring in the middle cerebral artery (MCA). This condition is more prevalent in males. Basal ganglia involvement leads to motor outcomes, while non-motor involvement can result in cognitive or behavioral outcomes and visual deficits. The remainder of perinatal strokes results from cerebral sinovenous thrombosis (CSVT) and hemorrhage, which are usually nonspecific, manifesting as seizures, lethargy, or irritability.[5]

Childhood stroke presents more similarly to adults, with 67-90% experiencing weakness and hemifacial weakness, 20-50% experiencing speech or language disturbances, 10-15% experiencing vision impairments, and 8-10% experiencing ataxia. There are also non-localizing symptoms, with 20-50% experiencing headaches, 17-38% experiencing altered consciousness, and 15-25% experiencing seizures. Based on the most common risk factors, strokes can be categorized into moyamoya disease, where 70% of cases have transient ischemic attacks (TIAs) and 50% have silent infarcts; cardiac-related strokes, which typically occur between 6 months and 8 years of age; and posterior circulation strokes, often associated with vertebral artery dissection due to minor head or neck trauma, especially in boys (67-77%).[5] Similar to perinatal strokes, childhood strokes result from AIS, CSVT, or hemorrhage. Moyamoya disease has higher prevalence and incidence rates in Asians, with a predominance in females.[6]

Management of pediatric stroke is often challenging due to the subtle or atypical clinical presentations.[7] Children have worse functional outcomes than adults due to the immaturity of the brain, which may lead to developmental milestone disruptions, making it critical to treat them.[3,5] This case report aims to describe a childhood ischemic stroke with unknown risk factors. A chronological and focused history was taken, detailing the onset and progression of symptoms over the past 10 years. Previously, the patient underwent treatment at another healthcare facility, and subsequent clinical improvements during the last 2 years of care at our hospital.

Case Report

A 23-year-old female patient presented with a 10-year history of right-sided weakness and slurred speech. She had been diagnosed and treated at a previous hospital, but was then referred to our center after showing no clinical improvement. The sequelae were accompanied by spasticity, resulting in flexion of the right elbow, wrist, and fingers. The patient also experienced tingling on the right side and right-sided headaches, but was still able to mobilize independently. She had no fever, joint pain, skin rashes, kidney problems, history of allergies, or family history of autoimmune disease, making autoimmune disease less likely. She also had no stiff neck, chills, seizures, drowsiness, or confusion, reducing the likelihood of meningitis. The patient had no intellectual disability, developmental delays, joint laxity, abnormal facial features, small hands and feet, congenital heart defects, or other organ problems, making congenital disorders like Down's syndrome less likely. She had no neurofibromas or bone deformities, making neurofibromatosis type 1 (NF1) less likely. Additionally, there was no history of trauma, balance problems, or substance abuse. However, congenital disorders such as antiphospholipid syndrome (APS), cerebral autosomal dominant arteriopathy with subcortical infarcts and leukoencephalopathy (CADASIL), cerebral autosomal recessive arteriopathy with subcortical infarcts and leukoencephalopathy (CARASIL), or even moyamoya disease were often undiagnosed due to financial constraints. The patient had been diagnosed with a non-hemorrhagic stroke at the age of 12 and was treated with clopidogrel and mecobalamin.

Neurological assessment revealed that the patient was alert and responsive. There was decreased muscle strength (4/5) and increased muscle tone, particularly in the upper limb flexor muscles. Spasticity was noted with a Modified Ashworth Scale (MAS) score of 2 in the right upper limb, indicating a marked increase in tone through most of the range of motion, but the limb was still easily moved. Deep tendon reflexes were increased on the right side. These findings are consistent with an upper motor neuron lesion.

Several laboratory tests were performed at our hospital as part of a workup to rule out possible differential diagnoses. The results were largely unremarkable, except for elevated uric acid levels, an increased erythrocyte sedimentation rate (ESR), and occasional elevations in blood glucose (Table 1). These

investigations aimed to identify or exclude underlying systemic, infectious, metabolic, or autoimmune conditions that could explain the patient's clinical presentation. A brain MRI without contrast revealed an old infarct in the posterior crus of the left internal capsule, while other intracranial structures were within normal limits (Figure 1). A brain CT scan without contrast showed chronic rhinitis, a deviation of the nasal septum to the right, and no signs of infarction, hemorrhage, or intracranial space-occupying lesions, ruling out brain tumors (Figure 2). CT angiography or digital subtraction angiography (DSA) was not performed due to limited financial resources and restricted access.

Table 1. Laboratory tests performed at our hospital over the past 2 years (listed from the most recent to oldest)

TEST	RESULT	
February 2024		
Haemoglobin	12.90 g/dL	11.70 - 15.50
Hematocrit	41.60 %	35.00 - 47.00
Erythrocyte	4.95 10^6/µL	3.80 - 5.20
White blood cells	8.15 10^3/µL	3.60 - 11.00
Differential count:		
Basophil	1%	0 - 1
Eosinophil	2%	1 - 3
Band Neutrophil	3%	2 - 6
Segment Neutrophil	60%	50 - 70
Lymphocyte	26%	25 - 40
Monocyte	8%	2 - 8
Platelet Count	296.00 10^3/µL	150.00 - 440.00
ESR	21 mm/hours (H)	0 - 20
MCV	84.00 fL	80.00 - 100.00
MCH	26.10 pg	26.00 - 34.00
MCHC	31.00 g/dL	32.00 - 36.00
Uric Acid	5.89 mg/dL	2.6 - 6.0
LDL Cholesterol (Direct)	95 mg/dL	<100 (optimal)
June 2023		
Uric Acid	5.70 mg/dL	2.6 - 6.0
LDL Cholesterol (Direct)	88 mg/dL	<100 (optimal)
Fasting Blood Glucose	99.0 mg/dL (desirable)	<100 (desirable)
September 2022		
Uric Acid	**6.50 mg/dL (H)**	2.6 - 6.0
LDL Cholesterol (Direct)	110 mg/dL	<100 (optimal)
Trygliceride	72 mg/dL	50-150
Fasting Blood Glucose	103.0 mg/dL (moderate)	101-125 (moderate)
Blood Glucose 2 hours PP	**176.0 mg/dL(H)**	<140

ESR: Erythrocyte Sedimentation Rate, H: High, L: Low, LDL: Low-Density Lipoprotein, MCH: Mean Corpuscular Hemoglobin, MCHC: Mean Corpuscular Hemoglobin Concentration, MCV: Mean Corpuscular Volume, PP: postprandial

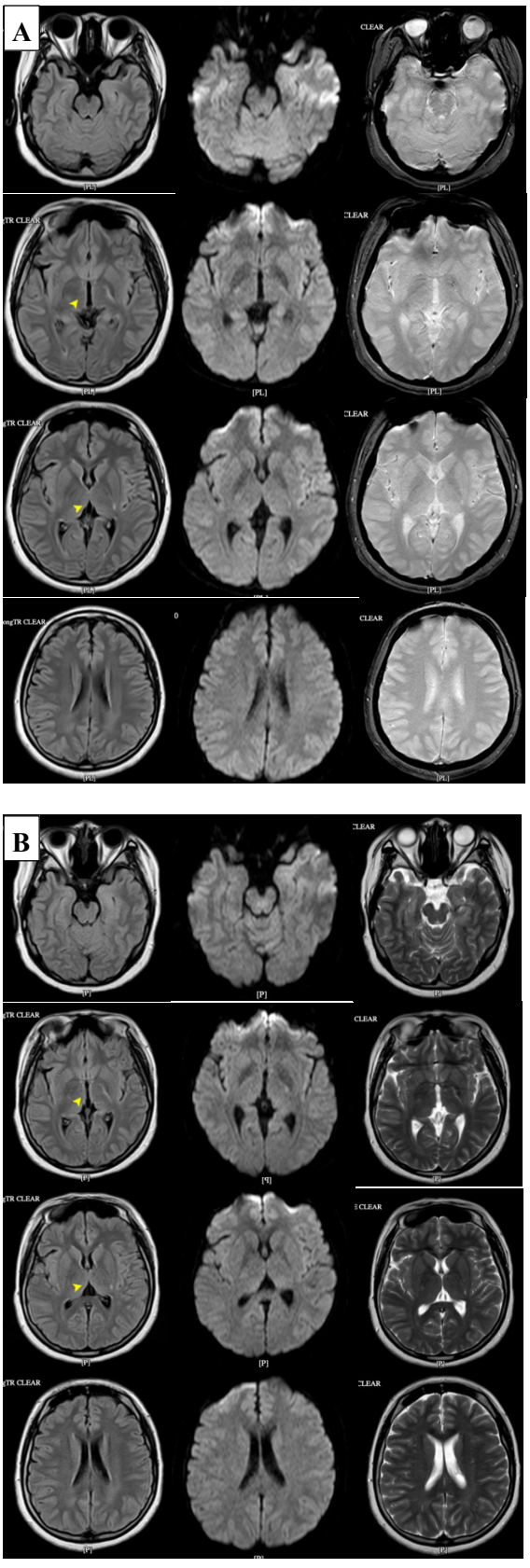

Figure 1. Brain MRI without contrast, performed in (A) 2023 and (B) 2022, showed pathological lesions in the posterior crus of the left internal capsule (yellow head arrow), consistent with an old infarct, while other intracranial structures were within normal limits

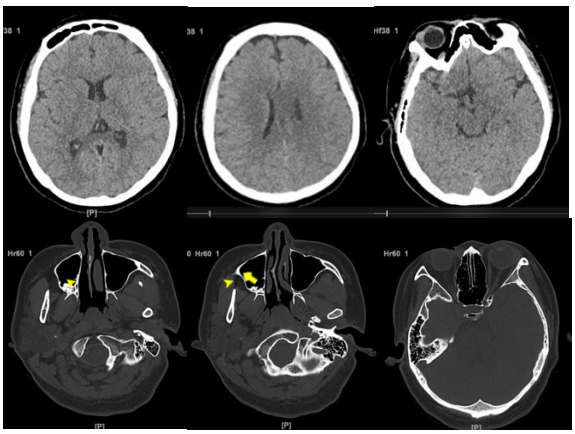

Figure 2. Brain CT scan without contrast, performed in 2023, showed bilateral thickening of the inferior nasal turbinates (yellow head arrow), suggestive of chronic rhinitis and a deviated nasal septum (yellow arrow). No signs of infarction, hemorrhage, or intracranial space-occupying lesions were observed

The patient was treated with low-dose BoNT-A (10 units) injections monthly for two years, as well as allopurinol 100 mg once daily, mecobalamin 500 mg every 8 hours, and clopidogrel 75 mg once daily, which was later changed to cilostazol 100 mg once daily due to gastrointestinal side effects. After the monthly BoNT-A injections, follow-up results showed that her right-sided weakness was no longer accompanied by spasticity. In the past year, she had shown a dysphoric mood and irritability, anxiety, difficulty sleeping, and persecutory ideas that many people held negative thoughts about her. She was treated with a low dose of quetiapine and showed high sensitivity to antipsychotics, specifically experiencing agitation when the dose was increased. Risperidone 1 mg per 12 hours, Lithium carbonate 200 mg once daily, trihexyphenidyl 1 mg per 12 hours.

Discussion

Pediatric stroke is an essential cause of acquired intracranial injury in young patients, with significant mortality and morbidity. It can result in death or severe disability if misdiagnosed or if there is a lack of clinical suspicion and appropriate workup.[5,8] The National Institutes of Health (NIH) Common Data Elements define pediatric stroke as the acute onset of neurological signs or symptoms attributable to focal brain infarction or hemorrhage.[5] Ischemic stroke is caused by a sudden loss of blood flow to the brain, leading to focal cerebral, spinal, or retinal infarction. This can result in irreversible disability, neurological function defects, and death.[9]

Several structural and physiological differences between adults and children lead to different stroke presentations. The pediatric brain is metabolically more active, with higher demands for cerebral blood flow and up to 200% greater glucose usage than the adult brain. This makes it more susceptible to focal neurological injury during hypoglycemia.[2] In neonates, the most common presenting symptom of stroke is seizures, while in older children, focal deficits such as weakness are more common. As many as 70% of non-neonates and 60% of neonates who present with focal deficits may experience lifelong residual focal deficits. Non-specific symptoms can also appear in 49% of non-neonates, such as headache, nausea and vomiting, fever, and cardiopulmonary dysfunction, with or without focal deficits.[2] Children with a sudden onset of focal weakness, headache, seizures accompanied by neurological symptoms, visual or speech disturbances, limb incoordination or ataxia, signs of increased intracranial pressure, or altered mental status are at high risk of stroke and require neurological examination and urgent neuroimaging.[3] As in this case, where the patient presented with sequelae of right-sided weakness, the clinical picture suggests a high risk of prior cerebrovascular events and underscores the need for a thorough neurological evaluation.

Etiology of Pediatric Stroke

Several conditions can be risk factors for stroke in children, and no single risk factor significantly reduces neurological harm. The most relevant risk factors include cardiac causes, vasculopathies, coagulopathies, and infections. Other risk factors include autoimmune diseases, metabolic disorders, hematological diseases, renal diseases, child abuse, and head trauma. Mastrangelo et al. summarize the main risk factors for childhood stroke in children aged 28 days to 18 years (Table 2).[7]

Genetic and hereditary disorders are also significant risk factors for pediatric stroke, necessitating an adequate assessment of family history and the identification of signs of underlying genetic disorders. Common genetic causes of strokes include Marfan syndrome, pseudoxanthoma elasticum, Ehlers-Danlos syndrome, Progeria, Fabry disease, Von Hippel-Lindau disease, Sturge-Weber syndrome, and Mitochondrial Encephalopathy, Lactic Acidosis, Stroke-like episodes (MELAS). These genetic disorders primarily cause vascular complications that can lead to ischemic or hemorrhagic strokes in pediatric patients.[2,10]

In addition, Cerebral autosomal dominant arteriopathy with subcortical infarcts and leukoencephalopathy (CADASIL) is one of the most common inherited cerebral small vessel diseases (CSVD). About 12% of all strokes are caused by small artery occlusion, with less than 2% genetically confirmed as CADASIL.[11] CADASIL is caused by pathogenic mutations in the NOTCH3 gene produced by vascular smooth muscle cells (VSMCs), resulting in

the thickening of vascular walls and luminal stenosis in the small vessels of white matter and deep gray matter. This leads to the degeneration of VSMCs and the formation of granular deposits of N3ED immunoreactivity in the tunica media, accompanied by the accumulation of extracellular matrices, including collagen, clusterin, and laminin, in the tunica adventitia. This pathomechanism in CADASIL leads to loss of compliance and autoregulation in arterioles, causing cerebral infarction, white matter disease, microbleeds, and vascular dementia.[12]

Table 2. Primary risk factors for childhood stroke (28 days to 18 years[7]

Risk factor	Underlying etiology
Arteriopathies (intra and extracranial) (21-53%)	Moyamoya disease
	Sickle cell arteriopathy
	Post-varicella arteriopathy
	Arterial dissection
	Primary CNS vasculitis
	Para-/post infectious vasculitis
	Fibromuscular dysplasia
Cardiac (24-31%)	Congenital/acquired heart diseases
	Patent foramen ovale (PFO)
	Arrythmia
	Previous cardiac surgery/catheterization
	Endocarditis
	Other
Hypercoagulable state (13-28%)	Protein C deficiency
	Protein S deficiency
	Factor V Leiden mutation
	Prothrombin mutation
	Antithrombin III deficiency
	Increased factor VIII
	Hyperlipoproteinemia (alpha
	Lupus anticoagulant
Acute systemic conditions (9-22%)	Infectious diseases
	Sepsis
	Shock
	Acidosis/anoxia
Hemato-oncological (9-19%)	Sickle cell disease
	Hemolytic/iron deficiency anemia
	Hematological malignancy
	Solid extracranial tumors
Chronic head and neck disorders (9-10%)	Brain tumors
	Aneurysm/arterio-venous malformations
	Other cranial/neck tumors
	Migraine
Genetic/metabolic (1%)	Mitochondriopathy
	Fabry disease
	PHACE syndrome
	Trisomy 21
	Deficiency of adenosine deaminase 2
	Connective tissue disorder
Others (10%)	Previous brain surgery
	Trauma

CNS: Central Nervous System, PHACE: Posterior Fossa Brain Abnormalities, Hemangiomas, Arterial Anomalies, Cardiac Anomalies, and Eye Anomalies

Cerebral autosomal recessive arteriopathy with subcortical infarcts and leukoencephalopathy (CARASIL) or Meada syndrome is an uncommon genetic disorder that also causes CSVD. CARASIL is caused by mutations in the HtrA serine peptidase/protease 1 (HTRA1) gene, which suppresses typical transforming growth factor-beta (TGF-β) signaling, leading to vascular changes.[13]

Lastly, moyamoya disease (MMD) is a non-atherosclerotic and non-inflammatory chronic occlusive cerebrovascular disease characterized by stenosis of the intracranial arteries, primarily the distal internal carotid artery (ICA) and its branches. Commonly involved branches are the anterior cerebral artery (ACA) and middle cerebral artery (MCA), with the retina also potentially involved.[6] The term “moyamoya” refers to the formation of abnormal collateral vessels, known as rete mirabile, characterized by the appearance of hazy,, smoke-like vessels at the base of the skull, as detected through angiography.[14] These "puffs of smoke" form due to the sprouting of groups of vessels to compensate for progressive stenosis.[6] Steno-occlusive changes are usually bilateral, but unilateral involvement is also possible. MMD has a bimodal age distribution pattern, with peak onset at ages 5 and 40.[14]

Clinical Approach in Pediatric Stroke

For suspected or confirmed childhood stroke, a complete blood count with differential, basic biochemistry (including electrolytes, glucose, urea, and creatinine), and a coagulation screen (prothrombin time, activated partial thromboplastin time, and fibrinogen) should be performed.[3] In this case, several laboratory investigations were performed to identify possible systemic or metabolic contributors to the patient’s condition. The findings offer indirect clues, although they are nonspecific. Results showing hyperuricemia can raise suspicion of metabolic disorders or underlying renal involvement; however, this was clinically ruled out in this patient. Intermittent hyperglycemia could indicate glucose dysregulation or early-stage diabetes mellitus. Still, given the patient’s young age, the absence of sustained hyperglycemia, and no family history of diabetes, it is less likely to be a primary cause. However, it may still contribute to vascular vulnerability. A mildly elevated ESR may suggest low-grade vascular inflammation or chronic small vessel arteriopathy, consistent with CADASIL or CARASIL. The definitive diagnosis of CADASIL or CARASIL requires genetic testing, which remains the gold standard for diagnosis. However, access to such testing is limited in developing countries due to cost and availability constraints.

Children with suspected acute ischemic stroke should undergo urgent brain MRI, including axial

diffusion-weighted imaging (DWI), axial gradient echo for hemorrhage detection (such as susceptibility-weighted imaging (SWI)), axial fast spin echo or turbo spin echo T2, FLAIR (if over 1 year old), axial T1, and magnetic resonance angiography (MRA). If an urgent MRI is not possible, CT imaging, such as computed tomography angiography (CTA) and CT perfusion of the intracranial and neck vessels, can be an alternative. Additionally, echocardiography and ECG should be performed on children with suspected acute ischemic stroke.[3] In this patient, only a brain CT and MRI without contrast were performed, and no risk factors were found that could explain the underlying etiology of the stroke. The MRI revealed old infarct lesions, while the brain CT scan showed no definitive abnormalities. The patient did not undergo brain CT angiography or digital subtraction angiography (DSA); therefore, moyamoya disease could not be excluded.

Management of Pediatric Stroke

In patients with acute signs of ischemia, large vessel occlusion, and excluded intracranial hemorrhage, eligibility for interventions should be assessed. Intervention options include IV-tPA, endovascular intervention, and antithrombotic therapy. IV-tPA is a reperfusion therapy with indications for (i) administration within 4.5 hours of known symptom onset, (ii) ages 2 to 17 years, (iii) acute focal neurological deficit with a pedNIHSS score ≥4 to ≤24, and (iv) radiologically confirmed arterial stroke with absence of hemorrhage. After treatment, monitoring is necessary for bleeding risk, blood pressure, normal blood volume, normal glycemia, acid-base balance, and respiratory function. However, reperfusion therapies remain controversial. If treatment is sought ≥4.5 hours after onset, antithrombotic therapy, including anticoagulation and antiplatelet therapy, may be considered after excluding hemorrhage. In cases secondary to cardioembolism, treatment with low-molecular-weight heparin or vitamin K antagonists is recommended for a minimum of three months, and a minimum of six months in cases secondary to dissection. For cases not caused by cardioembolism or dissection, aspirin is recommended for a minimum of 2 years.[3,7] However, in this patient, clopidogrel was initiated at the previous hospital, and we decided to switch to cilostazol 100 mg once daily due to gastrointestinal side effects. The use of cilostazol instead of aspirin in cases of ischemic stroke, particularly in younger patients or those suspected to have small vessel arteriopathies such as CADASIL or CARASIL, can be considered based on its dual antiplatelet and vasodilatory effects.

Spasticity in the patient was treated with monthly Botulinum toxin (BoNT) injections. Spasticity is a common complication of stroke and is a sensorimotor system disorder caused by stretch reflex hyperexcitability and increased muscle tone.[15] Spasticity can be managed with several pharmacotherapies, including benzodiazepines, baclofen, dantrolene, and phenol. However, some medications often have side effects like muscle weakness and fatigue.[16] On the other hand, BoNT-A has demonstrated promising results with minor side effects and thus is currently considered the gold standard for treating focal spasticity.[17]

BoNT is a neurotoxin produced by the gram-positive, rod-shaped, anaerobic, spore-forming bacterium Clostridium botulinum, Clostridium butyricum, Clostridium barati, and Clostridium argentinensis.[18,19] Based on serological typing, there are seven types of BoNT (A through G), but only types A and B are used clinically.[18,19] BoNT-A is a key therapeutic agent in managing spasticity, including in pediatric populations. BoNT-A consists of two peptide chains with various amino acid sequences depending on the subtype of the BoNT, connected by a disulfide bridge. The heavy chain has a molecular weight of 100 kilodaltons. It consists of two domains: the C-terminal domain, which is responsible for binding to the receptor site, and the N-terminal domain, which is responsible for translocation. Meanwhile, the light chain has a molecular weight of 50 kilodaltons and consists of a single domain responsible for the catalytic breakdown of the target protein.[18] The heavy chain specifically binds to neurons to facilitate the toxin's entry, the translocation domain transports the light chain into the neuronal cell cytosol, and the enzymatically active light chain cleaves the soluble N-ethylmaleimide sensitive fusion attachment receptor (SNARE) proteins, blocking vesicle fusion at the inner surface of the cellular membrane, thereby disrupting neurotransmitter release. Ultimately, BoNT-A effectively blocks the release of acetylcholine at motor terminals.[20] As a result, patients typically experience improvement after receiving a BoNT-A injection.

The optimal dose of BoNT-A needs to be tailored to each patient, generally ranging from 50 to 600 units (U), as increasing the toxin dose can lead to a reduction in the Modified Ashworth Scale (MAS) score (Table 3).[21] However, in this patient, a low dose of 10 units of BoNT-A was administered based on several considerations, such as the focal and mild-to-moderate spasticity that allows a lower dose to target specific muscle groups adequately; the patient's high sensitivity to medications, as observed with antipsychotics, warranting caution to avoid overtreatment or toxicity; the clinical improvement demonstrated with the low dose of BoNT-A, with the MAS score improving from 2 to 1; and financial considerations.[3]

Table 3. Modified Ashworth Scale (MAS) to measure the grade of muscle spasticity[22]

Grade	Description
0	No increase in muscle tone
1	Slight increase in muscle tone, with a catch and release or minimal resistance at the end of the range of motion when the affected part(s) is moved in flexion or extension
1+	Slight increase in muscle tone, manifested as a catch, followed by minimal resistance through the remainder (less than half) of the range of motion.
2	A marked increase in muscle tone throughout most of the range of motion, but the affected part(s) are still easily moved.
3	A considerable increase in muscle tone and passive movement is a complex process.
4	Affected part(s) are rigid in flexion or extension.

The limitation of this case report primarily lies in financial and availability constraints, which significantly hindered the ability to thoroughly investigate and establish the underlying etiology, particularly the potential presence of congenital or inherited disorders. Conditions such as APS, CADASIL, CARASIL, and MMD remain possible contributors to this patient's presentation but have not been definitively ruled out due to limited access to advanced diagnostic modalities, such as genetic testing and vascular imaging. This challenge is common in many developing countries, where resource limitations frequently impede comprehensive diagnostic workups, especially for rare or complex pediatric neurological conditions.

Conclusion

Stroke can occur at any age and is a critical condition with high morbidity and mortality. Although it commonly affects adults, pediatric stroke can lead to lifelong disabilities, developmental disruptions, and long-term complications due to the immaturity of the brain and its higher metabolic demands. Various underlying causes and risk factors must be considered, including cardiac abnormalities, vasculopathies, coagulopathies, infections, genetic factors, and other potential congenital disorders. This case report highlights a pediatric stroke patient with no identified risk factors to explain the underlying etiology of her ischemic stroke. Several congenital disorders that may have contributed to the condition have not been ruled out, as we were unable to conduct the necessary investigations due to resource limitations. The patient is currently undergoing treatment aimed at preventing further stroke episodes and continues to receive botulinum toxin type A injections, which have led to noticeable improvements in spasticity and right-sided weakness. A comprehensive understanding of the etiology and management of neurovascular conditions in pediatric stroke is crucial for optimizing outcomes and minimizing long-term complications.

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