Scholars Journal of Applied Medical Sciences
Abbreviated Key Title: Sch J App Med Sci
ISSN 2347-954X (Print) | ISSN 2320-6691 (Online)
Journal homepage: https://saspublishers.com

OPEN ACCESS

Pediatrics and Neonatology

Perinatal Stroke in Two Neonates: Divergent Clinical Presentations from Early Seizures to Incidental Neuroimaging Detection

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DOI: https://doi.org/10.36347/sjams.2026.v14i06.020 | **Received:** 09.05.2026 | **Accepted:** 26.06.2026 | **Published:** 29.06.2026

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Abstract **Case Report**

Perinatal stroke is defined as a cerebrovascular event that occurs between the 20th week of gestation and 28 days after birth. Perinatal stroke is one of the leading causes of neurological morbidity in newborn infants. The presentation of stroke in newborn infants can range from symptomatic seizures to completely asymptomatic that are only diagnosed with stroke through neuroimaging studies. The presentation of stroke in newborn infants can be challenging to recognize due to the non-specificity of the signs and symptoms. Herein we reported two newborn infants who were diagnosed with perinatal stroke. The first is a full-term female who developed seizures at the third day of life with an infarction in the territory of the left middle cerebral artery with hemorrhagic transformation. While the second case is a male neonate who developed respiratory distress after delivery; stroke was incidentally found on cranial ultrasonography, subsequently cortical infarction with cerebral sinovenous thrombosis confirmed by CT scan, followed by MRI studies.

Keywords: Perinatal stroke; Neonatal arterial ischemic stroke; Neonatal hemorrhagic stroke; Cerebral sinovenous thrombosis; Neonatal seizures; Magnetic resonance imaging; Middle cerebral artery infarction.

INTRODUCTION

Perinatal stroke can be defined as a cerebrovascular event that occurs between 20 weeks of fetal life and 28 days after birth [1]. Perinatal stroke is one of the leading causes of neurological morbidity in newborn infants. The estimated prevalence of stroke at birth is between 1 in 1, 000 and 1 in 5, 000 live births [2, 3]. Perinatal stroke includes variant types such as neonatal arterial ischemic stroke (NAIS), neonatal hemorrhagic stroke (NHS), cerebral sinovenous thrombosis (CSVT), arterial presumed perinatal ischemic stroke (APPIS), periventricular infarction (PVI), and presumed perinatal hemorrhagic stroke (PPHS) [4]. NAIS is the most common form of perinatal stroke, accounting for 70-80% of all cases of perinatal stroke. The incidence of NAIS is 1 in 2, 300 to 1 in 5, 000 live births [5]. NAIS occurs more commonly in male than female neonates infants, and it is most common in neonates who are born at term [6]. Perinatal stroke is the leading cause of hemiplegic cerebral palsy in children, as well as the leading cause of epilepsy in infants and young children [7]. The presentation of perinatal stroke is variable. The hallmark clinical manifestation of NAIS is focal seizures, which present in 70-90% of neonates with NAIS. These seizures typically begin within the first three days of life [8]. Other clinical features can include abnormal tone, level of consciousness, breathing effort, or feeding behaviours [7]. Approximately 40% of neonates infants with perinatal stroke have no presenting symptoms; however, these neonates infants will exhibit neurological deficits during the first year of life [9, 10]. The known risk factors for perinatal stroke include conditions related to preganat mother, conditions within the fetus and conditions that occur during labor and delivery. Maternal risk factors for developing a perinatal stroke include nulliparity, diabetes mellitus, pre-eclampsia, autoimmune disorders, and thrombophilia. Fetal risk factors include birth asphyxia, congenital heart disease, and in utero infections. Risk factors during labor and delivery include abnormal fetal heart rate tracings, meconium-stained amniotic fluid, ruptured membranes, and cesarean delivery [11]. Thrombocytopenia is of one potential contributing factor [12, 13]. Diagnosis of perinatal stroke includes, CT scans and MRI of the brain. Craniocerebral ultrasound is typically the first imaging modality used in the neonatal intensive care unit(NICU), but has less sensitivity. CT scans are used to detect hemorrhage within the brain, but utilize ionizing radiation. MRI of the brain with diffusion-weighted imaging (DWI) is considered the gold-standard modality for detecting perinatal stroke. Additionally, MR angiography and MR venography can provide

information regarding the blood flow in the arteries and veins of the brain, respectively [14]. Perinatal stroke is a condition that is common in Saudi Arabia. Salih et al reported perinatal stroke as present in 22% of 104 pediatric stroke cases [15]. Additionally, Alfayez et al reported 32 cases of perinatal stroke in a neonatal intensive care unit in Riyadh, Saudi Arabia, between 2000 and 2013 [9]. Studies of Saudi neonates-infants with presumed perinatal stroke have discovered that many of those with stroke have hereditary conditions that contribute to the stroke [12].

CASE ONE

A female neonate infant who was born at 41 weeks of gestation through emergency cesarean delivery. Cesarean delivery was indicated due to a non-reactive cardiotocography trace obtained during labour. The rest of the maternal medical history was unremarkable; the mother was healthy during pregnancy. The newborn emerged at delivery, but had grade II meconium-stained amniotic fluid. Apgar scores were 9 at both the 1- and 5-minute marks after birth. On day three of life, the infant began to exhibit focal seizures limited to the lower extremities. The infant's blood glucose level was within the normal range; however, the calcium level was low. Following administration of IV calcium, the seizures resolved. CT of the brain revealed a hypodense area within the left parietal, temporal, and occipital lobes, as well as a midline shift, indicative of a hemorrhage [Figure 1]. The infant was transferred from peripheral hospital to our unit. The infant was admitted with vital signs of HR 140 bpm, BP 69/40, RR 40-55 breaths per minute, Temp of 35. 1°C, and O2 saturation of 95% on room air. The infant's birth weight was 4, 300g, length was 51cm, and the head circumference was 34. 5cm. The infant was treated with phenobarbital to control the seizures. Culture of blood and CSF grew no pathogenic organisms, though antibiotics were administered for 7 days and later discontinued. MRI of the brain revealed a middle cerebral artery (MCA) infarction with hemorrhagic transformation [Figure 2]. On physical examination**;** The neonate infant was not dysmorphic, not in distress. The head was of normal shape with flat and soft anterior fontanelles. The pupils were equal in size and reacted normally to light, with normal muscle tone, alert and exhibited all of the neonatal reflexes. The nasal passages were clear and patent. The respiratory rate was rapid, but regular. The chest auscultation was normal. The heart sounds were audible in all auscultatory areas, no murmur was detected. The femoral pulses were strong and the capillary refill time was normal. The abdomen was soft with no masses. The external genitalia were appropriate for the infant's sex. There were no skin lesions. Laboratory investigations and neuroimaging are detailed in Table 1. The neonate infant was discharged from the hospital on postnatal day 7; the infant is being followed up with the high-risk and neurology clinics.

CASE TWO

A male neonate was born at 37 weeks of gestation to a 28-year-old mother with no significant antenatal medical history. The Apgar scores at one and five minutes of life were 7 and 9, respectively. Shortly after delivery from the mother, the neonate developed respiratory distress that required admission into the neonatal intensive care unit (NICU). The neonate was intubated and treated with surfactant for respiratory distress that was corrected after 24 hours. On the third day of life, a selective cranial ultrasound of the neonate revealed a hemorrhage outside of the ventricular zone. A CT scan showed a hypodense lesion in the temporal region of the brain [Figure 3]. The lesion was evaluated with MRI, which revealed an infarction of the occipital and parietal cortex with restricted diffusion. An MRI of the venous system of the brain revealed reduced flow within the superior sagittal sinus [Figure 4]. Upon admission heart rate 120-155 beats per minute, blood pressure: 60/38 mmHg-MAP (45mmHg), respiratory rate 55-70 per minute, temperatures 37, Oxygen saturation 98%, birth weight 3000grams, birth length 48. 5cm, head circumference 34. 7 cm. On examination; The neonate was somewhat distressed but otherwise non-dysmorphic. The skull was well-formed with flat and soft anterior fontanelles. The conjunctivae were normal with a normal red reflex. Both nostrils were patent. The neonate was alert and calm with normal reflexes. There were no abnormal movements observed. The respiratory effort revealed retractions and tachypnoea. The heart sounds were audible, and the femoral pulses were intact with normal refill time. The abdomen was soft, non-tender, and non-distended with normal bowel sounds. The external genitalia were normal for male with a patent anus. The back, spine, and extremities were normal. Bilateral hip laxity was noted. There were no skin stigmata. The blood investigations and neuroimaging are detailed in Table 1. The neonate did not experience any seizures during hospital admission. The neonate was taken off the NICU on day 5 of life and released into the care of his mother with follow-up appointments made with outpatient multi clinics.

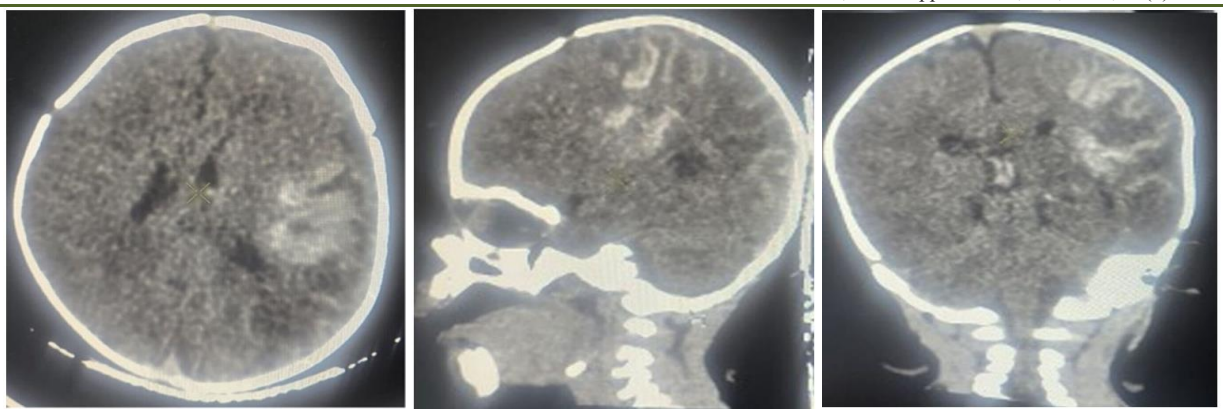

Figure 1: Axial, sagittal, and coronal sections: large left cortical temporo-occipital-parietal hypodense area with cortical hyperdensity, effacement of cortical sulci, and compression of the ipsilateral ventricle, consistent with acute hemorrhagic infarction in the territory of the left middle cerebral artery

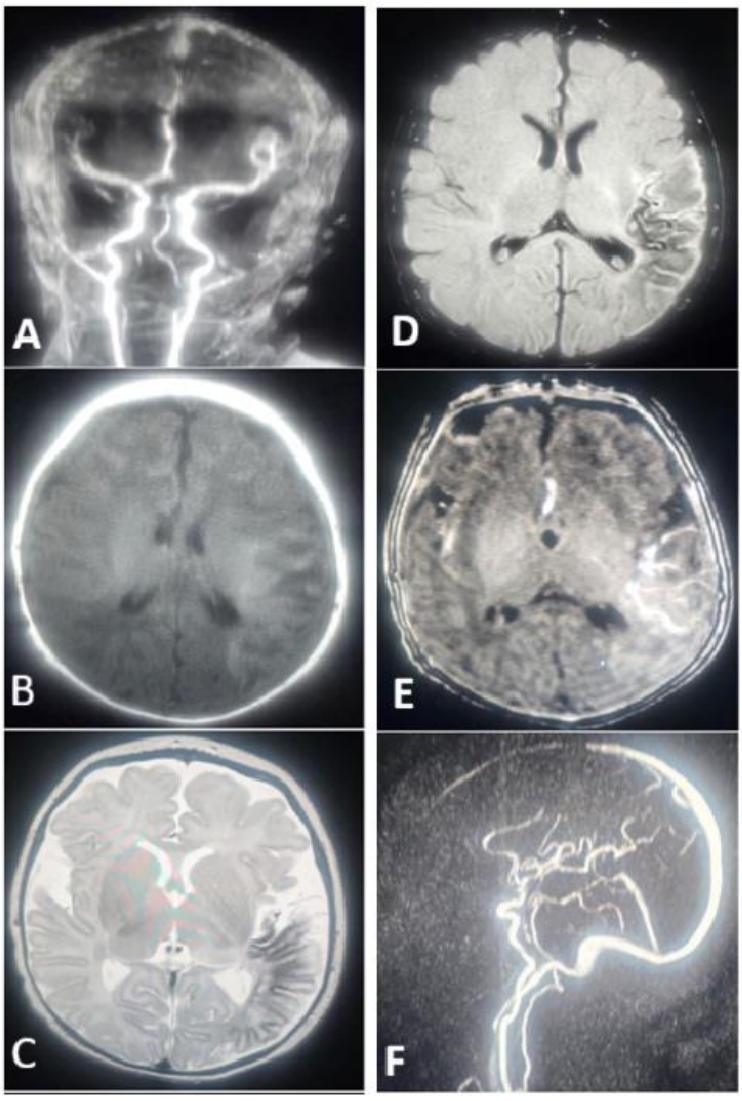

Figure 2:
(A) T1: left cortical hypointense area with peripheral hyperintensity.
(B) T2: left cortical hyperintense area with a central hypointense signal.
(C) FLAIR: left cortical hypointense area with peripheral hyperintensity.
(D) DWI: cortical restricted diffusion.
(E) MRV: normal signal from all dural venous sinuses.
(F) MRA: left collateral vessel formation.

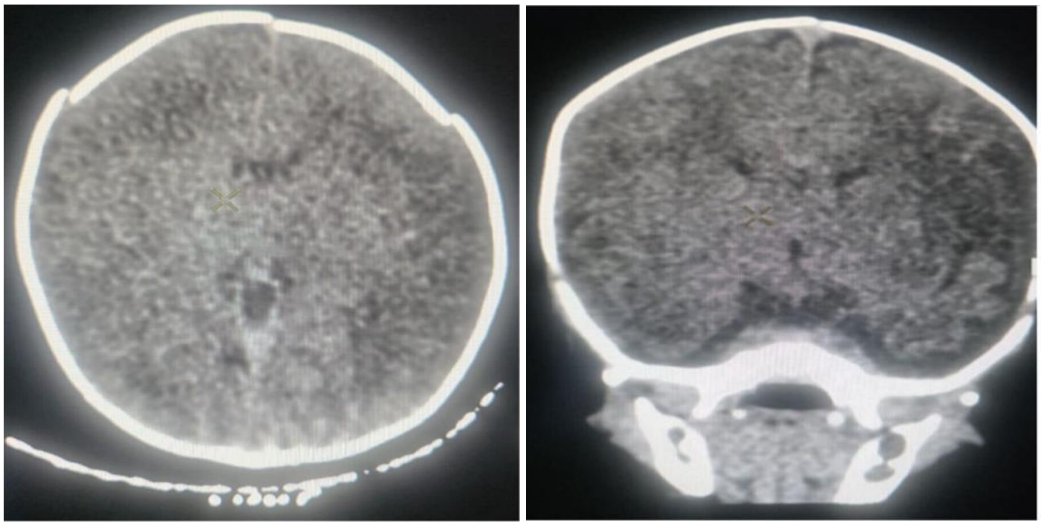

Figure 3: Axial and coronal sections: cortical left temporal hypodense area with effacement of cortical sulci and mild compression of the ipsilateral ventricle, consistent with acute cortical infarction

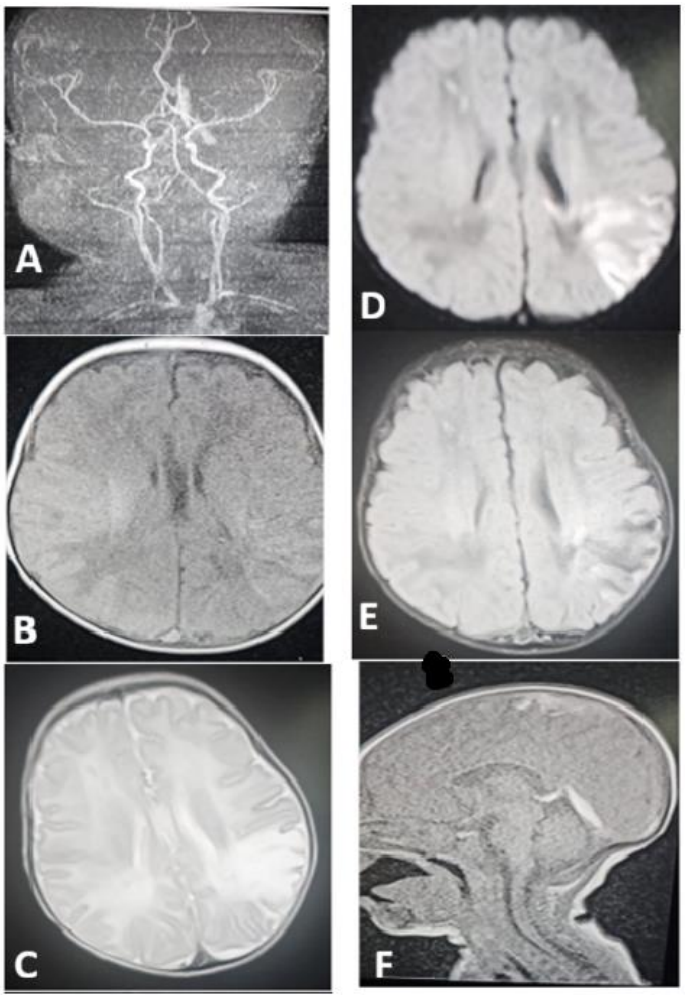

Figure 4:
(A) T1: left occipital hypointense area with cortical hyperintensity.
(B) T2: cortical and occipital hyperintense signal.
(C) DWI: restricted diffusion at the left cortical and occipitoparietal region.
(D) FLAIR: left occipital hyperintense signal.
(E) MRV: loss of flow signal at the superior sagittal sinus.
(F) MRA: normal study.

Table 1: Laboratory Investigations and Neuroimaging Findings

Investigation	Case 1 Results	Case 2 Results
Full blood count	All within normal ranges	Within normal ranges
Renal profile	Within normal limits	Normal
Liver profile	Normal	Normal
Coagulation profile	Normal	Normal
Blood culture	No growth	No growth
C-reactive protein	Negative	Negative
Thrombophilia workup	Negative	Negative
Metabolic screening	Negative	Negative
Echocardiography	Normal study	Normal study
Cranial ultrasound	Unremarkable	Suspicious hemorrhagic lesion outside the ventricular zone
CT scan	(Figure 1) Axial, sagittal, and coronal sections: large left cortical temporo-occipital-parietal hypodense area with cortical hyperdensity, effacement of cortical sulci, and compression of the ipsilateral ventricle, consistent with acute hemorrhagic infarction in the territory of the left middle cerebral artery.	(Figure 3) Axial and coronal sections: cortical left temporal hypodense area with effacement of cortical sulci and mild compression of the ipsilateral ventricle, consistent with acute cortical infarction.
Brain MRI/MRA/MRV	(Figure 2) (A) T1: left cortical hypointense area with peripheral hyperintensity. (B) T2: left cortical hyperintense area with a central hypointense signal. (C) FLAIR: left cortical hypointense area with peripheral hyperintensity. (D) DWI: cortical restricted diffusion. (E) MRV: normal signal from all dural venous sinuses. (F) MRA: left collateral vessel formation.	(Figure 4) (A) T1: left occipital hypointense area with cortical hyperintensity. (B) T2: cortical and occipital hyperintense signal. (C) DWI: restricted diffusion at the left cortical and occipitoparietal region. (D) FLAIR: left occipital hyperintense signal. (E) MRV: loss of flow signal at the superior sagittal sinus. (F) MRA: normal study.
CFM	Burst suppression for 24 hours with three seizure episodes, followed by discontinuous low-voltage activity, subsequently normalizing.	Normal continuous voltage with preserved sleep-wake cycles; no epileptiform discharges detected.

DISCUSSION

Perinatal stroke encompasses disorders of the central nervous system with cerebrovascular event between 20 weeks of gestation and 28 days of age after birth. Beyond being the leading cause of neurological injuries in newborns, perinatal stroke also leads to 20% of all cases of hemiplegic cerebral palsy and epilepsy in infancy and early childhood [7]. Strokes can present in a variety of ways in these age groups, from seizures alone to silent stroke with no presentation at all. The first case to be presented is that of a neonate who experienced focal clonic seizures on the third day of life. Such presentations are in keeping with the presentation of newborns with NAIS. NAIS is characterized by seizures as its hallmark presentation, and 70 to 90% of patients with the syndrome experience these seizures within the first three days of life of the newborn [8]. An MRI of the neonate's brain revealed both an infarction within the area supplied by the left MCA, as well as signs of hemorrhagic transformation of that infarction. For this infant, phenobarbital was administered as the treatment of choice for newborns who suffer from seizures due to perinatal stroke [1]. In contrast, the second neonate presented with no seizures of any kind during the length of the hospital stay. The stroke in this neonate was discovered during a cranial ultrasound which indicated owing to intubation and ventilation for respiratory distress. It is estimated that 40% of perinatal strokes are clinically silent during the period of the newborn but manifest as neurological deficits after the age of one year [9, 10]. Additionally, an MRV of the neonate revealed reduced blood flow within the superior sagittal sinus. This led to the diagnosis of CSVT in the neonate. The incidence of CSVT in neonates is between 1 and 12 per 100, 000 live births. In the majority of cases, the blood clots that form within the venous systems of these newborns are the result of the hypercoagulable state of the mother during the peripartum period and labor [12, 13]. Additionally, those same blood vessels may suffer

from thrombosis as a result of the physical pressure of labor. The presence of CSVT is an indication to perform an MRV in any neonate who is suspected of suffering from stroke. In both cases, the neonates were managed conservatively in the NICU. Additionally, no tests revealed that either neonate suffered from thrombophilia in their system or any metabolic issues with their blood. These findings are in accordance with numerous research studies that have shown that the majority of perinatal strokes have no identifiable cause [9]. An MRI of the brains of both of these infants was crucial to the diagnosis of stroke in each. While cranial ultrasound was utilized to diagnose stroke in neonate two, cranial ultrasound is not as sensitive to detecting strokes in the cerebral cortex. For neonate one, the cranial ultrasound of the infant's brain was normal, but an MRA and MRI of the brain revealed the presence of a stroke in the brain. An MRI is the imaging modality of choice for neonates infants and young children with stroke [14]. Additionally, the MRA allowed for assessment of the formation of collateral blood vessels to the area of stroke in the brain of the infant. Additionally, the MRV allowed for the detection of the venous thrombosis in neonate two. These various radiographic sequences are crucial to the diagnosis of stroke in neonates. The significance of these cases to the Kingdom of Saudi Arabia is that the Kingdom experiences high rates of stroke within its population of pediatric patients [9, 15]. Additionally, genetic analyses of infants with perinatal stroke in the Kingdom of Saudi Arabia have revealed the contribution of a single genetic disorder to the development of stroke in those infants [12]. These factors lead to the assumption of a higher rate of stroke among neonates in the Kingdom of Saudi Arabia due to these genetic factors. Thus, physicians within the Kingdom of Saudi Arabia should be highly aware of stroke among neonates. The Cerebral Function Monitoring (CFM) for each of these neonates' infants revealed electrographic seizures in the first infant, which resolved over time; however, the background signals on the second neonate infant with CFM were normal, indicating that the neonate did not suffer from seizures. These background signals for the neonates during the perinatal period have been associated with the neurological outcome of the infants after they suffer from stroke. For each of these neonates infants, long-term follow-up is essential to determine the effect that the stroke has on the brain's ability to perform its normal functions. Additionally, early intervention in these infants will yield the best possible outcomes [1, 7]. The care for each of these infants will require a multidisciplinary team of physicians, therapists, nurses, and any other staff members required to provide optimal care for each of these infants with stroke.

Conclusion

Perinatal stroke has a significant impact on the neurological function of neonates infants during the perinatal period. Some neonates infants may only display seizures as a symptom of stroke. For others, however, the neonates infants will be entirely asymptomatic. In either case, an MRI of the brain of neonates infants with stroke is essential. The MRI of the brain will include diffusion-weighted, angiographic, and venographic sequences to determine the cause of the stroke in those infants with perinatal stroke. The involvement of a multidisciplinary team to care for the neonates infants with perinatal stroke will ensure the provision of care that will lead to the best outcome for those infants. Additionally, the long-term follow-up of the neurological function of these infants will ensure that any issues are detected early in their lives, allowing for treatment to be provided to those infants whose neurological function is affected by stroke.

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