Broad Cerebral Infarct in a Term Neonate

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Abstract

Neonatal cerebral infarct is a very rare entity and such infarcts could regress as a transient event or result in severe neurological injuries such as hemiplegic cerebral palsy. Here, we report a case of cerebral infarct in a term male infant presenting with convulsions within the first day of life. Difficulty at birth might be one of the uncommon reasons of the infarcts. In this case, dystocia is supposed to be one of the associated events. The neurological deterioration was transient and no neurological deficit was noticed at 7 months after birth. The majority of neonates with large infarcts have a poor prognosis. Early imaging in this population may lead to prompt diagnosis, timely neuroprotection, rehabilitation, and improved outcome.

Keywords
- cerebral infarct
- newborn
- birth trauma
- neurological deficit

Introduction

Neonatal cerebral infarction or stroke causes severe disorganization of gray and/or white matter structures of the brain and can be caused by embolic, thrombotic, or ischemic events.[1] The advances in neonatal imaging in infants with abnormal neurological signs have increased the awareness of the wide spectrum of these lesions.[2,3] Although cardiac, hematological, metabolic, and primary vascular abnormalities account for most cases of neonatal stroke, the etiology remains unclear in nearly half of the patients.[4] The middle cerebral artery (MCA) is most affected in adult stroke and the left MCA is three to four times more frequently affected than the right one. Infants with the involvement of anterior cerebral artery (ACA) or posterior cerebral artery (PCA) may be asymptomatic and therefore undiagnosed.[5] Cerebral infarct due to head trauma is rarely reported in childhood in the literature and upon to our knowledge, there are only few case reports demonstrating cerebral infarct after birth trauma in the neonatal period.[6–8] The onset of convulsions in the first day of life is usually the first clinical sign leading to diagnostic investigations.[9]

Here, we report a term newborn who presented with convulsions on the first day of life and the diagnostic imaging techniques showed extensive, unilateral hemispheric infarct due to birth trauma.

Case Report

After an uneventful pregnancy, this first born boy of a 26-year-old mother was delivered at term (38 + 4/7 weeks) via C-section with a birth weight of 3,560 g. Considerable difficulty was met with while freeing head at the level of forehead vertex. His Apgar scores were 8 and 9 at 1st and 5th minutes, respectively. Evaluation of placenta reported was reported as normal. At first physical examination, his head and neck were deviated to right and no mass was palpable in the head and neck. Chest roentgenogram showed no clavicle fractures. Eight hours after the birth, he presented with a shrill cry and focal clonic seizures involved the right arm and leg.

An ultrasound (US) of the head revealed corticosubcortical edematous aspect in the left cerebral hemisphere and a 13-mm shift of midline structures to the right, while the computed tomography (CT) showed diffuse hypodensity in the left hemisphere except for the territory of the anterior choroidal artery and ACA, including basal ganglia, consistent with acute cerebral infarct. Also, a cephalohematoma was seen on the left parietal area (►**Fig. 1A, B**).

The lumens of bilateral common, internal and external carotid arteries, were reported as patent in Doppler US. Afterward diffusion-weighted imaging (DWI) was performed and diffusion limitations congruent with acute infarct in the

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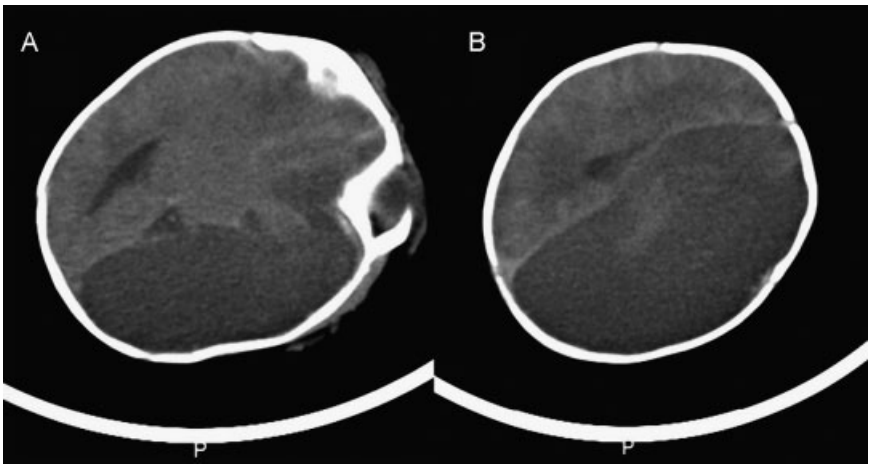

Fig. 1 (A, B) Axial computed tomogram image showing diffuse hypodensity of left hemisphere except the territory of anterior choroidal artery and anterior cerebral artery, including basal ganglia, consistent with acute cerebral infarct. Also, cephalhematoma is seen over the left parietal area.

territory of left MCA, left basal ganglia, and focal areas of right parietal lobe were demonstrated (►**Fig. 2A, B**). Cranial 3 T magnetic resonance (MR) angiography and MR spectroscopy findings revealed an acute cerebral infarct in the left MCA, ACA's superior territory, right parietal lobe's PCA territory, and cephalohematoma in the left parietal area. Disorganization considered to be a thrombus was noted in the left MCA's M1 segment (►**Fig. 3A, B**). Fundoscopic examination was normal. Due to the convulsions, phenobarbital therapy was started. Torticollis disappeared and tone was normal after phenobarbital therapy. Continuous multichannel video electroencephalography initiated at 11 hours after birth showed suppression of the background activity over the left cerebral hemisphere with no active epileptiform activity. Pediatric neurologist reported low amplitude fast background activity over the left hemisphere but relatively high amplitude slow wave activity over the frontal part of the right hemisphere. The patient had clinically apparent seizures only before phenobarbital therapy with resolution thereafter. Due to suspicion for thrombophilia studies for protein C activity, activated protein C resistance, factor V Leiden mutation, antithrombin III activity, homocysteine, and lipoprotein a levels were performed and were reported to be normal. D-dimer was 1,628.97 µg FEUa (fibrinogen equivalent units)/mL. Pediatric cardiologists reported normal physical and echocardiographic findings on examination of the heart. Abdominal US and Doppler US showed no thrombus or embolus.

The constellation of these clinical and radiological findings was thought to be suggestive of stroke due to birth trauma. At the follow-up visit of the patient, it was reported that the convulsions did not recur. His neurological examination improved. At postnatal day 8, the patient was

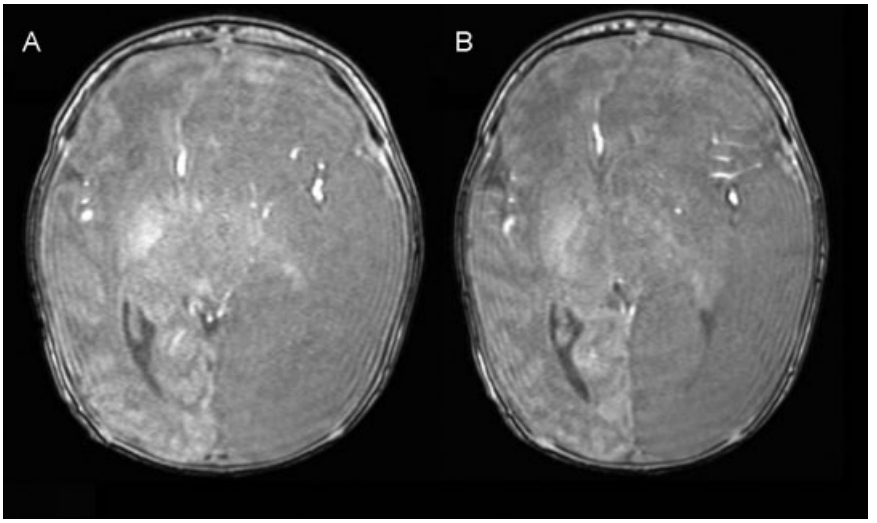

Fig. 2 (A, B) Axial diffusion-weighted image demonstrating diffusion limitations congruent with acute infarct in the territory of left cerebral artery, left basal ganglia, and focal areas of right parietal lobe.

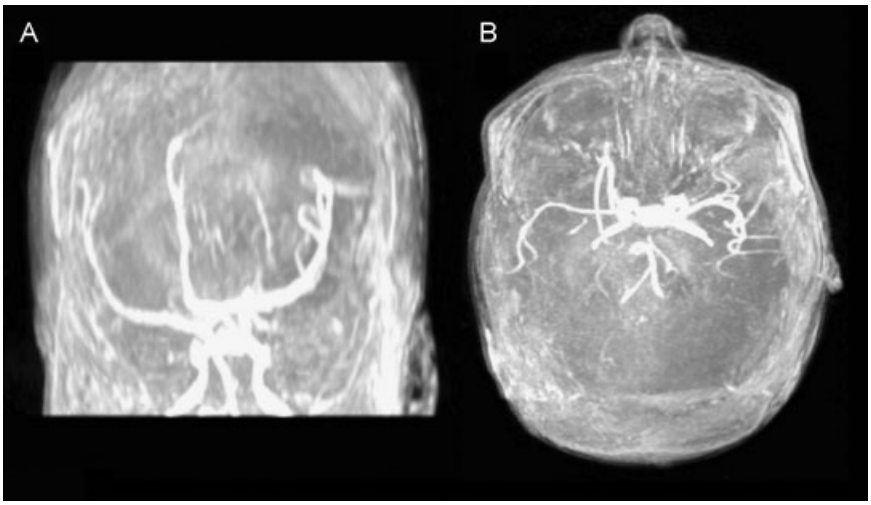

Fig. 3 (A, B) Coronal and axial magnetic resonance angiography images indicating disorganization, considered to be a thrombus, in the left middle cerebral artery's M1 segment.

discharged as he was feeding orally and there were no marked abnormalities on physical examination. The patient is currently 7 months of age and his neurological examination is reported to be normal.

Discussion

Perinatal stroke is a rare entity but a common cause of lifelong neurological disability.[3] Studies of neonatal arterial ischemic stroke (AIS) are limited as a result of limited sample sizes and heterogeneous populations. Kirton et al[3] evaluated newborns with AIS and provided an overview of clinical presentations, potential risk factors, laboratory investigations, treatment methods, and prognosis. Several risk factors have been identified, but their precise roles in causing stroke are not well understood.[9] Infarction as a result of birth injury is rare; direct trauma to an intracranial vessel, compression and vasospasm due to subdural or subarachnoid hematoma, stretch injuries of the arteries supplying the brain, and compression of the posterior circulation due to uncal herniation are some of the rare causes of cerebral infarct.[10] The most common clinical presentation is neonatal seizures. Diffuse neurological signs such as hypotonia or depressed level of consciousness may also be the presenting features. Less frequently, focal neurological signs such as lateralizing hemiparesis may be present. Nonspecific systemic findings included respiratory and feeding difficulties may be apparent. The most common time of presentation time is the first week of life.[3] Our patient's presentation was with convulsions and abnormal posture within the first day of life.

Perinatal AIS is generally asymptomatic and co-occurrence with an acute neonatal illness is frequent, including dehydration, fever, sepsis, acidosis, and meningitis. In children who have a delayed diagnosis the most common presenting symptom was reported to be hemiplegia.[3] Perinatal hypoxia was defined as the most common risk factor in neonates with complicated birth histories. Reported risk factors for perinatal AIS include primiparity, infertility, chorioamnionitis, maternal fever ($> 38°C$) during delivery, prolonged rupture of membranes, pre-eclampsia, cord abnormalities, gestational diabetes, fetal heart rate decelerations, prolonged second stage of labor, intrauterine growth retardation and emergency caesarean section, low Apgar scores (≤ 3 and <7, at 1st and 5th minutes, respectively), umbilical artery pH < 7.10, hypoglycemia <2.0 mmol/L, and early-onset sepsis/meningitis.[11–13] Maternal smoking before and during pregnancy was found to be associated with an increase in the risk of perinatal AIS.[11] Our patient had none of these risk factors. Perinatal AIS is seen mostly after spontaneous vaginal delivery with emergent cesarean section being the second most common mode of delivery that predisposes to this condition. Apgar scores were usually noted as normal. Significant early neonatal resuscitation (assisted ventilation, chest compressions, intubation, and medications) was documented in only a minority of the cases.[3] There have been reports of neonates who were delivered via elective cesarean section, with normal Apgar scores who developed a perinatal AIS.

Noninvasive vascular imaging is now routinely recommended by current pediatric stroke consensus guidelines. Head US is generally the first screening imaging method used in neonates suspected with cerebral events. Nevertheless, US may skip peripheral ischemic infarct and it does not clearly define the location or the size of an infarct.[14] Cerebral CT scan and MR imaging are major methods to help for making the diagnosis. We performed US as a first-line method of imaging that revealed corticosubcortical edematous aspect in the left cerebral hemisphere and a 13-mm right shift of the midline structures, while the CT showed diffuse hypodensity of left hemisphere except the territory of anterior choroidal artery and ACA, including basal ganglia, consistent with acute cerebral infarct. Also, cephalhematoma was seen on the left parietal area. DWI is the modality of choice to diagnose infarction in its early stages. DWI also provides prediction of the patient's motor outcome. This imaging method helps caregivers make important decisions regarding long-term neurorehabilitation intervention, which is critical to perinatal

AIS management.[4,5] Our patient's DWI showed diffusion limitations congruent with acute infarct in the territory of left cerebral artery, left basal ganglia, and focal areas of right parietal lobe. Our patient's MR angiography and MR spectroscopy findings revealed acute cerebral infarct in the left MCA, ACA's superior territory and right parietal lobe's PCA territory, and cephalohematoma in left parietal area, disorganization considered as thrombus at left MCA's M1 segment accompanied these findings. These findings were compatible with the current literature that the left MCA is the most common artery to be involved in perinatal AIS.[15]

Recanalization of cerebral arteries might occur quickly, therefore with most imaging performed several days after birth, occlusions might often not be evident.

Awareness of the neonatal stroke after traumatic birth and early diagnosis may play a significant role in timely administration of neuroprotective strategies such as therapeutic hypothermia. Therapeutic hypothermia is only an experimental treatment and offers potentially beneficial neuroprotection in patients with stroke.[16]

Our patient had a history of difficult delivery, difficulty while freeing head at the level of forehead-vertex. As laboratory evaluation for bleeding diathesis or thrombophilic disorders were normal, birth trauma was suggested to be the causative factor.

Supportive therapy and symptomatic treatment constitute the core treatment strategies in the acute stage of neonatal cerebral infarction. Those babies with large infarction involving cerebral hemisphere, thalamus, and basal ganglia tend to have a poor prognosis.

Westmacott et al[17] suggest that children with unilateral neonatal stroke, particularly males, are at increased risk for emerging deficits in higher level cognitive skills during the school years. Visual field problems are not diagnosed until school age when reading problems might develop. Thus continued follow-up of these children is needed, even those seemingly unaffected as toddlers or preschoolers.

In conclusion, neonatal cerebral infarct due to birth trauma is a very rare entity and long-term results are variable. In our case, neurological development was normal and the infarct caused no permanent defects till the child reached 7 months of age. However, cerebral infarcts must be kept in mind after difficult births and birth traumas as an important cause of poor neurological prognosis. Even when neurological development is assessed as normal, children must be followed up over the long term because deficits could be apparent as late as the early school years.

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