CLINICAL CASE REPORT

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Vehicle-associated closed trauma-induced stroke in a 27-day-old girl

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Key words: *perinatal stroke; traumatic stroke.*

Summary. *Birth trauma, but not postnatal trauma, has been recognized as a cause of cerebral infarction in newborns. We report a case of cerebral infarction in a 27-day-old girl after a car accident.*

During the car accident, the child was properly restrained to the child's safety seat. The patient was admitted to the hospital for observation because of pronounced irritability. There were no focal neurological symptoms on admission. Twenty-eight hours after the accident, the child developed focal tonic-clonic seizures and mild right-sided hemiparesis. The seizures were successfully treated with phenobarbital at a dose of 30 mg per day. Computed tomography and magnetic resonance imagining performed on the second and third days after the accident, respectively, showed subdural hemorrhage in the occipital regions and cerebral ischemia in the left parieto-occipital region. Control imaging 10 days later showed signs of reperfusion.

Persistent child irritability after head trauma is one of the indicating factors for performing an emergency computed tomography scan of the head.

Introduction

Perinatal arterial ischemic stroke (AIS) is a cerebrovascular event that occurs between 20 weeks of gestation and 28 days of postnatal age, with pathological or radiological evidence of focal arterial infarction of the brain (1). Childhood AIS occurs in children between 30 days and 18 years of life (2).

Clinical symptoms and triggering factors for perinatal AIS differ from childhood AIS (3). The main clinical features in the neonatal period are seizures, apnea, and depressed level of alertness (1, 3, 4). Hemiplegia, aphasia, or altered level of consciousness are common features of childhood AIS (3). Many antenatal, intranatal, and postnatal events may predispose perinatal ischemic stroke (5). Postnatal events predisposing cerebral infarction include paradoxical thromboembolism associated with cyanotic congenital heart disease, portal vein thrombosis, or iatrogenic vascular catheter-related thrombosis (5). Postnatal head injury, reported as one of them, has not been described in association with perinatal AIS.

Case report

A previously healthy 27-day-old female infant from a third uneventful pregnancy and delivery was admitted to the Department of Neurology, Children's Clinic of Tartu University Hospital, after a car accident. During the accident, the car ran off the road, hit a tree, and turned on the roof. The patient was properly restrained in a child's safety seat in the front seat. Initial examination of the child by a pediatrician revealed nothing remarkable except for irritability with temporary calm periods. There was no evidence of skin or other external injuries to the skull. However, due to irritability, restlessness, and young age, the infant was admitted to the hospital for further observation. After a few hours, the irritability abated, and the child's caretaker considered her behavior usual and unremarkable.

Next morning (24 hours after the accident), the patient was examined by a pediatric neurologist. The child acted normally, and no focal neurological signs were found. Fundoscopic examination revealed no

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pathology. Neurosonography and abdominal ultrasound were without pathology. About 47 hours after the accident, a focal tonic-clonic seizure in the right hand and mouth with deviation of the eyes and head to the right occurred. The seizure resolved spontaneously within a few minutes. An additional interview of the child's caretaker showed that during the previous day, namely, 28 and 36 hours after the accident, the child had had similar seizures. Emergency brain computed tomography (CT, Somatom Volume Access, Siemens, 2001) was performed, and subdural hematomas 2–3 mm in size in the occipital region, hemorrhagic focal cortical contusions 3–4 mm in size in the left temporal lobe, and a large edematous area in the left occipital-parietal region were found (Fig. 1). The edematous area was interpreted as a traumatic edema or an emerging posterior cerebral artery ischemic stroke.

During the same day, 3 more episodes of seizures occurred. Intravenous phenobarbital at a dose of 5 mg/kg was administered. Electroencephalogram (EEG), performed on the same day, showed continuous focal epileptic activity in the left hemisphere (Fig. 2). Regular treatment with oral phenobarbital was initiated. Seizures recurred twice during the following two days. At the same time, neurological examination revealed mild right-sided hemiparesis.

A control magnetic resonance imaging (MRI; Magnetom Symphony 1.5 T, Siemens, 2002) scan of the brain was performed on days 4 and 10 after the accident (Fig. 1). In addition to CT findings, the first MRI scan showed a subarachnoid hemorrhage and a moderate edema in the left temporo-parieto-occipital region. MRI scan with diffusion-weighted imaging on day 10 showed a clearly bordered region with features of ischemic injury and cortical reperfusion signs in parietal-occipital and posterior parts of the temporal lobe supplied by the left posterior cerebral artery.

Heart ultrasound investigation revealed a patent foramen ovale 4 mm in diameter. Thrombophilia screening did not reveal any inherited prothrombotic state. Repeated EEG on day 8 showed no epileptic activity. The patient was discharged from the hospital on day 14 with oral phenobarbital (7.5 mg/kg/day).

One and a half months later, the infant was readmitted for investigations and rehabilitation. Neurological examination showed mild right-sided hemiparesis. Control MRI showed a decrease in the volume of brain parenchyma in the region of the posterior cerebral artery and compensatory dilatation of the occipital horn of the left lateral ventricle (Fig. 1). Neuropsychological development was estimated as near normal except for relatively poor head control on pulling to the sitting position.

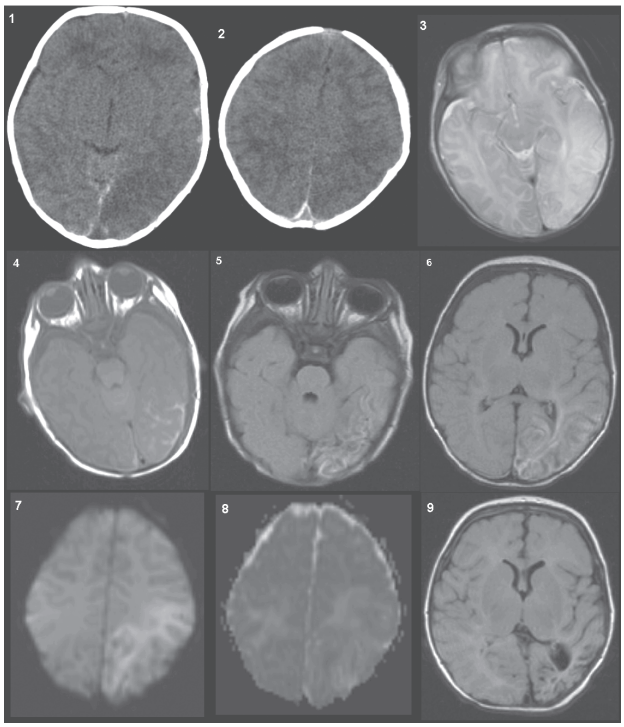

Fig. 1. Neuroradiological images of a 27-day-old patient with traumatic stroke

Initial CT images (1 and 2) show bilateral subdural hematomas in the occipital region, hemorrhagic focal cortical contusions in left temporal lobe, and a large hypodense area in the left occipital-parietal region. A follow-up T2-weighted magnetic resonance tomography image (3) on the second day shows a high-signal lesion in the left parieto-occipital region, and T1-weighted image (4) shows subarachnoid hemorrhage in the same region. Follow-up fluid-attenuated inversion recovery images (5 and 6), diffusion-weighted imaging (7), and corresponding apparent diffusion coefficient map (8) obtained 10 days after the accident show ischemic injury in the left parieto-occipital region. A follow-up fluid-attenuated inversion recovery image (9) obtained 1.5 months after reveals a decrease in the volume of brain parenchyma corresponding to the infarct area and compensatory dilatation of the occipital horn of the left lateral ventricle.

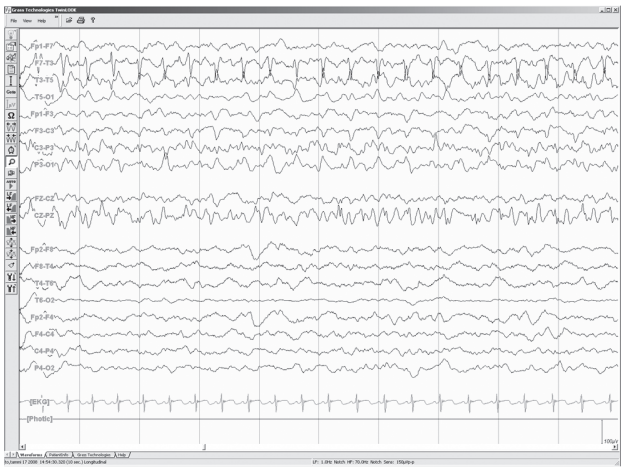

Fig. 2. Electroencephalography of a 27-day-old-patient with traumatic stroke

Electroencephalography with a sensitivity of 150 V; filters: LF 1.0 Hz notch; HF 70 Hz notch, montage longitudinal. Epileptic activity over the left hemisphere.

Discussion

We describe an unusual case of perinatal stroke. Firstly, although stroke in newborns aged up to 30 days has been classified as perinatal stroke, most cases occur within the first week of life and are closely related to ante- or intranatal factors (1, 4). Secondly, perinatal ischemic stroke has been associated with head trauma, but only when the trauma is related to birth (6, 7). Therefore, our case of cerebral ischemia in a nearly one-month-old girl caused by vehicle-related trauma may belong more properly to the childhood stroke.

Trauma is a known risk factor for childhood ischemic stroke, and blunt trauma to the posterior pharynx, cervical spine rotation/dislocation, and dissection have been suggested as a possible underlying mechanism (3). MRI angiography scanning was not performed for our patient because of technical reasons. Arterial dissection may account for up to 20% of strokes in children and adolescents (3, 8) and should be actively excluded, particularly when there is a history of trauma.

There are descriptions of a series of young children (ages 1–7 years) with acute hemiparesis caused by striatocapsular infarction following mild head trauma (9, 10). The proposed mechanism is the disruption of the lenticulostriate branches of the cerebral arteries between the mobile extracerebral portion and the fixed intracerebral portion (9). In our case, ischemic lesion was located in the left parieto-occipital region in the supply area of the left posterior cerebral artery. The first CT scan of our patient revealed a large edematous area in the left occipital-parietal region. Occlusion of the posterior cerebral artery after medial temporal lobe (uncal) herniation due to intracranial hypertension, e.g., edema, is one of the most recognized mechanisms leading to posttraumatic cerebral infarction (7, 11).

In light of the above-mentioned motor vehicle accident, any direct head trauma was unlikely in our patient, and only forces of acceleration should be considered as a source of injury. There are no reports concerning such specific accidents in children associated with this type of injury. According to the study by Muszynski et al., the likelihood of head injury for infants properly restrained in the child safety seat is extremely low (12). The risk of sustaining no head injury is 92.8% for restrained infants compared with 15.2% for unrestrained infants. Moreover, unrestrained infants have a 7% risk of moderate-to-maximum head injury compared with only 0.5% for properly restrained infants. It is obvious that the probability of intracranial injury depends also on the direction and rate of forces involved in every particular car accident.

It is important to stress that clinical symptoms in neonatal stroke are often nonspecific, e.g., muscular hypotonia, apnea, or lethargy, with or without focal neurological deficit (2). Our patient was irritable during the first hours after the accident. The proposed guidelines for apparently minor head trauma suggest: the younger the child, the lower the threshold should be for imaging studies; the greater the severity and number of historical symptoms and physical signs, the stronger the consideration for imaging studies; the more pronounced the physical findings and the younger the age, the greater the risk of intracranial injury (13). According to these guidelines, child's irritability or a history of prolonged irritability is one of the indicating factors for performing a CT/MRI scan. The presented case report underlines the possibility of an association between infant irritability and potential intracranial injury.

In conclusion, forces of acceleration may cause ischemic stroke in a newborn. Persistent child irritability after head trauma is one of the indicating factors for performing an emergency CT scan of the head.

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Autoavarijos metu uždarosios traumos sukeltas insultas 27 dienų amžiaus mergaitei

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Raktažodžiai: perinatalinis insultas, trauminis insultas.

Santrauka. Gimdymo trauma (ne ponatalinė trauma) yra pripažinta naujagimių galvos smegenų infarkto priežastimi. Straipsnyje aprašomas klinikinis atvejis, kai 27 dienų mergaitė po autoavarijos patyrė galvos smegenų infarktą.

Autoįvykio metu vaikas buvo tinkamai prisegtas prie vaikiškos kėdutės. Pacientė buvo hospitalizuota stebėsenai dėl aiškiai pastebimo dirglumo. Pirminės apžiūros metu židininių neurologinių požymių nepasireiškė. Praėjus 28 valandoms po autoįvykio, naujagimį ištiko židininiai toniniai-kloniniai priepuoliai ir švelni dešinės pusės hemiparezė. Priepuoliai veiksmingai gydyti fenobarbitaliu 30 mg per dieną. Kompiuterinės tomografijos ir magnetinio rezonanso tyrimai, atlikti atitinkamai antrąją ir trečiąją dienomis, parodė subduralinę hemoragiją pakaušio srityje ir smegenų išemiją kairėje sieninėje pakaušio srityje. Praėjus 10 dienų rentgenologinių kontrolinių tyrimų metu pastebėti reperfuzijos požymiai.

Besitęsiantis vaiko dirglumas po galvos traumos yra vienas iš veiksnių skubiai atlikti galvos kompiuterinę tomografiją.

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