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Diffusion-weighted MRI of middle cerebral artery stroke in a newborn

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C. Fusch Department of Neonatology, University of Greifswald, Germany Abstract Diffusion-weighted MRI of the brain is becoming clinically available as a tool to investigate cerebral ischaemia. We report a newborn girl presenting with seizures in whom diffusion-weighted MRI showed a large hyperintensity in the area perfused by the left middle cerebral artery. Short-term neurological follow-up before discharge was uneventful and the patient was discharged without sequelae. On follow-up clinical examination, right-sided spastic signs were noted which disappeared with time.

Introduction

Major cerebral artery stroke is rare in neonates and sometimes difficult to diagnose [1]. While conventional methods such as cranial US and CT have shown themselves to be of use, MRI is becoming the method of choice for imaging of neonates and children in general [2]. Diffusion-weighted MRI (DWI) has established itself as a sensitive method for the detection of ischaemia, both in adults and children [3–6]. In this technique, a pair of diffusion-sensitising pulses is placed in a spinecho sequence: the imaging sequence is rendered sensitive to motion. In cerebral ischaemia, the diminished molecular motion associated with dysfunction of the membrane-bound Na⁺K⁺ATPase leads to intracellular accumulation of water and cytotoxic oedema. This is seen as a decrease in the apparent diffusion coefficient

and as increased signal on diffusion-weighted images acquired at a high b value. We report a newborn presenting with seizures in who MRI of the brain with DWI was performed.

Case report

This girl was born to a 42-year-old mother by caesarean section indicated by oligohydramnios and fetal intrauterine position, after an uneventful pregnancy. At birth the general and neurological examinations were normal and the patient was initially transferred to the post-natal unit. After the first myoclonic seizure of the arms, neck and mouth, deviation of gaze and nystagmus towards the right and hypersalivation on the second day of life, she was transferred to the neonatal intensive care unit. There she suffered a further seizure. Injection of vitamin B_6 was ineffective, but the seizures were stopped with phenobarbital. Blood electrolytes, haemoglobin and

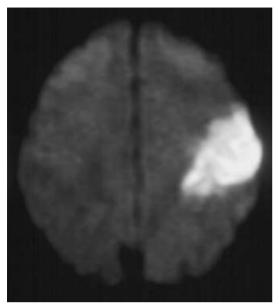


Fig. 1 Axial isotropic DWI showing a hyperintense area in the left hemisphere corresponding to the middle cerebral artery territory

urine and plasma amino acids were normal. Ultrasound of the brain was normal, as was the EEG. On the following night, seizures recurred and were again stopped with phenobarbital. The next morning, decreased consciousness and generalized hypotonia were noted. MRI was performed on the morning of the 3rd day of life.

MRI was performed on a 1.5-T system (Siemens Vision) equipped with a head coil and capable of echo-planar imaging. T2-weighted (T2-W) (TR 3,5000 ms; TE 98 ms) and isotropic diffusionweighted MR sequences with b values of 0 and 972 s/mm² (TR 4,000 ms; TE 136 ms; 4 acquisitions) were performed, followed by MR angiography (MRA) done with a time-of-flight sequence. The DWI sets showed the presence of a large area of high signal on the images at the high b value in the frontal area perfused by the left middle cerebral artery (MCA) (Fig. 1). The ischaemic region seemed to encompass the posterior part of the insula, part of the inferior frontal gyrus and the postcentral gyrus. Apparent diffusion coefficient (ADC) values were subsequently calculated from pixelby-pixel ADC maps generated by the MR console software. The slices with b values of 0 and 972 s/mm² were matched and ADC maps were obtained for each slice. ADC values were calculated according to the Stejskal and Tanner equation. Areas of disturbed diffusion on the high b value images, seen as hyperintensities, corresponding to low signal on the ADC maps, were then measured on the corresponding ADC maps. Regions of interest were determined using the console software as circles in the small areas of disturbed signal on the ADC maps. ADC values were also measured in the contralateral region. The relative ADC (rADC) was then obtained by dividing the value in the ischaemic area by a measure in the contralateral non-affected region. ADC values varied between 638 and 840 mm²/s in the ischaemic region, and 1170 to 1300 mm²/s in the contralateral normal-appearing region. Relative ADCs in the lesion thus varied between 0.49 and 0.61.

T2-W MRI showed slight effacement of the cortical sulci in the corresponding area (Fig. 2). MRA did not show any occlusion of the appropriate vessel (Fig. 3).

Although CMV and herpetic encephalitis were not the cause, she was initially treated with aciclovir. Repeated lumbar punctures

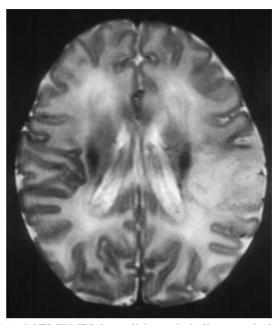


Fig. 2 Axial T2-W MRI shows slight cortical effacement in the corresponding area

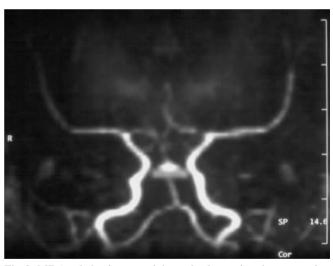


Fig. 3 MRA of the intracranial cerebral arteries demonstrating patent middle cerebral arteries on both sides

were normal. Phenobarbital treatment was discontinued and there was no recurrence of seizures. Neurological progress was normal: consciousness was full and there were no detectable neurological deficits, feeding was normal with progressive weight gain and the patient was discharged after 2 weeks with a normal neurological examination.

Further neurological examinations were undertaken at 4 and 8 months after birth. At 4 months, movements were asymmetrical with less movement of the right side of the body. Muscular tone was higher on the right side and there was hyperreflexia of the right peripheral tendon reflexes. However, at 8 months these findings had almost completely disappeared and both hands manipu-

lated objects equally. The child was reassessed at 22 months of age and found to have almost normal psychomotor development; speech development was, at most, 2 months behind schedule. At 2 years and 3 months of age she was brought to medical attention because of slight dysuria and superficial neurological examination at that time revealed no gross abnormality.

Discussion

We have reported the case of a newborn child presenting with seizures, in whom DWI showed the presence of a left-sided partial MCA infarct. Based on MRA, it must be concluded that a MCA branch had been occluded. Pregnancy had been uneventful; there were no positive findings suggestive of an underlying systemic disease, even at 3 years of age, and no recurrence of neurological signs. Initial EEG and cranial US were normal. This might in part be due to the focal and superficial location of the lesion (on coronal US the lateral cortical areas are not well seen).

Major cerebral infarction in newborns is rare with an incidence estimated to be 1 in 4,000 newborns [1]. Since the course is often subclinical, the true incidence is probably higher. Ultrasound remains an important non-invasive screening tool for its diagnosis. Definitive diagnosis is made by CT or MRI. Besides symptomatic anticonvulsive therapy, the aim of treatment is the prevention of secondary ischaemic injury. Clinically, seizures are an important initial symptom, as they were in this case. MRI techniques with their non-ionising nature and multiplanar capabilities are now establishing themselves as an important adjunct in the armamentarium for the diagnosis of brain diseases in children and newborns.

DWI is becoming established in adults as a non-invasive method for the investigation of acute ischaemia [2] since it has been shown both in animal models and in the human setting of cerebral stroke to be sensitive to early changes induced by ischaemia. Indeed it can reliably demonstrate the diminished motion of water molecules associated with water redistribution from the extracellular to intracellular compartments. A few cases reporting its use in neonatal ischaemia have been re-

ported, such as in periventricular leucomalacia (PVL) [3] and stroke [4–6]. PVL, which is the principal form of brain injury in the premature infant, is characterised by overt focal necrotic lesions in the periventricular white matter and less prominent, more diffuse cerebral white-matter injury. Early detection of the diffuse component of PVL has been demonstrated by DWI as a striking bilateral decrease in water diffusion in cerebral white matter. The DWI abnormality (i.e. decreased ADC) was similar to that observed with acute cerebral ischaemic lesions in adults. At 10 weeks of age, conventional MRI and US showed striking changes consistent with PVL, including the presence of small cysts. Johnson et al. [5], in a clinically defined cohort of 26 consecutive neonates and infants with suspected hypoxicischaemic injury, found that echo-planar DWI revealed a greater extent of, and a larger number of, abnormalities compared with FLAIR or turbo T2-W images in 11 (92%) of 12 patients with abnormal DWI.

Rumpel et al. [6] described a case of neonatal stroke in the territory of the left MCA. Although the ischaemic lesion appeared rather homogeneously hyperintense on T2-W MRI, corresponding DWI clearly delineated two separate zones of different cellular swelling and, thus, different prognoses. They found that cytotoxic oedema heralded infarction. In our case, we found the hyperintensities on DWI to correspond to decreased ADC values, signifying predominant cytotoxic oedema. Connelly et al. [4] reported two children with regions of DWI hyperintensity which did not progress to infarction. In our case, while early MRI and DWI findings were consistent with ischaemia, follow-up imaging with US failed to show leucomalacia and the neurological examination at discharge was normal. Apart from slight signs of spasticity at the first follow-up, the child was found to have an almost normal neurological development at the age of 22 months.

We conclude that DWI can demonstrate tissue which is compromised. The initial finding on DWI corresponds to an infarcted core, often surrounded by an area of potential penumbra. There might not always be correlation between the early extent of the lesion and later clinical signs, as is observed in the adult setting.

References

- Govaert P, Matthys B, Zecic A, et al (2000) Perinatal cortical infarction within middle cerebral artery trunks. Arch Dis Child 82: 59–63
- 2. Lovblad KO, Laubach HJ, Baird AE, et al (1998) Clinical experience with diffusion-weighted MR in patients with acute stroke AJNR 19: 1061–1066
- 3. Inder T, Huppi PS, Zientara GP, et al (1999) Early detection of periventricular leukomalacia by diffusion-weighted magnetic resonance imaging techniques. J Pediatr 134: 631–634
- 4. Connelly A, Chong WK, Johnson CL, et al (1997) Diffusion weighted magnetic resonance imaging of compromised tissue in stroke. Arch Dis Child 77: 38–41
- 5. Johnson AJ, Lee BC, Lin W (1999) Echoplanar diffusion-weighted imaging in neonates and infants with suspected hypoxic-ischemic injury: correlation with patient outcome. AJR 172: 219–226
- 6. Rumpel H, Fermi B, Martin B (1998) Lasting cytotoxic edema as an indicator of irreversible brain damage: a case of neonatal stroke. AJNR 19: 1636–1638