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case report

Old middle cerebral infarction in a neonate

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Background. *Cerebral arterial thrombosis is a relatively rare occurrence in the neonatal period. Some children having this condition as neonates, present recurrent local seizures with radiological evidence of acute infarction. Several studies have shown this to be a rare cause of neonatal seizures.*

Case report. *We describe the case of a newborn infant with localized clonic seizures of the lower right extremity on the fourth day of life. Ultrasound examination of the brain showed only mild abnormalities. However Magnetic Resonance Imaging showed evidence of a middle left cerebral arterial infarction.*

Conclusions. *The newer imaging methods, such as central nervous system Magnetic Resonance Imaging can be helpful in the diagnosis of rarer disturbances and can further reveal cases which would otherwise pass unnoticed even where ultrasound imaging is negative.*

Key words: cerebral infarction; infant, newborn; middle cerebral artery

Introduction

Neonatal seizures can be symptoms of numerous underlying disorders in the neonate. Cerebral arterial thrombosis is a relatively rare occurence in the neonatal period. Some children with this condition as neonates have recurrent local seizures with radiological evidence of infarction. Several studies have shown that the infarction is a rare cause of neonatal seizures. Asphyxia, birth trauma or polycythemia can be predisposing factors; however, the majority of reported cases in full-term neonates are considered idiopathic. In the full-term newborns, infarction most commonly occurs in the tissue supplied by the middle cerebral artery, with the left side more often affected than the right one.[1,2]

We describe the case of a newborn infant who presented local clonic seizures of the right lower extremity on the fourth day of life. Ultrasound examination of the brain showed mild abnormalities only. Magnetic Resonance Imaging however revealed the signs of a pre-

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vious middle-left side cerebral arterial infarction, which could occur *in utero*. A reduction in size of the left cerebral peduncle was observed, and was attributed to transaxonal degeneration.

Case report

A newborn male weighted 3100 gr., was delivered after a full-term uneventful pregnancy in uncomplicated vaginal delivery. Apgar scores reported were 8 and 9 at 1 and 5 minutes respectively. For the first 72 hours, the newborn was in good condition and remained in the maternity clinic. On the fourth day after birth, three attacks of local clonic seizures in his lower extremities were reported and the newborn was admitted to our department.

Upon admission, the newborn infant was in good condition and the results of physical examination, as well as retinal examination were normal. Hematological and biochemical tests (hemoglobin, white cell count, platelet count, hemoglobin electrophoresis, glucose, calcium, magnesium, cholesterol, electrolytes and BUN) were within normal ranges. Glucose and protein levels in the cerebrospinal fluid were normal. Anion gap, blood gas, aminoacid chromatography, organic acid chromatography and homocystin serum levels, were within normal ranges. Blood, urine, and cerebrospinal fluid cultures were negative. Toxoplasma, rubella, herpes simplex virus, syphilis, and cytomegalovirus titers did not reveal any infection.

Immediately after admission, anti-convulsive therapy was administered to the newborn, initially phenobarbital followed by phenytoin and diazepam. Phenytoin and diazepam were administered for ten days and phenobarbital for two months. The newborn, during the course of hospitalization, suffered repeated episodes of localized seizures of the right lower extremity which were gradually brought under control with anticonvulsive

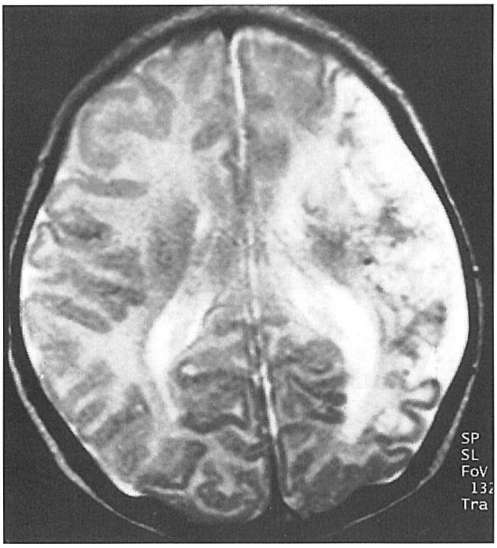

Figure 1a. Axial T_2 weighted (TR:5.400, TE:99) image through the bodies of the lateral ventricles. High signal intensity lesion in the left parietal lobe and mild dilatation of the body of the left lateral ventricle. Note the decreased size of the left peduncle in comparison with the right one.

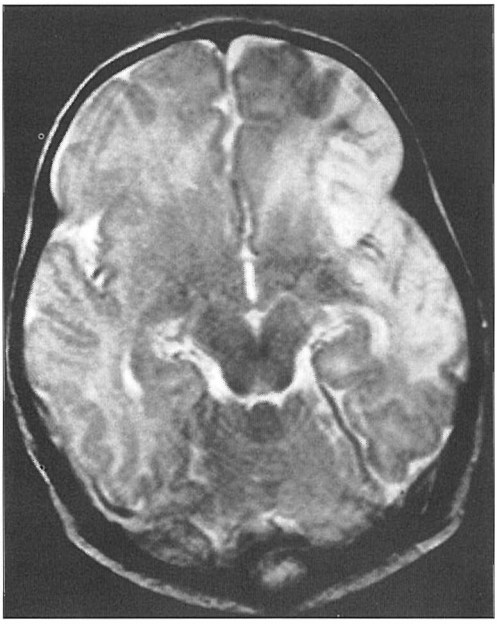

Figure 1b. Axial T_2 weighted (TR:5.400, TE:99) images through the mesencephalon. Extension of the lesion to regions of the left temporal and frontal lobes supplied by the middle cerebral artery. Note the decreased size of the left peduncle in comparison with the right one.

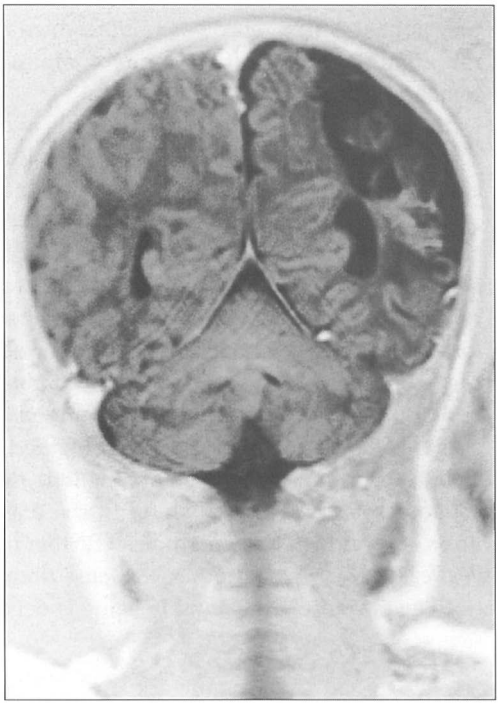

Figure 2. Coronal T_1-weighted turbo inversion recovery image. The signal intensity of the lesion did not significantly differ from the signal of the cerebrospinal fluid, indicating cystic evolution in the affected area.

therapy. Electroencephalogram findings were normal. Prothrombin and partial thromboplastin times and the fibrinogen, protein C, protein S and antithrombin III levels were also normal. No antinuclear antibodies, anticardiolipin antibodies or lupus anticoagulant were detected, either in the newborn or in his mother, while the levels of proteins C and S were within the normal ranges.

Ultrasonography examination using an ATL Ultramark-9 5 MHz transducer demonstrated a mild, non-specific increase in echogenicity in the region of the left parietal and temporal lobes. Then MR imaging (Siemens, Vision plus, 1.5T) was performed and revealed a destructive lesion with loss of brain tissue in the region supplied by the main branch of the left middle cerebral artery. The lesion was of low signal intensity on T_1 weighted images, of high signal intensity on T_2 weighted images, and exhibiting predominantly low signal intensity with some peripheral poorly demarcated areas of moderately high signal in heavily T_2 weighted images, with cerebrospinal fluid saturation (FLAIR). A mild dilatation of the left ventricle was also found. A reduction in the size of the left cerebral peduncle was observed and it was attributed to transaxonal (wallerian) degeneration (Figure 1a,b and Figure 2). After ten days treatment, phenytoin and diazepam were discontinued. The infant remained in hospital for ten more days free of seizures. The infant was discharged from our department at the age of 28 days in good condition with slight hypertonia of the right upper and lower extremities. He remains under regular observation, with monthly visits to follow his psychomotor progress and continues with the course of physiotherapy which was started during hospitalization.

The last clinical evaluation done at the age of six months did not reveal any localized neurological signs, while his psychomotor development is in accordance with his age group.

Discussion

Unilateral neonatal cerebral infarction, or neonatal stroke, can occur in both pre-term and full-term infants and usually manifest itself as localized neonatal seizures in an otherwise healthy full-term neonate; it is one of the causes of spastic hemiplegic cerebral palsy.[1]

A number of causes have been reported for neonatal localized infarction. Usually perinatal asphyxia, birth-trauma, and resuscitation by assisted ventilation, either by "bag-and-mask" or endotracheal intubation, are more common occurrences in such neonates than in healthy ones.[1] Other factors, like polycythemia, dehydration, exchange transfusion, indwelling (temporal) arterial catheters, per-

sistent *ductus arteriosus,* cocaine abuse and coagulopathy, such as protein C and S deficiency, and factor V have all been mentioned in literature. The presence of an arteriovenous malformation should also be considered.[1,2] Recently an association between cerebral infarction and antiphospholipid antibodies has been found to be a significant factor.[3] However cerebral infarction can also occur before or after birth in healthy infants without any overt underlying pathology.[1] The varied and sometimes subtle symptomatology raise the possibility that some cases may not be detected in the perinatal period.[1] The outcome of each case depends on its predisposing factors and the underlying pathology. It has been reported that full-term infants with the infarction unrelated to significant birth asphyxia usually have a favorable outcome.[4]

Although periventricular and subcortical infarction may be visualized very well by cerebral ultrasonography, it has been found that scanning with 5 and 7.5 MHz transducers is often unhelpful in the diagnosis of unilateral neonatal cerebral infarction. In the presented case, the ultrasonographic findings were nonspecific and the diagnosis of the middle cerebral artery infarct was confirmed on Magnetic Resonance Imaging. The loss of brain tissue associated with mild dilatation of the ventricle and of the regional peripheral subarachnoid space, the signal intensity of the lesion that did not differ significantly from the signal of the cerebrospinal fluid with the findings consistent with wallerian degeneration, were suggestive of a chronic infarct which had occurred during the inta-uterine development life. To our knowledge, transaxonal degeneration has not been previously documented in the first weeks of life. It has only been described in infants of more than two and half months of age.[5] Ultrasound imaging may appear normal even when interpreted in the light of information from computed tomography scanning.

In conclusion; in every newborn with seizures, especially when localized, the clinician, beside considering the usual causes for this, must be aware of the possibility of rarer causes. Cerebral infarction can even occur *in utero,* as was the case of our patient; it was revealed by Magnetic Resonance Imaging performed on the 12th day of life, despite the absence of other predisposing factors. Cerebral infarction can result in seizures or semi-paresis in newborns. The newer imaging methods, such as central nervous system Magnetic Resonance Imaging can be helpful in the diagnosis of rarer disturbances and can further reveal case which would otherwise pass unnoticed even where ultrasound imaging is negative.

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