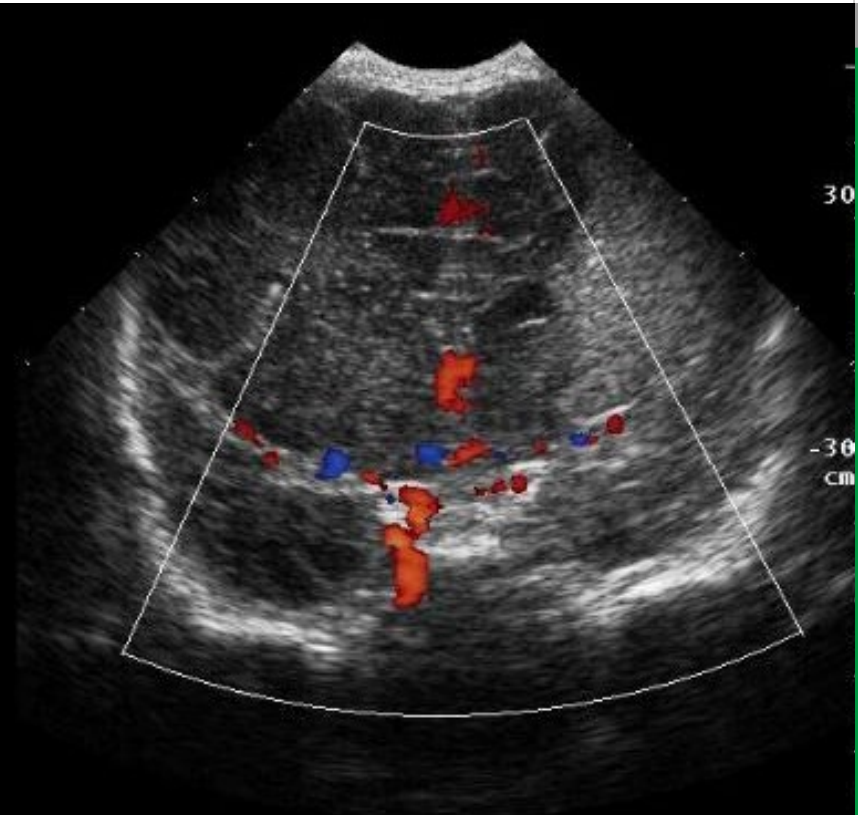
SWISS SOCIETY OF NEONATOLOGY

Neonatal cerebral infarction



May 2002

Mann C, Neonatal and Pediatric Intensive Care Unit,
Landeskrankenhaus und Akademisches Lehrkrankenhaus
Feldkirch, Austria

Pregnancy had been uneventful until the 30th week of gestation when unilateral fetal hydronephrosis was noted (a ureteropelvic junction obstruction was later diagnosed and surgically corrected). Follow-up ultrasound examinations, including a scan 24 hours prior to delivery, were normal except for a slightly decreased resistance index for one of the middle cerebral arteries.

A male infant was delivered at term by emergency Cesarian section because of fetal tachycardia and severe decelerations. There was meconium-stained amniotic fluid. The umbilical cord was tightly wrapped around the infant's body and there was a true knot. Arterial umbilical cord pH was 7.18, and Apgar scores were 5, 8, and 8 at 1, 5, and 10 minutes, respectively. Birth weight (3150 g) and head circumference (36 cm) were appropriate for gestational age. The infant was admitted to the neonatal ICU because of mild respiratory distress with a transient oxygen requirement. On admission, his neurological assessment was normal.

On the second day of life, a routine ultrasound scan of the head revealed a wedge-shaped hyperechogenic area corresponding to the territory of the left middle cerebral artery. The Doppler signal of the left internal carotid artery was completely missing. The flow signal of the left middle cerebral artery was diminished with some flow originating from the anterior communicating artery (Fig. 1).

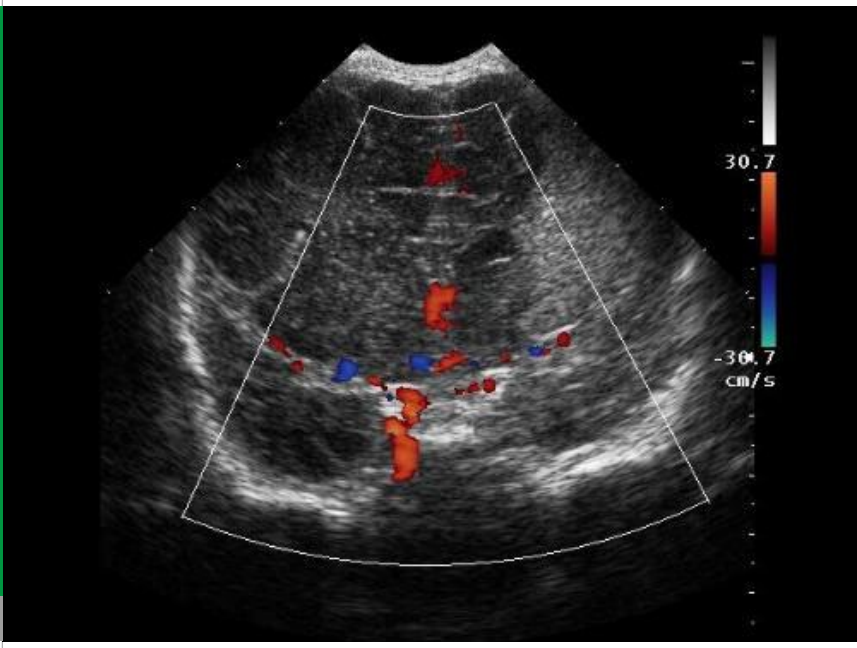


Fig. 1

Doppler US examination (DOL 2): diminished flow in the left middle cerebral artery.

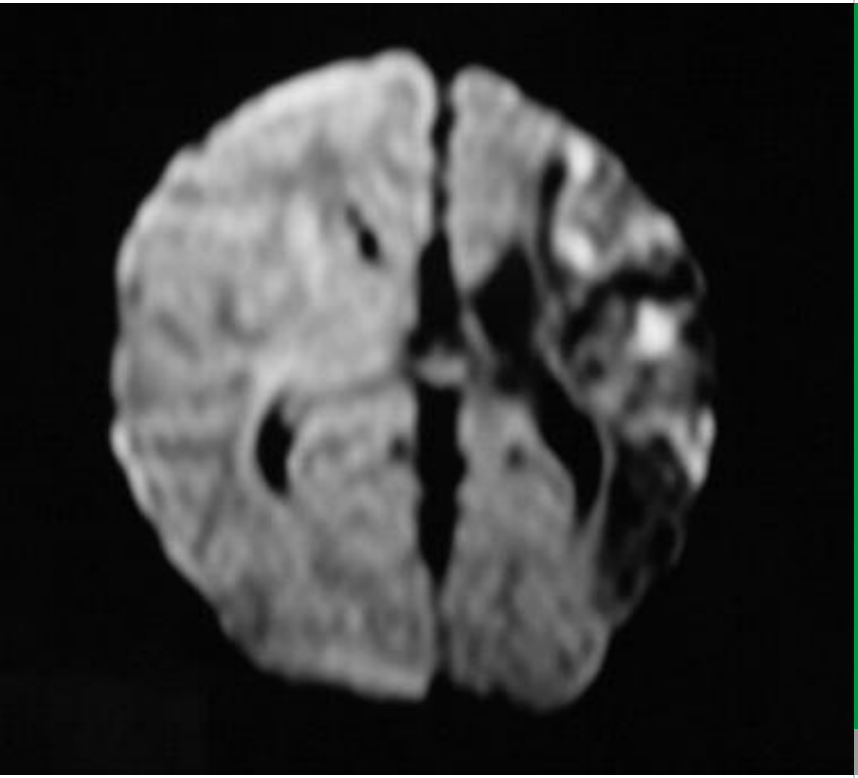


Fig. 2

MRI DWI (DOL 3): cytotoxic edema.

An echocardiogram was normal without evidence of atrial septal aneurysm, atrial thrombi or abnormalities of the large vessels. Extensive laboratory investigations, including hematocrit, thrombocytes, prothrombin time, partial thromboplastin time, fibrinogen, antithrombin III, plasminogen, activated protein C resistance, factor V Leiden, protein C, protein S, prothrombin variant G20210A, antiphospholipid antibodies, homocystein, rheumatoid factor, ANA, Lp(a), were normal and not consistent with a prothrombotic state.

On the 3rd day of life, an MRI showed cytotoxic edema in diffusion weighted images (Fig. 2), signs of edema and gliosis in the flare sequences (Fig. 3) and the T2-weighted images (Fig. 4), as well as hemosiderin deposition in the T2-weighted images (Fig. 4). Hemosiderin can be detected as early as 2 days after the extravasation of blood into the tissue, however, the presence of gliosis indicates that tissue hypoxia had occurred at least 2 weeks earlier. The absence of a mass effect and evidence of some tissue deficit on the left side supported this interpretation. Magnetic resonance angiography (MRA) confirmed the obliteration of the left internal carotid artery (Fig. 5).

On the 6th day of life, a Doppler signal could be detected over the left internal carotid artery; apparently, spontaneous recanalisation had taken place. In the 7th week of life, MRA was repeated and confirmed patency of left-sided internal carotid and middle cerebral arteries, however, the vascular diameters were smaller than on the contralateral side (Fig. 6).

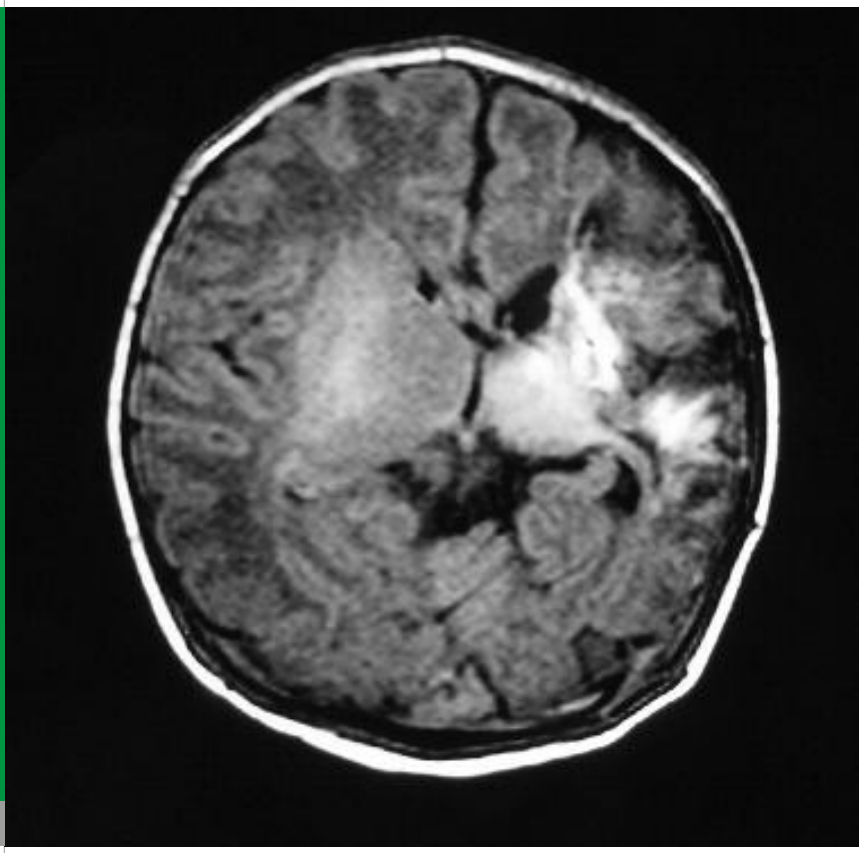


Fig. 3

MRI flare sequence (DOL 3): extensive edema, gliosis.

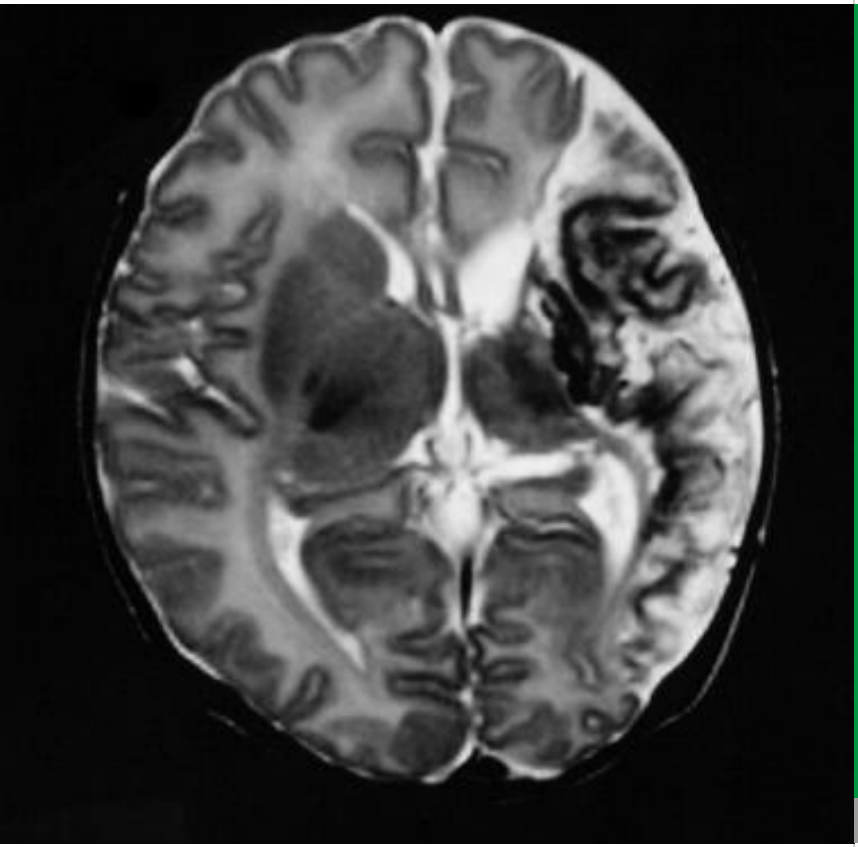


Fig. 4

MRI T2-weighted images (DOL 3): extensive edema, gliosis, and hemosiderin deposition.

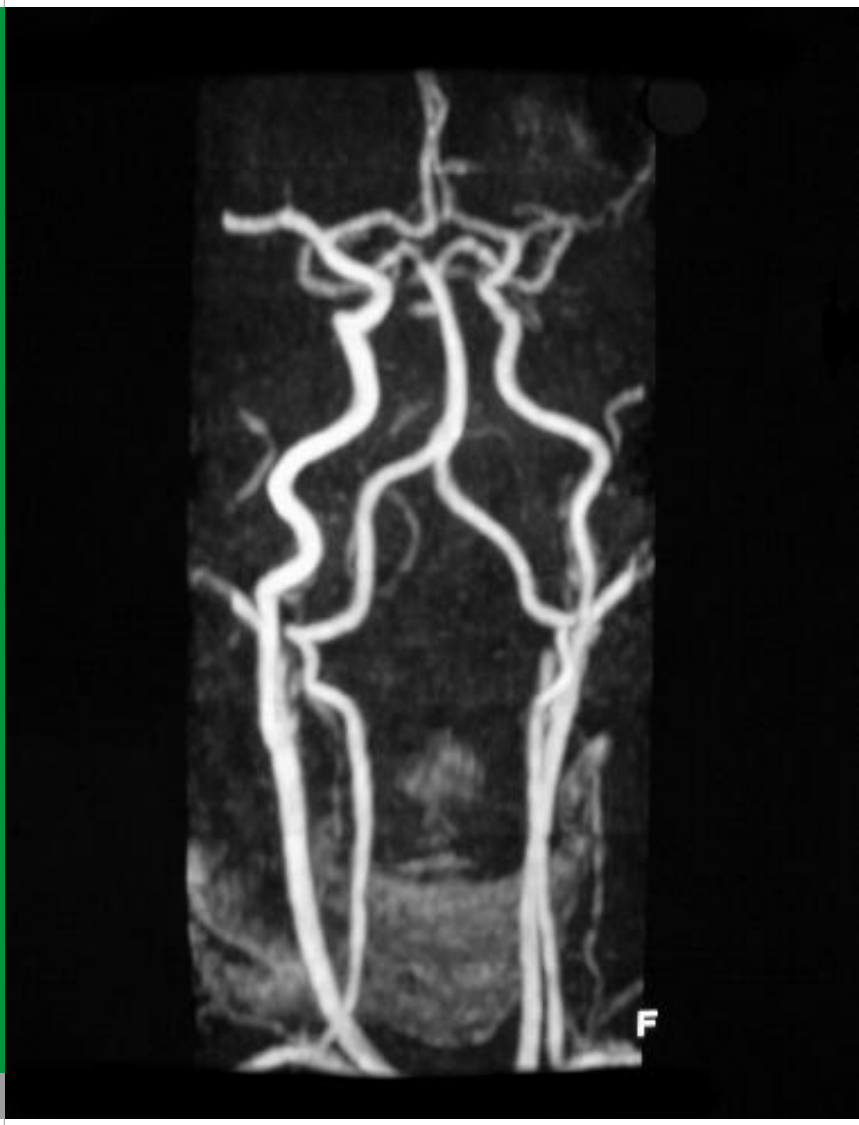


Fig. 5

MRA (DOL 3): obliteration of the left internal carotid artery.

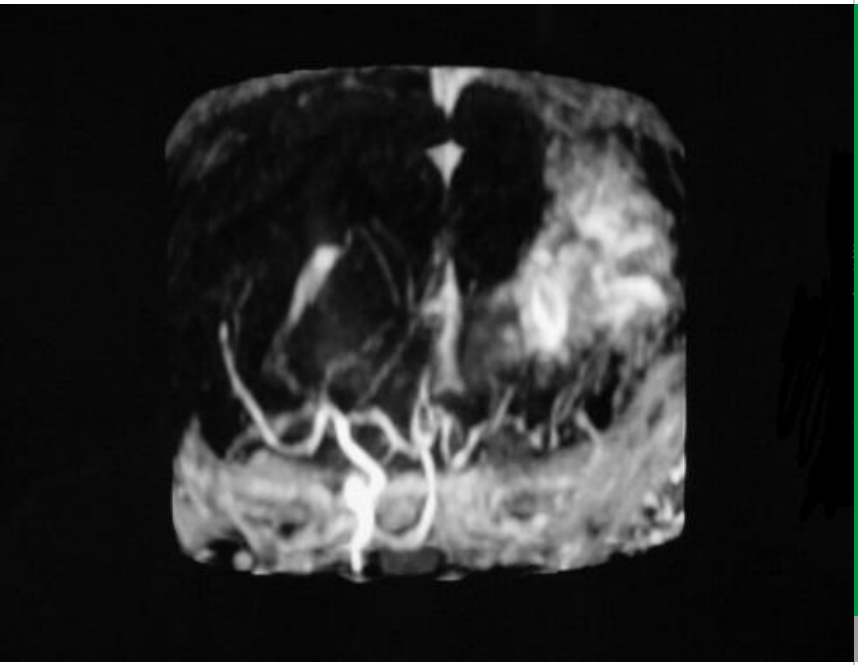


Fig.6

MRA (DOL 42): patent left internal carotid artery.

DISCUSSION

Neonatal cerebral infarctions typically present with seizures or lethargy in the neonatal period (1). In the present case, without the ultrasound investigation on the 2nd day of life, it is very likely that the diagnosis would only have been made at the time when hemiparesis became evident. Approximately 25% of patients with neonatal strokes develop hemiparesis, typically at 4 to 8 months of life (1). In our patient, early MRI (3rd day of life, Fig. 2-4) documented that the infarction must have occurred at least 2 weeks earlier. In addition, areas of different stages of cytotoxic edema pointed to the fact that repetitive episodes of ischemia must have taken place. We speculate that a thrombus may have developed in the umbilical vein due to decreased flow secondary to the true knot and may have embolized into the brain (2). Data in the literature does not clearly support the use of heparin in neonatal stroke due to arterial thrombosis or embolism, especially in the absence of a prothrombotic state (1,3). Twenty percent of neonatal infarctions are hemorrhagic (3), and anticoagulation may be dangerous in those cases.

Prognosis of neonatal cerebral infarction depends on several factors. The likelihood of a functionally disabling hemiparesis is increased if the cerebral infarction involves the cortex as well as the basal ganglia, which was the case in our patient. On the other hand, the infant's normal background activity on EEG during the neonatal period is associated with better developmental outcome.

1. Silverboard G, Roger T. Cerebrovascular arterial dissection in children and young adults. Semin Pediatr Neurol 2000;7:289-300 (*Abstract)*

2. deVeber G, The Canadian Pediatric Ischemic Stroke Study Group. Canadian paediatric ischemic stroke registry: analysis of children with arterial ischemic stroke. Ann Neurol 2000;48:526

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CONTACT

Swiss Society of Neonatology
www.neonet.ch
webmaster@neonet.ch