Umbilical cord thrombosis and chorioamnionitis in neonatal arterial ischaemic stroke

A baby boy was delivered at term by emergency caesarean section because of abnormal fetal heart rate patterns during spontaneous labour. The umbilical cord arterial blood obtained at delivery had a pH of 6.97 and a lactate concentration of 12.1 mmol/L. The Apgar scores were 3 and 7, respectively, at 1 and 5 min. The proximal umbilical cord had a thrombotic aspect at delivery (figure 1). A thrombus was also identified in the ductus venosus at 1 hour of life. The boy presented left hemicorporeal seizures at 21 hours of life, which revealed a neonatal arterial ischaemic stroke (NAIS; figure 2). Placental histology revealed acute chorioamnionitis (online supplemental file). Screening for thrombophilia was negative in the boy and his mother.¹

The pathophysiology of NAIS remains unclear but may be multifactorial.² Two main mechanisms of NAIS are currently suspected: a cerebral embolism of a placental thrombosis and a focal cerebral arteritis secondary to chorioamnionitis.³ However, the intuitive embolic hypothesis is challenged,³ and perinatal inflammation is consistently reported as a major independent risk factor of NAIS in recent case-control studies.^{3,4}

The baby combined a lot of NAIS risk factors: nulliparity, male sex, multiple markers of difficulty with transition—fetal heart rate abnormality, emergency caesarean section, low Apgar score, low umbilical artery pH—and perinatal inflammation.²⁻⁴ Proximal umbilical cord thrombosis is highly unusual and has never been described in NAIS.

Figure 1 Umbilical cord at delivery.

Figure 2 Focal restriction of diffusion on cranial MRI at 24 hours of life, delineating a recent ischaemic lesion in the superficial posterior territory of the right median cerebral artery.

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