An Unusual Cause of Neonatal Seizures in a Newborn Infant

ABSTRACT. Neonatal seizures in the neonatal period are symptoms of numerous underlying disorders of the neonate. We present a case in which neonatal seizures due to cerebral infarction led to a diagnosis in the mother.

Neonatal convulsions caused by cerebral artery thrombosis is relatively rare in the neonatal period and is often secondary to indwelling intravascular catheters that cause thromboembolism, but may be associated with many conditions.1 Cerebral arterial thrombosis in newborns, in which antiphospholipid antibodies (APA) were found in the mother, has been described in three case reports.2,3 Two of these premature infants were born with other risk factors for thrombosis. APA could not be identified in any of these three infants. In the two cases reported by Silver et al4 the diagnosis was made several months after birth.

This case is unique in the fact that no other risk factors for thrombosis could be identified to explain the infarction, and that APA were found in the offspring of an apparently healthy mother. Whether the prior fetal death was caused by APA remains unclear. The finding of lupus anticoagulant in her child led to the diagnosis of antiphospholipid antibody syndrome in her. We believe that in case of cerebral artery thrombosis in a neonate, with no trivial cause such as an indwelling catheter or sepsis, both mother and infant should be tested for presence of APA, even when the mother seems healthy. Pediatrics 1997;100(4). URL: http://www.pediatrics.org/cgi/content/full/100/4/e8; neonate, cerebral infarction, maternal antiphospholipid syndrome.

COMMENTS

In the neonatal period, convulsions are mostly attributable to perinatal asphyxia, metabolic derangements, or infections.2 Thromboembolism is a relatively rare cause of convulsions in the newborn and is often secondary to indwelling intravascular catheters,3 but may be associated with many conditions. Thromboembolism in newborns of LAC-positive mothers has been described.4,5 Affected women characteristically have poor pregnancy outcomes that may be improved with prednisone and low-dose aspirin treatment.6 Zurgil7 showed transplacental transfer of APA in 18 pregnant women, but in none of the cases could clinical manifestations of APA syndrome be detected. Titers of APA in affected women may fall after pregnancy. This may explain why antiphospholipid antibodies, and not LAC, could be detected in the mother at the time of delivery.

Cerebral artery thrombosis in newborns, in which APA were found in the mother, has been described in three case reports.6,9 Two of these premature infants were born with other risk factors for thrombosis. In none of the three cases could APA be identified in the infant. In the two cases reported by Silver et al10 the diagnosis was made several months after birth.

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factors for thrombosis could be identified to explain the infarction and that APA were found in the offspring of an apparently healthy mother. Whether the prior fetal death was caused by APA remains unclear. The finding of LAC in her child led to the diagnosis of antiphospholipid antibody syndrome in her. This is comparable with diagnosing systemic lupus erythematosus in mothers of children with Ro-SSA positive congenital heart block or neonatal thrombocytopenia.10

**Fig 1.** MRI image of the brain made 1 week postpartum showing edema of the left hemisphere with a small hemorrhage, corresponding with a left middle cerebral artery infarction.

**Fig 2.** MRI image made at 3 months after birth showing focal cortical atrophy of the left parietal lobe, suggesting an old infarction of the left middle cerebral hemisphere.
We believe that in the case of thromboembolism in a neonate, with no trivial cause such as an indwelling catheter or sepsis, both mother and infant should be tested for the presence of APA, even when the mother seems healthy.

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DOI: 10.1542/peds.100.4.e8

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