Stroke in newborns with encephalopathy

In the prospective study by Ramaswamy et al. 124 encephalopathic term infants were evaluated. Six had an acute stroke and all six presented with seizures. The infants had a poor neurodevelopmental outcome.

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Neonatal stroke and encephalopathy: Refining our understanding

Commentary by Gabrielle deVeber, MD, MSc

The association of stroke and neonatal encephalopathy has been difficult to establish. The similar clinical presentation of the two conditions results in diagnostic confusion and difficulty in estimating the individual and combined frequencies. The prospective design, robust neuroimaging techniques, and application of strict criteria for neonatal encephalopathy enable the Ramaswamy et al. study to make a firm conclusion regarding the association of stroke in 5% of neonates with encephalopathy.

The current study provides additional insight into questions regarding the timing and mechanisms of neonatal brain injury. All five neonates with stroke and encephalopathy had “severe clinical encephalopathy.” While stroke may be a marker for more severe encephalopathy it may also simply add to the clinical signs of global encephalopathy. The increased frequency of complicated delivery (failure to progress) in infants with stroke and encephalopathy compared with encephalopathy alone is intriguing. This observation suggests that instrumentation at delivery may not be the cause for cerebral insults in these infants but rather an appropriate response to the need for assisted delivery in prolonged labor. Stroke and encephalopathy may be separate outcomes of more difficult and prolonged labor. In the hours surrounding the birth process, the ex-

Right middle cerebral artery infarct with bilateral watershed injury: (A) axial T1-weighted image, (B) T2-weighted image.

act timing of the cerebral insults is not known. Whether the prolonged labor is itself causative for stroke and encephalopathy or the result of decreased participation in the delivery process by the acutely neurologically impaired infant also remains an unanswered question.

The mechanism for stroke may differ in neonates with encephalopathy compared to other neonates with stroke. In the latter, embolism and cerebral arterial compression (via placental insults and nuchal cord) have been reported, but were not observed in this study. Globally reduced cerebral perfusion could well result in focal arterial thrombosis through blood flow stasis.

Undoubtedly the presence of an increased volume of injured brain when stroke and encephalopathy

coexist will reduce the ability of the brain to recover. In adults with stroke, global cerebral insults including systemic hypotension clearly result in worse outcomes. However there may also be mechanisms beyond the additive effects of the two insults. The severe outcome when stroke and encephalopathy coexist emphasizes the importance of documenting all mechanisms of brain injury in neonates. Modified radiographic assessment may be required to detect stroke including delayed CT and diffusion MRI. The coexistence of stroke and encephalopathy should intensify our surveillance and early referral for rehabilitation in such infants, and increases the need for early supportive and neuroprotective strategies.

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