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Symmetrical thalamic lesions in a term infant

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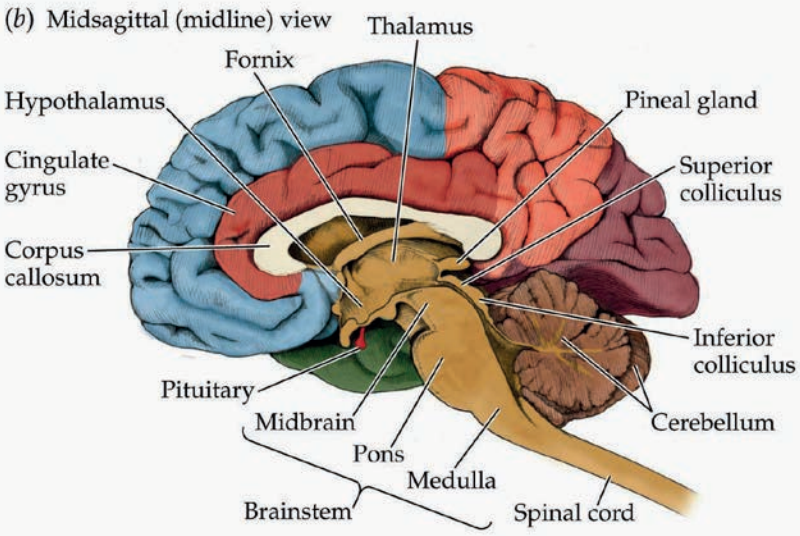

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Hypoxic-ischemic brain injury is a very important neurological problem in the perinatal period. The neuropathological features of neonatal hypoxic-ischemic encephalopathy vary considerably with the gestational age of the infant, nature and time of onset of the insult, type of interventions, and other factors (1). Selective neuronal necrosis is the most common variety of injury observed and refers to necrosis of neurons in a characteristic distribution.

We describe the case of a term infant with typical clinical settings for neonatal hypoxic ischemic encephalopathy, a history of prenatal onset of symptoms, and symmetrical thalamic lesions on magnetic resonance imaging.

The mother was a 30-year-old G3/P3. Pregnancy had been uneventful until about 35 weeks' of gestation. At that time, the mother reported decreased fetal movements and polyhydramnios was detected.

The boy was born by vaginal delivery at 39 weeks of gestation. The amniotic fluid was clear, the umbilical cord venous pH was 7.35 and the Apgar score at 1 minute was 4. The birth weight was 2400 g (P10), the head circumference was 32 cm (< P10).

The infant made no spontaneous movements, did neither suck nor swallow. In addition, there was a pronounced spasticity with a predominance of flexor tone in the arms and of extensor tone in the legs with bilateral subluxation of the hips. The infant required ventilation by mask and subsequently had to be intubated because of lack of spontaneous breathing. Cranial ultrasound on the first day of life showed a small subependymal echolucent focus („cyst") (Fig. 1). Magnetic resonance findings on the first day of life (T1 weighted) demonstrated increased signal in both thalami with a patchy appearance suggesting necrosis (Fig. 2). TORCH serology was negative. During the next days, neurological status (Sarnat Stage III) was unchanged and in view of the bad prognosis, the child was extubated on the 9th day of life and died 90 minutes later.

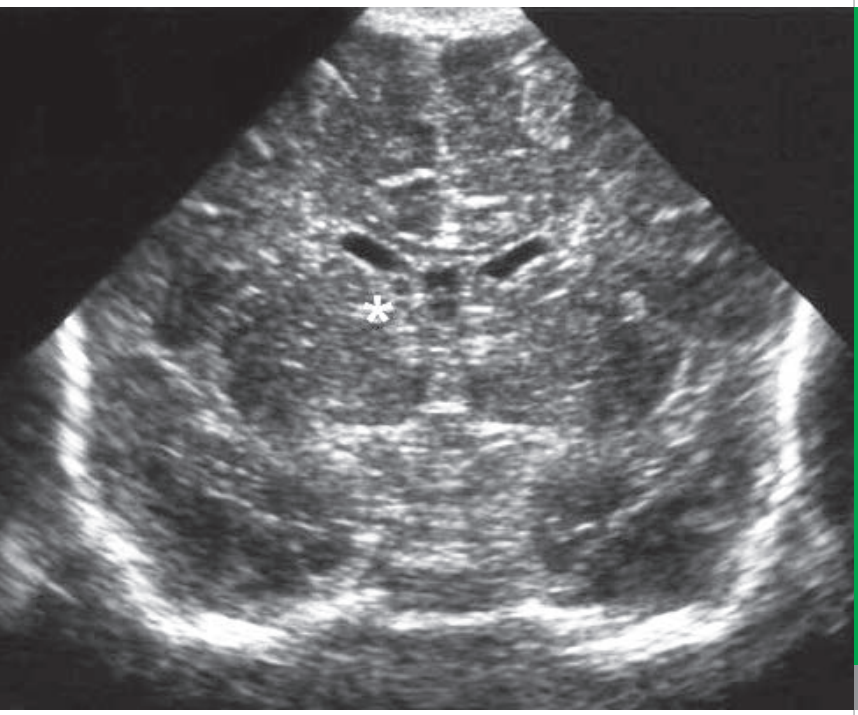

Fig. 1

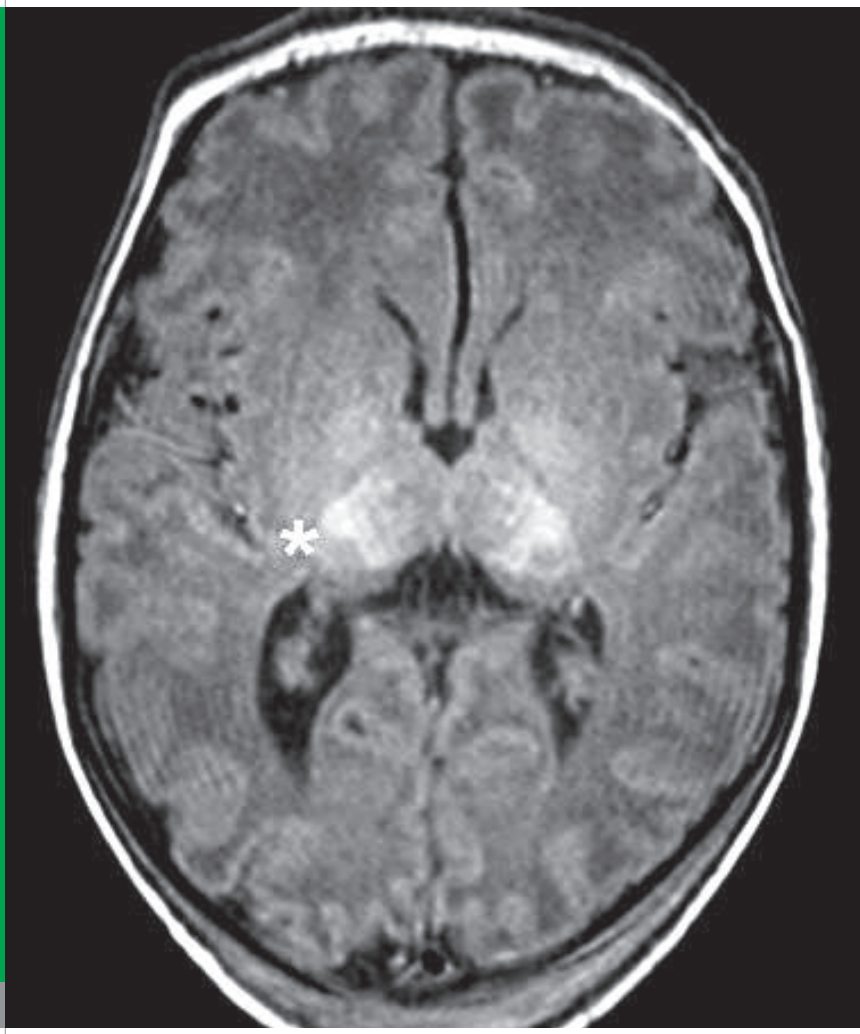

Fig. 2

Symmetrical thalamic lesions (STL) have a characteristic constellation of findings. Polyhydramnios is common and is likely to be caused by impaired intrauterine swallowing of amniotic fluid. This supports the idea of a prenatal origin of STL, which is also suggested by the presence of spasticity at birth. An acute hypoxic-ischemic event occuring two to four weeks before birth is considered a possible explanation for STL (1, 2). As a common feature, absence of suck, swallow, and primitive reflexes have been reported (2). Neuroimaging may be normal at an initial stage and has to be repeated on clinical grounds. The prognosis of STL seems very poor (2).

REFERENCES

1. Volpe JJ. Neurology of the newborn. 4th ed, 2001, WB Saunders Comp.

2. Eicke M, Briner J, Willi U, Uehlinger J, Boltshauser E. Symmetrical thalamic lesions in infants. Arch Dis Child 1992;67:15-19 (*Abstract*)

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