



Unusual Cranial Sonographic Findings in the Newborn: Lenticulostriate Vasculopathy

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Özet

Yenidoğanlarda nadir rastlanan kraniyal sonografik bulgular: Lentikülostriat vaskülopati

Lentikülostriat vaskülopati, neonatal dönemde rastlanan nadir bir sonografik bulgudur. Özellikle intrapartal kafa travması, neonatal hipoksi ve iskemi, TORCH enfeksiyonları, neonatal hipoglisemi, trizomi 13 ve 21, ensefalit, sifiliz, neonatal lupus, ikizden-ikize transfüzyon, intrauterin kokain maruziyeti ve fetal alkol sendromunda izlenir. Yazımızda, bu sonografik bulgu 9 olgumuz nedeniyle tartışılmıştır.

Brief Report:

Stripes of high echogenicity in the basal ganglia and thalamus of newborn infants have been rarely observed on cerebral ultrasound. Of newborn infants who undergo cranial ultrasonography, 0.27 to 0.42% exhibit hyperechogenic

lesions in the basal ganglia and thalamus (1,2). These lesions, which appear to be due to a non-calcific vasculopathy, are caused by fetal head injury at birth, neonatal hypoxia and ischemia, TORCH infections, neonatal hypoglycemia, trisomy 13 and 21, encephalitis, syphilis, neonatal lupus, twin-twin transfusion, intrauterine cocaine exposure, and

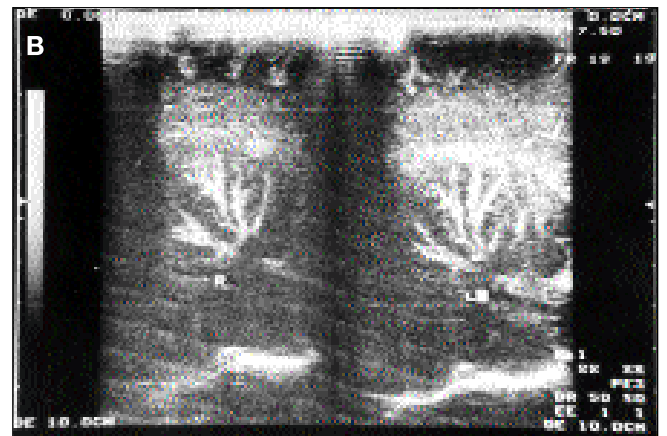


Figure 1. The typical sonographic pattern of lenticulostriate vasculopathy in an infant having hypoglycemia.

A, Coronal view, gray scale. There is bright linear echogenicity (,) lateral to the thalami, bilaterally.

B, Parasagittal view, gray scale. Bright linear echogenicity with ramifications radiating in basal gangliae.

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fetal alcohol syndrome, in the majority of cases (3). The echogenic foci follow the distribution of the lenticulostriate arteries. The exact cause is unknown, but histopathologic studies have shown deposits of amorphous basophilic material within the vessel walls. The clinical significance in terms of the neurodevelopmental outcome of this radiological abnormality is unknown. One study has shown no significant difference between the average Developmental Quotient of the target population and the normal population in regard to developmental status (4). When associated with etiologies such as TORCH infections, asphyxia, chromosomal abnormalities, such lesions are probably followed by a poor developmental outcome. Although non-specific, these findings should alert the physician to the possibility of congenital infection or chromosomal abnormality. These patients warrant complete screening for possible in utero infection and perhaps also chromosomal analysis.

We describe this finding (Figure 1) in 9 infants, in 3 with microcephaly, in 1 premature birth (34 weeks' gestation), and 5 having hypoglycemia. We suggest that sonographic lenticulostriate vasculopathy is a nonspecific marker of a previous insult to the brain.

References

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