Periventricular Leukomalacia.
Ultrasonographic diagnosis and follow-up
Published on 24.04.2001

DOI: 10.1594/EURORAD/CASE.819
ISSN: 1563-4086
Section: Paediatric radiology
Imaging Technique: Ultrasound
Case Type: Clinical Cases
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Patient: 1 weeks, female

Clinical History:

A very low birth weight premature infant under mechanical ventilation for 34 days, with high oxygen needs, episodes of cyanosis and electrolyte disturbances, developed spasticity and left facial nerve palsy 4 months after birth.

Imaging Findings:

A very low birth weight (920gr) girl, born at 27 weeks gestation was delivered by cesarean section. The infant was the second child of a twin pregnancy and was transferred to the neonatal intensive care unit intubated. She remained for 34 days under mechanical ventilatory support due to high oxygen needs and episodes of cyanosis. The situation was complicated with electrolyte disturbances, which were corrected on occasion. The infant developed spasticity and left facial nerve palsy at 4 months of age. Serial cranial ultrasonograms were performed at 1 week, 1, 2 and 3 months of age with 5 and 7 MHz transducers through the anterior fontanelle.

Discussion:

Periventricular Leukomalacia (PVL) is defined as infarction of the periventricular white matter in the preterm infant or preterm fetus, leading to deep white matter necrosis that may be followed by cavitation and cyst formation. PVL is the main form of ischemic brain injury in premature infants, with an incidence increasing with prematurity, low birthweight and survival longer than one week on assisted ventilation. At approximately 28 to 34 gestational weeks, the maturation of the centrifugal arteries is not complete and the vascular supply to the periventricular area that is considered the watershed area, is tenuous. The most severely involved areas include the occipital area, the frontal periventricular white matter at the level of the foramen of Monro. Transcranial ultrasound (US) is a very useful tool for early diagnosis of hemorrhagic PVL with an accuracy of 78-100%. US does not have any ionizing radiation effects and can be performed in the Intensive Care Unit without the need to transport the infant. Increased periventricular echogenicity caused by hemorrhagic infarction and edema of the affected area is the initial sonographic finding in PVL. When mild, increased periventricular echogenicity is a non-specific finding, and should be differentiated from the "normal periventricular halo" in an infant that will never develop PVL. In normal neonates a hyperechoic trigone posteriorly to the occipital horns of the lateral ventricles is usually identified. The distribution and degree of echogenicity in relation to the echogenicity of the choroid plexi can be useful for the differential diagnosis. Confident diagnosis of ischemic PVL is difficult and most patients with mild to
moderate lesions escape detection during the neonatal period, possibly with the exception of patients studied with MRI. More precise sonographic findings are periventricular cysts that appear later around the ventricles in the previously echogenic areas. These cysts are usually observed fourteen days or more after acute brain ischemia and may be seen for only a 2- to 3-week period. Therefore, when one is confronted with the dilemma of “normal periventricular halo” or early PVL, one could perform a follow-up US scan within 4-6 weeks. Late neurosonographic screening on the 28th day of life or later is recommended for detection of PVL in all premature infants regardless of birth weight, clinical course or a normal initial sonogram. PVL has been graded depending on the ultrasonographic appearances into 3 grades: Grade I: periventricular areas of increased echogenicity for 7 days or more. Grade II: periventricular areas of increased echogenicity that evolve into small cysts frontoparietally. Grade III: periventricular areas of increased echogenicity that evolve into extensive periventricular cysts fronto-parietally and occipitally. Eventually the cysts are replaced by scars, are incorporated into the lateral ventricles and disappear. The ventricles dilate due to cerebral atrophy. The typical pattern of PVL-related-atrophy with reduction of periventricular white matter, atrophic ventricular dilatation and occasionally cystic spaces around the ventricles can be seen with US, such as in our case, but is readily detected and recorded later during infancy by CT or MRI. MRI can also show evidence of delayed myelination and gliosis in the remaining periventricular white matter. Depending on the site of the lesion, PVL affects the visual tracts causing visual impairment, the auditory radiations and corticospinal tracts causing cerebral palsy. Thus the usual clinical sequelae are spastic diplegia or quadriplegia and cortical blindness with relative preservation of cognitive functions. Intellectual impairment has also been reported. Neurological deficits and convulsive disorders may develop later, generally months after the child has left the nursery. During the neonatal period there are no specific neurological abnormalities pathognomonic for PVL. Mental retardation and cerebral palsy occur in infants with large bilateral cysts while even smaller, focal, unilateral cysts are associated with a higher risk of mental retardation or cerebral palsy.

**Differential Diagnosis List:** Periventricular Leukomalacia

**Final Diagnosis:** Periventricular Leukomalacia

**References:**


**Description:** Increased periventricular echogeneity located around the frontal horns (arrow) shown on coronal anterior plane. **Origin:**
**Description:** Increased periventricular echogenity located at the angles of the lateral ventricles (arrows), shown on coronal posterior plane. The degree of white matter echogenicity was similar to that of the choroid plexi. **Origin:**
Description: Coronal scan showed multiple periventricular cysts, mainly located around the frontal horns and the trigones of the lateral ventricles (arrows). Origin:
Description: Multiple cystic areas of various size were re-identified at the deep white matter (arrowheads), shown on parasagittal plane. Origin:
Description: A larger area of low echogenicity was identified at the right hemisphere (arrow) on coronal plane. Origin:
Figure 4

Description: Coronal posterior scanning shows PVL-related atrophy with loss of white matter around the occipital horns. Origin:
Description: Distortion of the contour of the lateral ventricles due to incorporation of the necrotic cysts into the lateral ventricles shown on anterior coronal plane (arrow). Origin: