Periventricular leukomalacia

This article has been translated from WikiSkripta; ready for the **editor's review**.

Periventricular leukomalacia (PVL) is an ischemic necrosis of the brain's white matter adjacent to the lateral ventricles that affect premature neonates. It is most often diagnosed during an ultrasound examination of a newborn's brain, where periventricular echo-densities or cysts are visible. Magnetic resonance can also demonstrate a diffuse non-cystic form of PVL. It can lead to the development of cerebral palsy, impaired intellect, or visual impairment. Newborns born before the 32nd week of pregnancy are most at risk.[1][2]

In the case of hypoxic-ischemic brain damage before the 20th gestational week, **neuronal migration disorders** arise, between the 26th and 34th gestational weeks a picture of **periventricular malacia** appears, and between the 34th and 40th gestational week it is **diffuse cortical damage** or more often **focal ischemic brain damage.**[3]

Etiopathogenesis

PVL develops on the basis of ischemic damage to the cerebral white matter in the area adjacent to the lateral cerebral ventricles of the developing brain. It is the result of damage to oligodendrocytes in the a. cerebri media - in the area of deep penetrating arteries. This is the dividing area between the centripetal and centrifugal arterioles, moreover, the vascularization of this area is not yet complete in immature children. In this area, they have fewer vascular anastomoses and lack autoregulation of cerebral blood flow. Oligodendrocyte damage occurs as a result of hypotension, ischemia, and coagulation necrosis. Free radicals and/or excitotoxicity is involved in oligodendrocyte damage. In addition to ischemia, it appears that damage from cytokines released during intrauterine infection may also be the cause of PVL.[2][3]

Causes of PVL include hypoxia, ischemia (perinatal asphyxia, prolonged apneic pauses), and prenatal infections (acute chorioamnionitis, sepsis, meningitis).[1]

Clinical picture

In the acute stage, it is often asymptomatic. It can be manifested by hypotonia, apnoeic pauses, bradycardia, lethargy or, conversely, irritability.[1][2]

60-100% of children with PVL later develop cerebral palsy, typically with spastic diplegia of the lower extremities. Severe forms of PVL can also result in quadriplegia with mental disability, psychomotor retardation and visual impairment (such as nystagmus, strabismus, blindness).[2]

Diagnostics

- Ultrasound examination of the CNS, magnetic resonance, CT.
- Histological picture: loss of oligodendrocytes and proliferation of astrocytes in leukomalacia lesions.[2]

Treatment

There is no causal treatment.[2]

Links

Related Articles

- Hypoxic-ischemic encephalopathy

External links

- Atlas of the pathology of immaturity (https://atlases.muni.cz/atlases/novo/atl_cz/main+novorozenec+patolnezral.html)

References

1.
2.
3. https://atlases.muni.cz/atlases/novo/atl_cz/main+novorozenec+patolnezral.html