Hypoxic ischemic brain injury in neonates - early MR imaging findings

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Learning objectives

To learn about imaging findings in early brain MRI of newborns that suffered from mild to severe hypoxic ischemia during birth, focusing on diffusion weighted imaging. The aim of this poster is to outline the different findings depending on gestational age and severity of the hypoxic event.
Background

Hypoxic-ischemic injury is the most common cause of encephalopathy in neonates occurring in up to 6 of 1000 live births (1). It is usually due to reduced cerebral blood flow caused by fetal or neonatal cardiovascular compromise. The pathogenesis for such a compromise can be multifactorial. In case of intrauterine impairment it can be either of fetal origin (fetal thrombosis, fetal bradycardia), or inadequate placental perfusion (placental abruption, preeclampsia, maternal hypotension) or maternal origin (impaired maternal oxygenation due to asthma, pulmonary embolism)(2). Hypoperfusion and hypoxia lead to metabolic acidosis. In addition, they cause the release of inflammatory mediators and other biochemical substances that result in loss of cerebral autoregulation and neuronal cell death (3).

Typically, the areas that are damaged and, consequently, the expected changes in brain imaging depend on the severity and duration of the hypoxic event as well as on the brain maturation status (2). It is important to promptly and accurately identify neonates who have sustained a hypoxic-ischemic brain injury to facilitate optimal management. Hence, an early brain MRI is mandatory and its correct interpretation essential (4). In this poster we present typical findings in early brain MRI of term and preterm neonates depending on the severity of the ischemic event.
Fig. 1: Preterm newborn (delivered at week 30 post conception) with mild hypoxia during birth. Axial diffusion weighted, ADC and T2-weighted images of the brain on day 4 of life. Diffusion weighted imaging shows diffusion restriction in left paraventricular region, seen as low values on the corresponding apparent diffusion coefficient (ADC) map. On T2 weighted image no abnormalities are seen at that moment.

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Findings and procedure details

The most sensitive imaging technique during the first few days of life (especially day 3-6) is magnetic resonance imaging, especially diffusion weighted imaging performed with apparent perfusion coefficient map (ADC-map)(2)(5). Cytotoxic edema representing areas of ischemic injury can be detected as restricted diffusion (bright signal on perfusion weighted imaging and signal loss on ADC-map)(6). Compared with alterations that conventional imaging can pick up, perfusion restriction can be seen earlier (7). The areas of cytotoxic edema can show a characteristic pattern depending on the severity of the ischemic event and the maturation status of the brain.

In case of mild hypoxia due to prolonged partial asphyxia, cerebral blood flow is redistributed to the highly metabolically active grey matter such as the basal ganglia. This redistribution results in a deficit of blood supply which is particularly pronounced in the border zones between two vascular territories (2). In the immature brain there is a border zone in the periventricular white matter because it is supplied by cortical arteries extending inwards into the brain parenchyma. In preterm infants with mild hypoxia the cytotoxic edema is, therefore, usually located in the periventricular white matter (Fig. 1 and 2). With the maturation of the brain there will be a development of vessels extending from the lateral ventricles into the brain parenchyma resulting in a peripheral shift of the intervascular border zones towards the parasagittal region. It is the watershed zone between the anterior- middle and the posterior-middle cerebral arteries. Restricted diffusion in term neonates with mild hypoxia can therefore be seen primarily in the cortex and the underlying white matter of the parasagittal region. Lesions can be located uni- or bilateral, anterior or posterior (Fig. 3 and 4).

If there is an acute event, for instance a ruptured uterus, placental abruption or a prolapsed cord resulting in near total asphyxia and severe hypoxic injury, different patterns of brain damage can be expected (1). There will be no more redistribution of blood but a complete lack of blood supply. The absence of oxygen results in a particular suffering of the highly metabolically active areas which are usually the areas of myelinization.

Myelinization is a process that begins in the fetal period in the brainstem and cerebellum and terminates almost completely by the age of about 2 years. In the premature brain the highly metabolically active areas are therefore located in the brainstem, thalami and cerebellum whereas in the term neonate the myelinization has already proceeded. Typical areas of high metabolic activity and the term neonate are the posterior limb of the internal capsula, the lateral thalamus, posterior putamina, hippocampi, corticospinal tracts and the sensorimotor cortex (perirolandic cortex). Hence, perfusion restriction in preterm infants with severe hypoxic brain injury is usually located in brainstem and cerebellum whereas characteristic locations of perfusion restriction in term infants are the perirolandic cortex and the deep gray matter nuclei of the basal ganglia (Fig 4-7). Needless to say, there can be overlapping findings and exceptions from this general tendency. In addition, deep gray matter pattern can be associated to another pattern linked with severe encephalopathy which is called total cortical injury. This can also be an
isolated finding. In case of total cortical injury there will be diffusion restriction involving the whole cortex resulting in a so called "white cerebrum"(1) (Fig.8).
**Fig. 1:** Preterm newborn (delivered at week 30 post conception) with mild hypoxia during birth. Axial diffusion weighted, ADC and T2-weighted images of the brain on day 4 of life. Diffusion weighted imaging shows diffusion restriction in left paraventricular region, seen as low values on the corresponding apparent diffusion coefficient (ADC) map. On T2 weighted image no abnormalities are seen at that moment.

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**Fig. 2:** Preterm newborn (delivered at week 33 post conception) with mild hypoxia during birth. Axial diffusion weighted, ADC and T2-weighted images of the brain and lateral ventricles on day 5 of life. Diffusion weighted imaging shows diffusion restriction in right periventricular region, seen as low values on the corresponding apparent diffusion coefficient (ADC) map. On T2 weighted image there is a subtle hypointensity in the right periventricular region.

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**Fig. 3:** Term neonate with mild hypoxia during birth. Axial diffusion weighted image and ADC map of brain and lateral ventricles on day 5 of life. There is bilateral diffusion restriction in the parasagittal white matter and cortex corresponding to ischemic lesions in the watershed zones between middle and anterior and middle and posterior cerebral arteries.

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**Fig. 4:** Term neonate with mild hypoxia during birth. From left to right: Three axial diffusion weighted images and axial ADC map of the frontoparietal region on day 4 of life. There are various foci of diffusion restriction in the parasagittal region of the left frontal and parietal lobe.

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Fig. 5: Preterm newborn (delivered at week 32 post conception) who suffered from severe hypoxic ischemic event. Axial diffusion weighted and T2-weighted images of the brainstem on day 3 of life. Diffusion weighted imaging shows diffusion restriction in brainstem. Notice the beginning myelinization of that area seen as hypointensity on the corresponding T2 weighted imaging.

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Fig. 6: Term newborn with severe hypoxia during birth. Axial diffusion weighted, ADC and T2-weighted images of the basal ganglia on day 5 of life. Bilateral focal diffusion restriction in globus pallidus and putamen. Nor abnormality seen on T2-weighted images. Note the beginning of myelinization in the posterior limb of the internal capsule - a sign of myelinization statues normal for gestational age (40 weeks).

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Fig. 7: Term newborn with severe hypoxia during birth. Axial diffusion weighted image and ADC-map on day 4 of life. Diffusion restriction and low ADC values are seen in parietal cortex bilateral corresponding to the perirolandic region.

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**Fig. 8:** Total cortical injury in a term newborn with severe asphyxia due to rupture of placenta. There is diffuse cortical diffusion restriction seen in both hemispheres.

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Conclusion

It is essential to identify neonates with ischemic encephalopathy to facilitate optimal treatment. Knowing the characteristic patterns of hypoxic ischemic encephalopathy in neonates is useful for interpreting MR imaging findings, especially in order to identify ischemic lesions, exclude other causes of neurologic deficits and estimate the severity and prognosis.
References


