Hypoxic-Ischemic encephalopathy in adults

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Learning objectives

- Identify the various findings CT, and MR imaging features of hypoxic-ischemic brain injury.

- Describe the appropriate imaging work-up for suspected hypoxic-ischemic brain injury.
Background

The brain is our most essential organ but also the most sensitive to oxygen deprivation. Hypoxic-ischaemic encephalopathy (HIE) can be a devastating neurological injury and prompt recognition of it can result in significant changes in patient management. HIE insults develop in varying regions of the brain depending on the severity and duration of hypoperfusion or hypo-oxygenation.  

Diffuse hypoxia and ischemia result in global cerebral damage that follows a typical pattern defined by the selective vulnerability of brain regions. Irreversible injury occurs when systemic blood pressure drops below the minimal levels required for sustaining effective brain metabolism and energy production.

Brain parenchyma changes have corresponding MR signal characteristics that are often obvious but can be subtle. It is important for radiologists to be aware of the variations in the appearance of HIE in order to be alert to the diagnosis in subtle cases, recognise unusual patterns and be aware of the clinical ramifications.

- Topographic distribution of the lesions can help exclude other causes of neurologic impairment.
- Widespread symmetric pattern of injury that often involves the deep gray nuclei and cerebral cortex is seen.
- Imaging findings in HIE are highly variable and depend on a number of factors, including brain maturity, severity and duration of insult, and type and timing of imaging studies.
Findings and procedure details

The most characteristic example of hypoxic-ischemic brain damage is produced by cardiac arrest. Attempts to prognosticate outcome accurately after cardiac arrest have generated abundant research. Although clinical examination remains the preeminent tool to predict the chances of recovery after cardiac resuscitation, a number of electrophysiological and neuroimaging techniques provide valuable aid.\(^\text{2,3}\)

Mild to moderate global ischemic insults to the brain usually result in watershed zone infarcts.\(^\text{4,5}\)

Severe HIE, in this population primarily affects the gray matter structures:

- The basal ganglia
- Thalami
- Cerebral cortex (in particular the sensorimotor and visual cortices, although involvement is often diffuse)
- Cerebellum
- Hippocampi.

This predominance of gray matter injury is related to the fact that gray matter contains most of the dendrites where postsynaptic glutamate receptors are located and are, therefore, the sites most susceptible to the effects of glutamate excitotoxicity. As a result of synaptic activity, gray matter is also more metabolically active than white matter. Although cerebellar injury can be seen in neonates, it tends to be more common in older patients. The reason for this predilection is not entirely clear, but it has been suggested that the relative immaturity of Purkinje cells (which are normally exquisitely sensitive to ischemic damage) in neonates somehow protects the cerebellar cortex.

Computed tomography (CT) scan has limited sensitivity to diagnose the extent of brain damage after a diffuse hypoxic insult.

CT findings include:

- Loss of the normal differentiation between cortical gray matter and subcortical white matter and effacement of the delineation of deep gray matter structures are the best known signs of global hypoxia on CT scan.

- Decreased bilateral basal ganglia attenuation Fig. 1 on page 8.

They represent early stages of brain swelling, mostly due to cytotoxic edema. Fig. 2 on page 8.
However, these findings may be subtle and difficult to recognize. Additionally, CT scans can be deceiving, showing little change in patients with severe hypoxic damage or presenting signs that may be confused with other conditions. In patients who develop areas of infarction, CT scans may fail to reveal any focal hypodensities until 24 to 48 hours after the episode. Fig. 3 on page 9

In older patients, CT is generally the initial imaging study performed when brain injury is suspected.

As in young children, the reversal sign and the white cerebellum sign may be seen in adults and indicate severe injury with a poor prognosis Fig. 4 on page 10.

Diffusion-weighted MR imaging is the earliest imaging modality to become positive, usually within the first few hours after a hypoxic-ischemic event. During the first 24 hours, diffusion-weighted imaging may demonstrate increased signal intensity in the cerebellar hemispheres, basal ganglia, or cerebral cortex (in particular, the perirolandic and occipital cortices). Fig. 9

Early cytotoxic edema in these injured cells is responsible for the bright signals seen on DWI and the corresponding low apparent diffusion coefficient (ADC) values.\textsuperscript{6} Fig. 6 on page 12, Fig. 7 on page 13.

The thalami, brainstem, or hippocampi may also be involved. As in younger patients, conventional T1- and T2-weighted images are often normal or demonstrate only very subtle abnormalities.

In the early subacute period (24 hours- 2 weeks), conventional T2-weighted images typically become positive and demonstrate increased signal intensity and swelling of the injured gray matter structures, although these findings may be subtle. As mentioned earlier, diffusion-weighted imaging abnormalities usually pseudonormalize by the end of the 1st week.

Gray matter signal intensity abnormalities at conventional MR imaging may persist into the end of the 2nd week. In the chronic stage, T2-weighted images may demonstrate some residual hyperintensity in the basal ganglia, and T1-weighted images may show cortical necrosis Fig. 8 on page 14, which is seen as areas of high signal intensity in the cortex.

Acute phase
Hyperintensity on DWI involving the cortical component (mainly watershed areas) and basal ganglia (selective vulnerability) due to cytotoxic edema is the most relevant finding. Cytotoxic edema is characterized by restricted water diffusion, which increases the signal intensity on DWI and reduces the ADC.

Early subacute phase

The cortex and basal ganglia show high signal intensity on DWI and T2-weighted images (T2WI), indicating concomitant cytotoxic and vasogenic edema. Progressive signal intensity loss on DWI is observed usually after 6 days due to ADC pseudo-normalization. In this situation, the likely irreversible tissue damage appears hyperintense on T2WI and isointense on ADC maps.

Late subacute phase

At this phase, there is no more brain swelling, and atrophic changes progress through affected areas resulting in cerebrospinal fluid spaces dilatation and progressive increase of ADC values of GM, depicted as normal or slightly high signal on DWI. Hypointense white matter areas may be present on T2WI and are secondary to iron deposition due to axoplasmic flow failure and loss of control of the intracellular iron pool during reperfusion.

Chronic phase

The main finding is brain volume loss, reflecting atrophy, associated with white matter hyperintensity on T2WI and FLAIR images, and isointensity or hyperintensity on DWI. The hyperintensity is explained by the "T2 shine through" effect and does not represent true diffusion restriction. Paramagnetic substances can accumulate in the basal ganglia leading to hypointense foci on T2WI. Signs of laminar necrosis in the cortex and basal ganglia are present in a variable fashion.

- The high signal intensity on T1WI due to accumulation of fat-laden macrophages generally lasts up to 2 months, but can persist for 6 months, and is infrequent thereafter. Gadolinium enhancement is usually not observed after 2 months.

- Hyperintensity on T2WI is due to gliosis, which can affect structures that were damaged in the early phases, such as the subcortical and deep white matter, thalami, and basal ganglia.
Fig. 1: A 33-year-old female who experienced a cardiorespiratory arrest and was without respiration at least 10 min. Axial non-contrast computed tomography (CT) of the basal ganglia showing symmetrical hypodensity in the caudate nuclei.

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**Fig. 2:** Female, 77 years old, victim of cardiac arrest. Early subacute CT examination showing diffuse edema, sulci effacement, low GM/WM differentiation, and hypodense basal ganglia.

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Fig. 3: 49 year old male patient with severe chronic obstructive pulmonary disease 12 h after prolonged cardiac arrest. a),b) Axial non-contrast CT of the basal ganglia showing symmetrical hypodensity in the caudate nuclei. c) coronal.

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**Fig. 4:** a),b). Axial CT demonstrate hypodense of widespread white matter that respects the basal ganglia and the posterior fossa. Indicate severe injury with a poor prognosis.

Fig. 5: Diffusion-weighted imaging sequence of MR. Note restricted diffusion in the lenticular nuclei and cerebellum.

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Fig. 6: Diffusion-weighted imaging sequence (right) (b) and corresponding apparent diffusion coefficient maps (left) (a) of MR. Note restricted diffusion in the lenticular nuclei and cerebellum.

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**Fig. 7:** A 55 year old male who had severe cardiopulmonary failure secondary to sepsis. Diffusion-weighted images show high signal intensity cerebral cortex, basal ganglia and thalami. Occipitoparietal areas are clearly more involved than other cortical areas. (arrows)

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Fig. 8: a) Coronal Flair sequence an b) axial T2 disclosing hyperintense signal in the basal ganglia and cerebellum indicative of laminar necrosis.

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Conclusion

The anatomical site of tissue injury varies following a concept known as selective vulnerability. Particular sites with high energy demands face cytotoxic crisis when presented with mandatory anaerobic metabolism due to low oxygen delivery. It is well known that Diffusion-weighted MR imaging is the most sensitive imaging modalities in the early hours following injury. In clinical practice, imaging could be considered an outcome predictor in HIE when definitive clinical, electrophysiological, or biochemical indicators of poor clinical outcome are negative or inconclusive.

However, even severe cases of HIE may have normal or subtle findings on DWI initially. That is why a radiologist must be very aware of the potential diagnosis, know the patterns that are seen and be able to detect subtle findings of diffuse HIE.
References


