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A pictorial review of CT imaging features in hypoxic brain injury

R. Vaja, E. Carr, W. Topping, A. Patel, K. Jain (Lister Hospital, Stevenage, UK)

Introduction

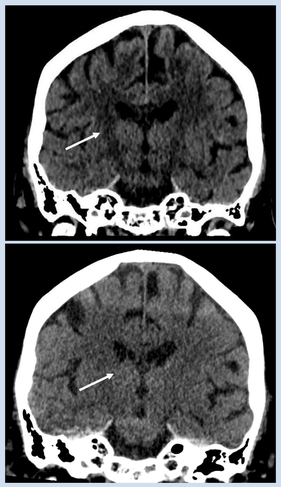
Hypoxic brain injury (HBI, also known as hypoxic-ischemic or anoxic brain injury) in adults is seen after a global reduction in oxygen supply to the brain tissues, commonly caused by cardiac arrest. Other causes of cerebral hypoperfusion can also result in HBI e.g. sepsis, intraoperative hypotension, and carbon monoxide poisoning (1).

Imaging plays a key role in assessing the stage and severity of injury, and supports clinical examination and EEG in prognostication, and therefore guides management of the patient (2). CT is often the first step in imaging due to its accessibility.

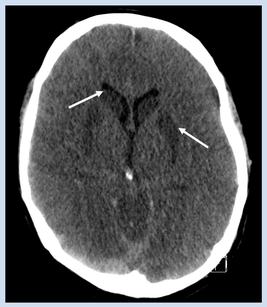
Certain areas of the brain are more vulnerable to injury due to their increased metabolic activity /oxygen requirement, typically grey matter structures. The CT imaging features at each stage of the condition reflects this, and will be demonstrated in the selected cases.

Hypodense basal ganglia

The basal ganglia are often the first structures affected in HBI, as they are metabolically active grey matter structures. Cell death in the lentiform nuclei leads to cytotoxic oedema, which causes the nuclei to appear iso- or hypo-attenuating to surrounding brain tissue.



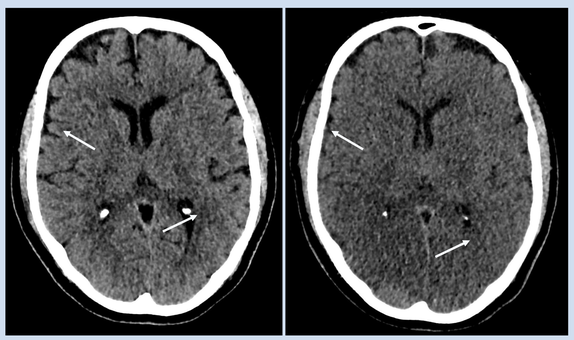
➤Figure 1a, b: CT head 48♂ following out of hospital cardiac arrest (OOHCA). Coronal slices on day 0 (a) and day 4 (b) of admission, between which times the patient failed to regain consciousness. The basal ganglia demonstrate a reduced attenuation in image 1b, rendering them isodense to surrounding tissue. This stage precedes the classical 'hypodense basal ganglia' appearance (arrows).



▲Figure 2: Unenhanced axial CT head. 69♂ who had an OOHCA. Note the hypodense appearance of the head of caudate and putamen (respective arrows).

Generalised loss of grey-white matter differentiation

The cortical grey matter becomes oedematous, and the usual differentiation between the grey and white matter is no longer made on CT. Although this is easier to identify in acute territorial infarction, it is more difficult to appreciate when the brain is globally affected. This is a later finding than the hypodense basal ganglia sign.

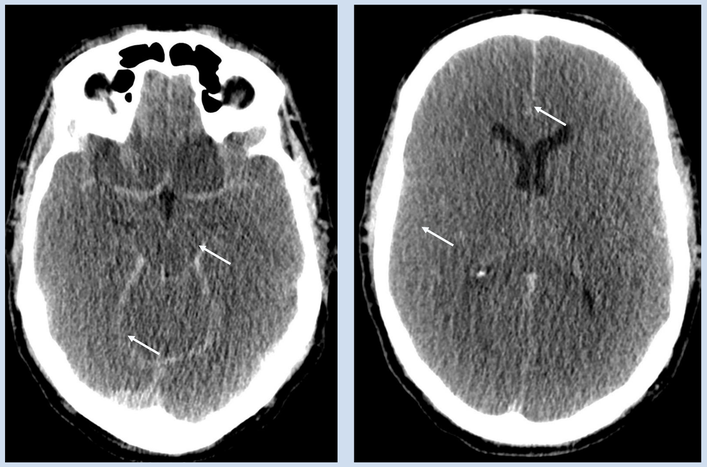


➤Figure 3a, b: Axial unenhanced CT head slices of a 67♂, 5 hours and 2 days post OOHCA respectively. Note the interval loss of the definition of the cortex (arrows). The hypodense basal ganglia sign is also present.

Pseudo-subarachnoid haemorrhage sign

This sign should be described with care, as the radiologist should consider the possibility that there is true subarachnoid blood present, based on the clinical details and presence of ancillary features. In HBI, the appearance of hyperdense subarachnoid space is created by the raised intracranial pressure causing obliteration of the cisterns and close apposition of the meninges and veins, which appear bright compared to the adjacent oedematous brain parenchyma. Associated features of raised intracranial pressure include effacement of the ventricles and cerebellar tonsil herniation.

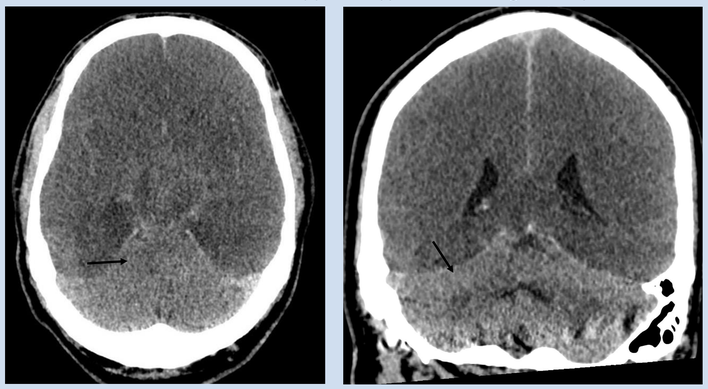
➤Figure 4a, b: unenhanced axial slices of a 48♂ following prolonged cardiac arrest. There is a dense appearance to the subarachnoid space with effacement of the basal cisterns. Superiorly the sulci are also effaced with the appearance of high density in the sulcal space.



White cerebellum sign

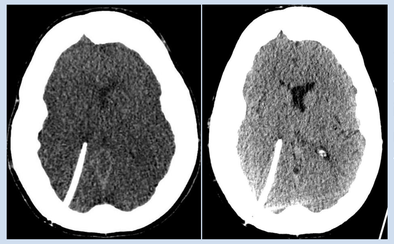
Although the term 'white cerebellum' implies that the abnormal tissue is cerebellum, it actually describes the contrast seen between the oedematous and hypoattenuating cerebral hemispheres and the normal cerebellum. This causes the spared cerebellum to appear 'whiter' than the rest of the brain. It has been suggested that blood flow is diverted to the posterior circulation in preference during a hypoxic insult. The finding carries a very poor prognosis (2, 3).

➤ Figure 5a, b: Axial and coronal CT head of a 56♂, 5 days post cardiac arrest. The supratentorial brain tissue has a much lower attenuation with loss of grey-white matter differentiation. The infratentorial brain appears hyperattenuating in comparison.



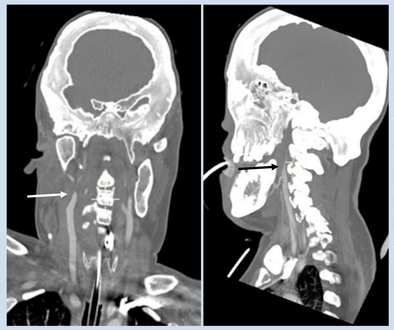
Reversal of cerebral blood flow (CT angiogram)

In advanced hypoxic brain injury, the raised intracranial pressure prevents the arterial flow of blood into the brain.



➤ Figure 6a, b: Unenhanced axial CT head of a 39 year old man day 2 post-arrest (a) and 1 year pre-arrest for comparison (b). VP shunt is in situ. There is interval loss of grey-white matter differentiation and slight effacement of the ventricles.

➤ Figure 6c, d: arterial phase axial CT, day 2 post-arrest, bone and brain windows. The patient also has hyperostosis of the skull (c). Note the complete effacement of the basal cisterns (d). No contrast is demonstrated in the parenchymal or vascular spaces.



➤ Figure 6e, f: Arterial phase coronal and sagittal plane MIPs to demonstrate the course of the internal carotid arteries. There is gradual tapering of the arteries as they approach the skull base. This is due to the intracranial pressure exceeding the arterial pressure. In this particular case it is possible that the hyperostosis has exacerbated the reversal of flow due a reduction in cranial fossa volume and a degree of carotid canal stenosis.

Conclusion

This poster has highlighted both the subtle and overt signs that are associated with HBI, to enable recognition of a hypoxic injury when it might not have been suspected clinically e.g in sepsis or post-operatively. In particular the pseudo-subarachnoid sign, when thought to be present, is to be evaluated carefully to weigh up the probability of true subarachnoid haemorrhage versus hypoxic brain injury.

Although there are multiple features of hypoxic brain injury that can be appreciated on CT, unfortunately these features manifest late and are best appreciated about 48hrs after the insult. Therefore the use of CT in the setting of HBI is more helpful in confirmation of suspected HBI, and providing clinicians with additional information as to the stage of hypoxia and presence of certain signs that predict poorer outcomes.

References

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2) R.S. Howard P.A. Holmes A. Siddiqui D. Treacher I. Tsiropoulos M. Koutroumanidis. Hypoxic–ischaemic brain injury: imaging and neurophysiology abnormalities related to outcome. QJM: An International Journal of Medicine. 2012; Vol 105, Issue 6; 551–61.
3) Dwarakanath S, Bansal A, Rudrappa S, Gopal S, Venkatramana NK. White cerebellum sign - A case report and review of literature. J Pediatr Neurosci. 2006;1:22–3.