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Images in Medicine

Hypoxic–Ischemic brain injury in an adult: Magnetic Resonance Imaging findings

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Introduction

Hypoxic–ischemic brain injury (HIBI) is not uncommon in day to day clinical practice. It occurs in cases of cardiac arrest that are revived by cardiac resuscitation. Though the diagnosis of HIBI in these patients is never in doubt due to the sequence of events, however imaging is done to assess the amount of damage and to prognosticate the outcome. We present here magnetic resonance imaging (MRI) findings in a case of severe hypoxic ischemic brain injury.

Clinical and imaging findings

A 37 year old male patient, a known case of carcinoma hypopharynx with recurrence was admitted in hospital for

workup and further management. While in hospital, he developed sudden onset severe stridor at 1 O'clock in the late night and went into cardiac arrest. He was started on advanced cardiac life support immediately and resuscitated within 5 min of cardiac asystole. However he went into deep coma and his Glasgow coma scale was 3/15 after revival. The patient was placed on continuous life support. MRI brain was done three days later to assess the damage and to rule out any other concomitant cause. MRI brain revealed T2W and FLAIR hyperintensity in the basal ganglia bilaterally (Fig. 1a & b). Diffusion weighted images showed bright signal in Globus pallidus bilaterally with corresponding low signal on ADC map suggestive of restricted diffusion (Fig. 2a & b). T1WI images showed hyperintensity involving the perirolandic and right occipital cortex suggesting early cortical laminar necrosis (Fig. 3a). GRE images did not reveal any susceptibility artifacts. Post contrast images revealed gyriform pattern of enhancement in right occipital cortex and bilateral perirolandic cortex (Fig. 3b). Based on the MRI findings, diagnosis of HIBI was confirmed.

Discussion

Clinical diagnosis of HIBI is obvious due to sequence of events leading to such a situation. Neuroimaging plays a major role in work up of these patients in not only diagnosing the severity of damage but also helps in prognostication.

CT scan has a limited role in diagnosing the extent of brain injury. Loss of cortical gray white matter differentiation,

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Fig. 1 – (a) T2W Image showing hyperintense signal in bilateral basal ganglia. (b) FLAIR image showing hyperintense signal in bilateral basal ganglia.

Fig. 2 – (a) Diffusion weighted image showing bright signal in globus pallidus bilaterally. (b) ADC map showing corresponding low signal in globus pallidus bilaterally.

Fig. 3 – (a) T1W image (non-contrast) showing gyriform hyperintensity in bilateral perirolandic cortex consistent with cortical laminar necrosis. (b) Post contrast study (at the same level as in Fig. 3a) demonstrating gyriform enhancement in the bilateral perirolandic gyri.

reduced delineation of deep gray matter and effacement of cortical sulci may be the only signs of global hypoxia seen on CT which do not help in assessing the severity and determining the prognosis of the patient.

MR imaging is modality of choice due to its inherent ability to delineate the gray and white matter differentiation, identify the cytotoxic edema which not only helps in assessing the severity but also helps in predicting the prognosis. MR findings in HIBI depend on the duration, severity of insult and time period after which MRI is performed.

In adults, mild to moderate HIBI leads to water shed infarcts. Severe HIBI affects gray matter of the perirolandic cortical neurons, occipital cortex, medial prefrontal cortex, basal ganglia, thalami, hippocampus and cerebellum.¹ In acute stage (less than 24 h), affected cortex and deep gray matter may show restricted diffusion in the form of bright signal on. Diffusion Weighted Images (DWI) and corresponding low signal on apparent diffusion coefficient (ADC) map due to cytotoxic edema.² Restricted diffusion was seen in this case in bilateral globus pallidus (Fig. 2a & b). DWI starts to pseudo-normalize after one week.³ In early subacute stages (24 h–2 weeks) T2WI/FLAIR images start showing hyperintense signals in these areas as seen in this case (Fig. 1a & b). In chronic stages, T2W/FLAIR hyperintensity decreases, but the cortical laminar necrosis seen as gyriform hyperintensity on T1W images starts to become more prominent.⁴ Early evidence of cortical laminar necrosis was seen in our case as T1W hyperintensity involving the right occipital and perirolandic cortex (Fig. 3a). On contrast study gyriform pattern of cortical enhancement was seen (Fig. 3b) which is likely due to breakdown of blood brain barrier and impaired autoregulation.

Contrast enhancement is usually seen in such cases after 1–2 weeks, peaks after 1–2 months, and commonly resolves after 6 months.⁵ Early gyral contrast enhancement as seen in our case could be related to the severity of hypoxic brain damage leading to breakdown of blood brain barrier and reperfusion of the hypoxic ischemic brain. Overall, our case depicts MRI findings of HIBI in an adult patient as described in literature.

Conflicts of interest

The authors have none to declare.

REFERENCES

1. Norton L, Hutchison RM, Young GB, Lee DH, Sharpe MD, Mirsattari SM. Disruptions of functional connectivity in the default mode network of comatose patients. *Neurology*. 2012;78:175–181.
2. Els T, Kassubek J, Kubalek R, Klisch J. Diffusion-weighted MRI during early global cerebral hypoxia: a predictor for clinical outcome? *Acta Neurol Scand*. 2004;110:361–367.
3. Forbes KP, Pipe JG, Bird R. Neonatal hypoxic ischemic encephalopathy: detection with diffusion-weighted MR imaging. *Am J Neuroradiol*. 2000;21:1490–1496.
4. Takahashi S, Higano S, Ishii K, et al. Hypoxic brain damage: cortical laminar necrosis and delayed changes in white matter at sequential MR imaging. *Radiology*. 1993;189:449–456.
5. Siskas N, Lefkopoulos A, Ioannidis I, Charitandi A, Dimitriadis AS. Cortical laminar necrosis in brain infarcts: serial MRI. *Neuroradiology*. 2003;45:283–288.