Editorial

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The hypoxic-ischemic brain injury: Beyond semantics

Harjinder S Bhatoe, M Ch
Command Hospital (Western Command), Chandimandir Cantt. Panchkula 134107. Haryana

The term, hypoxic ischemic brain injury (HIBI) is bandied freely in intensive care and neurorehabilitation environment. Grossly, a patient, most often following an episode of respiratory failure develops features of encephalopathy (often explained as 'brain failure' to the lay people), and post recovery exhibits persistently vegetative state or other sequelae. Simplified explanation for the event is decreased oxygen delivery to the brain along with decrease in cerebral perfusion (although in carbon monoxide poisoning the cerebral perfusion may not be much affected). Another term often used to denote the injury is 'anoxic brain damage'. This term is best avoided, since it implies frank absence of oxygen in blood, which would be incompatible with survival. Moreover, the term ignores the equally important pathophysiologic event of ischemia. Even in the event of respiratory arrest, oxygen remains available for diffusion, albeit in diminishing quantities, for several minutes thereafter. Purely hypoxic brain injury (as in transient strangulation, near drowning, etc) will result in transient pathophysiologic disturbance, less severe brain damage[1]. HIBI accurately describes the event as well as the sequlae. As opposed to stroke, the disturbances in cerebral metabolism are global. Brain is starved of not only oxygen, but also of the other metabolic substrate, glucose. In addition, there is failure of nutrient/waste product exchange, resulting in an environment toxic to the cell and organelle. There is membrane depolarization, increase in intracellular calcium, release of excessive amounts of excitatory amino acids, cerebral edema and failure of autoregulation[1,2]. Sufficiently prolonged, the injury can lead to permanent neuronal damage. It is important to distinguish it from ischemic stroke, in which there is perfusion failure in one or more territories; hypoxic brain injury in contrast is a global phenomenon. Although the injury is global and widespread, certain areas are more vulnerable. These are, the superior brainstem, cerebellum, white matter, subcortical structures supplied by deep and superficial branches of penetrating blood vessels, the areas between the distribution of major arteries (watershed areas), hippocampus, layers 3,5 and 6 of neocortex, producing laminar cortical necrosis[1,3,4].

Address for correspondence:
Harjinder S Bhatoe
Command Hospital (WC), Chandimandir Cantt 134107
Panchkula (Haryana) Tel: 0172-2554786

Sequelae of hypoxic ischemic brain injury can be varied. Usually, the patient suffers from seizures (during the event, or as a consequence thereof). These are complex partial or myoclonic in character and occur due to excitotoxic process on the cortical neurons. While early seiaures do not portend HIBI-induced epilepsy, post HIBI status epilepticus carries a high mortality[5].

Movement disorders (Parkinsonism, dystonia, choreoathetosis, tremor) are rare after HIBI, and usually manifest late (months to years) after the initial cerebral insult. Post-HIBI Parkinsonism and dystonia are more common, and indicates the selective vulnerability of deeper nuclei to ischemic insult. Parkinsonism is usually akinetic-rigid type, that responds less readily to medical treatment as compared with primary Parkinsonism, indicating destruction of the neurons targeted in pharmacotherapy.

Disturbances motor function can occur due to involvement of corticospinal fibers in crura cerebri, leading to weakness of all four limbs. An interesting sequel to HIBI is the "man in the barrel" syndrome, in which there is preferential weakness of proximal upper limb musculature with sparing of hands and lower limbs. This occurs due to ischemic involvement of watershed zones between the territories of anterior and middle cerebral arteries. Impairment of vision may occur due to infarction of watershed zones in the posterior cortex. This can manifest as cortical blindness and Balint syndrome (ocular apraxia, optical ataxia, simultagnosia).

Cognitive disorders following HIBI are most exhaustively studied sequelae. The selective vulnerablity of cognitive areas results in a wide range of these disorders, ranging from disorders of consciousness (coma, persistent vegetative state), disorders of behaviour, mood and affect, impairment of attention, memory dysfunction,

execution of sequential tasks, etc. Recovery, though variable is to be anticipated over one or two years. Extreme cases of HIBI take the form of delayed ischemic leukoencephalopathy, which can occur after an apparent recovery from HIBI insult[7]. There is widespread bilateral demyelination, sparing the cerebellum.

Another important sequel of HIBI can be cerebral palsy: epidemiological studies of children with cerebral palsy have shown that 12-20% may be related to intrapartum hypoxic ischemic insult[8,9].

HIBI can result from a variety of non-traumatic and traumatic CNS insults. The severe sequelae present challenges for study of pathophysiology, and long-term management. Management essentially involves adaptation to disability, programmed re-entry into community and learning of new skills.

REFERENCES

1. Busl KM, Greer DM. Hypoxic-ischemic brain injury: pathophysiology, neuropathology and mechanisms. *NeuroRehabilitation* 2010; 26:5-13.
2. Arbelaez, A, Castillo M, Mukherji SK. Diffusion-weighted MR imaging of global cerebral anoxia. *AJNR: American Journal of Neuroradiology* 1999; 20:999-1007.
3. Burke DT, Shah MK, Dorvlo AS, Al-Adawi S. Rehabilitation outcomes of cardiac and non-cardiac anoxic brain injury: a single institution experience. *Brain Inj* 2005; 19:675-80.
4. Chalela JA, Wolf RL, Maljian JA, Kasner SE. MRI identification of early white matter injury in anoxic-ischemic encephalopathy.[see comment]. *Neurology* 2001; 56:481-5.
5. Calvert JW, Zhang JH. Pathophysiology of an hypoxic-ischemic insult during the perinatal period. *Neurological Research* 2005; 27:246-60.
6. Rossetti AO, Logroscino G, Liaudet L, et al. Status epilepticus: an independent outcome predictor after cerebral anoxia. *Neurology* 2007; 69:255-60.
7. Shprecher D, Mehta L. The syndrome of delayed post-hypoxic leukoencephalopathy. *NeuroRehabilitation* 2010; 26:65-72.
8. Nelson KB. Can we prevent cerebral palsy? *New Engl J Med* 2003; 349:1765-6.
9. Badawi N, Keogh JM, Allessandri LM, et al. Antepartum risk factors for newborn encephalopathy: the Western Australian case control study. *Br Med J* 1998; 317:1554-7.