

An unusual case of paediatric cerebral anoxia!

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We report a 2-year-old female child diagnosed as a case of multiple skull fracture with a frontal contusion. There was a history of loss of consciousness, bleeding from ear, nose and throat following a fall from height. On examination, her Glasgow Coma Scale (GCS) was 6 (E1V1M4), blood pressure was 48/25 mmHg, and heart rate was 180/min. Bilateral pupil was normal in size and sluggishly reacting to light. The computerized tomographic (CT) scan of head revealed multiple fractures including right parietal bone, left temporal bone, left lateral orbital wall, left sphenoid wing and right frontal contusion with depressed fracture and diffuse cerebral oedema [Figure 1]. However, the cervical spine injury was ruled out. The arterial blood gas analysis showed severe respiratory acidosis with compensatory metabolic alkalosis. After securing the airway, hypotension was managed with normal saline and inotropic support (dopamine infusion at 15 mcg/kg/min). For further management, she was shifted to the neurosurgical intensive care unit (NICU). In NICU, her GCS further decreased to 3 (E1VtM2). The surgical intervention was deferred due to deranged coagulation profile, low haemoglobin percentage, and unstable haemodynamic status requiring high dose inotropic support. Her course in NICU included transfusion of packed red blood cells and fresh frozen plasma along with osmotic diuretics and antiepileptics. A repeat CT scan head on next day was suggestive of diffuse hypoxic changes with effaced basal cisterns and significant cerebral oedema [Figure 2]. Her GCS continued to be 3 (E1VtM2). By observing the decreasing scale of GCS she was expected to remain on ventilator for a prolonged period, hence a surgical tracheostomy was performed on 4th postoperative day. She remained on a ventilator for next few days along with intravenous sedation. On 5th postoperative day, there was a slight improvement in her GCS from 3 to 4 (E1VtM3). Gradually, she showed the spontaneous

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movement of all four limbs (right > left) and spontaneous eye opening. She was weaned off from the ventilator on 14th postoperative day and shifted to ward on room air with a tracheostomy *in situ*. A repeat CT scan of the head showed normal study [Figure 3].

The brain is the most essential organ in human body. It is very sensitive to oxygen deprivation and hypoxia. Hypoxic-ischemic injury of the brain can occur due to cardiac arrest, profound hypotension or hypoxia. Moderately severe reduction in cerebral blood flow and oxygen supply suppress brain tissue metabolism whereas critically severe reduction can cause irreversible disruption of cellular membranes, cytotoxic oedema, and cell death. There may be raised the intracranial pressure (ICP) due to hypoxic brain swelling. In emergency situation the priority is maintaining arterial oxygen saturation, ensuring normal osmosis, as well as normoglycaemia.^[1] Otherwise, the first line treatment of raised ICP includes avoidance of pyrexia, seizure control, CSF drainage either through intraventricular catheter or lumbar drain, head end elevation of bed by 30°, an intravascular osmotic agent mannitol and hyperventilation. The clinical features and outcome of hypoxic-ischaemic brain injury depend on the severity of the initial insult and the effectiveness of resuscitation. The patient with anoxic brain injury will require drugs to maintain adequate blood pressure and normal heart beat. Convulsions are very common after anoxic brain injury. Hence, a full range of Intensive care will be needed in such patients to bring out the best possible outcome. Therapeutic hypothermia may have a protective effect on brain by decreasing the overall cerebral metabolic rate of oxygen consumption. However, the present evidence



Figure 1: (Day 0) computerized tomographic scan image showing multiple fractures including right parietal bone, left temporal bone, left lateral orbital wall, left sphenoid wing, and right frontal contusion with depressed fracture



Figure 2: (Day 2) computerized tomographic scan S/O diffuse hypodensity with loss of differentiation between white and gray matter S/O hypoxic changes and significant cerebral oedema

does not support its use because of increased risk of infection and its interference with clotting system.^[2] However, the prognosis after hypoxic brain injury is usually extremely poor. Only one fourth of patients survive to hospital discharge, and often with severe neurological or cognitive deficits.^[3]

In our case, the child had suffered severe traumatic head injury with a GCS of 4/15 at the time of presentation in emergency department. As it was evident from the blood gas analysis of day 2, there was severe hypoxia, hypercarbia and severe hypotension, factors known to hamper cerebral perfusion. The absent or sluggish pupillary response and motor response to pain in the child were associated with a poor prognosis.^[4] There are no completely reliable clinical signs or investigations which can determine the prognosis of hypoxic-ischaemic brain injury conclusively and therefore repeated clinical examinations and appropriate investigations are necessary to determine the outcome. Various clinical prognostic criteria have been widely used, most of them including brainstem reflexes (absence of pupillary, light, corneal, and oculocephalic reflexes), spontaneous eye movements, and motor responses.^[5] However, the limitations in the sensitivity and specificity of these tests, which are made even more difficult by the use of sedatives, ventilation, hypothermia, neuromuscular blockade, and haemodynamic management make the prediction of outcome a difficult task. Various modalities that can add to clinical testing include imaging studies, short latency somatosensory evoked potentials (SSEP), biomarkers, etc.,. Imaging studies used include CT and magnetic resonance imaging (MRI). In this child, a repeat CT scan of the patient on day 2 was suggestive of diffuse hypoxic changes and cerebral oedema. Whereas a scan

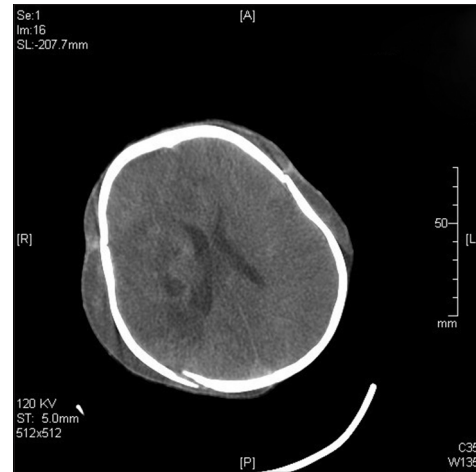


Figure 3: (Day 13) computerized tomographic scan image showing grossly normal study

on day 21 was suggestive of significant improvement. CT scan is known to have limited sensitivity to diagnose the extent of brain damage after a diffuse hypoxic insult. Loss of the normal differentiation between cortical gray matter and subcortical white matter interface and effacement of the basal cisterns, ventricles and sulci are the best-known signs of global hypoxia on CT scan.^[6] MRI is undertaken less commonly because these patients often require sedation and ventilation and therefore, transfer to and from radiology department is often difficult, but it may be particularly helpful in revealing the extent of damage. Diffusion weighted imaging allows good visualization of laminar necrosis and other characteristic signs of hypoxic injury, and it offers reliable information of prognostic importance.^[7] SSEP is another noninvasive bedside test that can be used in predicting the outcome,^[8] but its recording is difficult in ICU environment. Some biomarkers for e.g. S100 beta as a predictor of poor outcome is also less proven.^[9]

Our case report demonstrates how conventional clinical and radiological evaluation of neurological recovery in a patient of hypoxic brain injury can be misleading. Great caution needs to be exercised while assessing clinical and radiological data in these patients, especially when sedative and anaesthetic agents are concurrently being used. Any decision must be taken while keeping the unexpected neurological recovery in mind.

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Conflicts of interest

There are no conflicts of interest.

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