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# MRI IMAGING FEATURES IN GLOBAL HYPOXIC ISCHEMIA

**SUBHASH SINGLA MD AND ANKUR WADHERA MBBS**

*Department of Radiodiagnosis, Christian Medical College and Hospital, Ludhiana - 141008, India.*

## ABSTRACT

Hypoxic changes in brain can vary from mild focal ischemia/ infarction to severe global changes causing permanent neurological disability or death. It is important to recognise and describe the extent of these changes so that the treating physician and the relatives can take the best possible curative/palliative treatment decision for the patient. We present one such case showing global hypoxic ischemic changes in the brain in a patient following a cardiac arrest.

**KEY WORDS:** **Global Hypoxia, Cerebral Ischemia, Brain, MRI.**

**SUBHASH SINGLA MD**
**Department of Radiodiagnosis, Christian Medical College and Hospital, Ludhiana - 141008, India.**

***Corresponding author***

# CASE STUDY

A 52 year old male presented with complaints of chest pain 5-6 hours prior to admission. He was a known hypertensive for the past 20 years on medication and a chronic smoker since past 25 years. On presenting to casualty, an echocardiogram (ECG) was done which showed ST elevation in V1-V6 and aVL leads and anterolateral wall myocardial infarction was diagnosed. Soon after, the patient had a cardiac arrest, and asystole was noted on the monitor. After about 15-20 minutes of cardio-pulmonary resuscitation (CPR), the patient was revived. However, his blood pressure, post resuscitation was 60mmHg (systolic). He was maintained on inotropes - Dopamine and Noradrenaline. He also had altered sensorium after the cardiac arrest and was intubated due to low Glascow Coma Scale (GCS). Next day, he was brought for MRI brain to evaluate the cause for his persistent drowsiness. His condition did not improve and he expired 4 days later.

***MR BRAIN FINDINGS***

Figure 1 (Axial T2FLAIR image) shows abnormal hyperintensity involving the basal ganglia, centrum semiovale and grey matter in bilateral cerebral hemispheres. Gyral edema with sulcal effacement of the bilateral cerebral hemispheres is also seen.

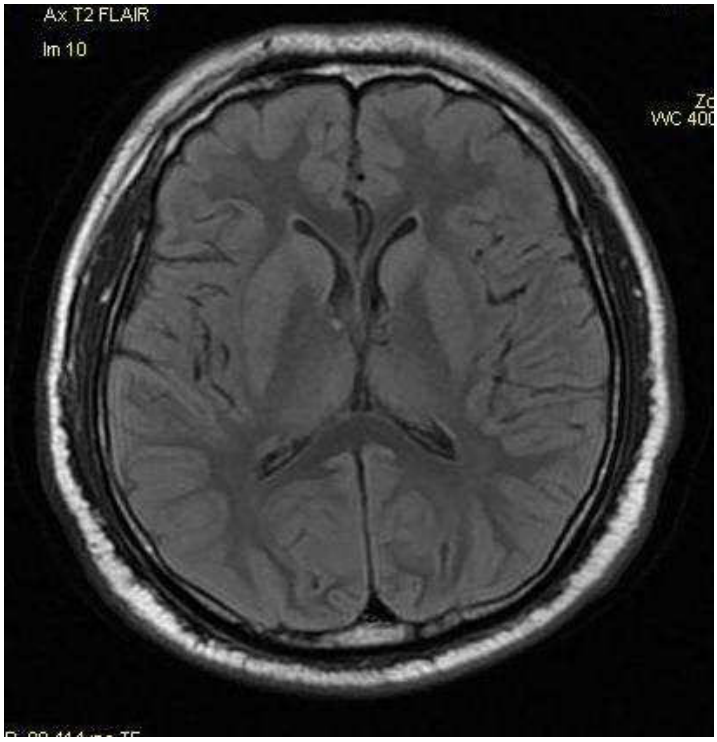


**Figure 1**

***Axial T2 Flair image shows subtle symmetrical hyper-intensity of the cortical grey matter and of the basal ganglia bilaterally along with features of mild cerebral edema***

Fig 2 (DWI) shows diffuse areas of restricted diffusion involving bilateral basal ganglia, centrum semi-ovale and the entire grey matter. ADC mapping shows hypodense areas in the bilateral cerebral hemispheres. IMPRESSION: GLOBAL HYPOXIC INSULT

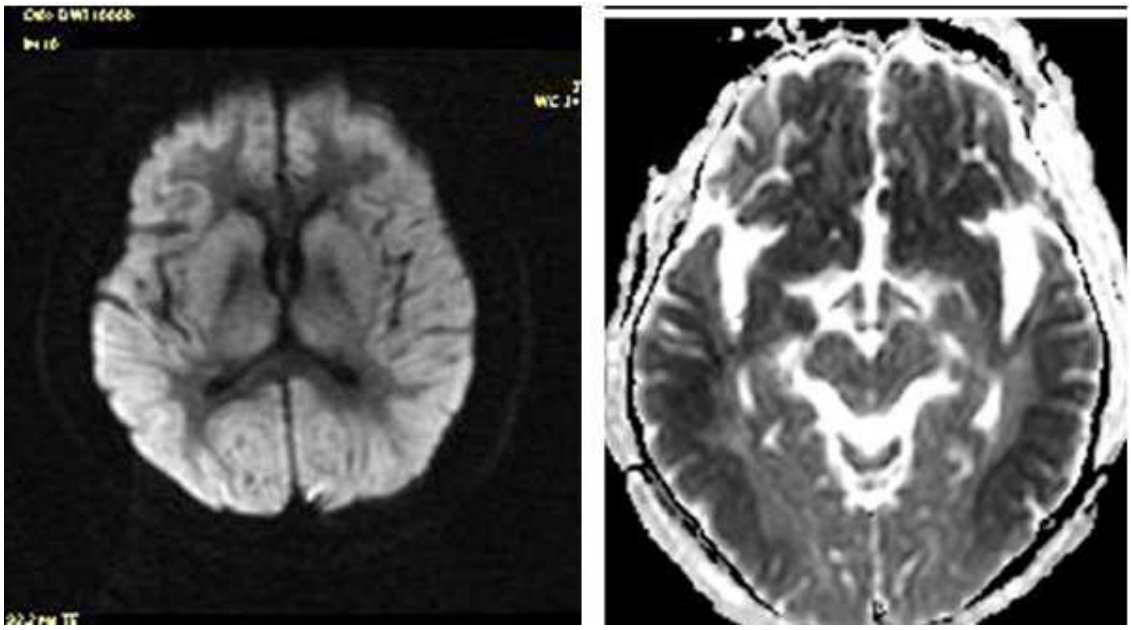

**Figure 2**
***Diffusion weighted image at the same level as figure 1 shows diffuse hyperintense signal involving the bilateral caudate and lentiform nuclei and the grey matter, s/o restricted diffusion. ADC mapping shows hypodense areas in the bilateral cerebral hemispheres***.

## DISCUSSION

***The brain has high metabolic requirements and requires adequate supply of blood mainly for oxygen and glucose. Etiologies of Hypoxic Ischemic Injuries include***

1. Cardiac arrest
2. Cerebrovascular disease
3. Drowning
4. Asphyxiation

The common underlying process regardless of the cause are 1) Decreased cerebral blood flow and decreased blood oxygenation. 2) Switch from oxidative phosphorylation to anaerobic metabolism and 3) Glutamate related cytotoxic processes. Shorter resuscitation times with normal findings on DWI are associated with good prognosis[1].

***Imaging findings***

Injury patterns are highly variable depending upon the brain maturity, severity and the length of the insult[2]. There are mild to moderate-watershed zone infarcts and involving the grey matter structures (basal ganglia, thalami, cortex, cerebellum, hippocampi)[3]. Cerebellar injury tends to be more common in older patients, since Purkinje cells are more sensitive to ischemia.

***CT Findings***

These include a) diffuse cerebral edema with effacement of CSF containing spaces, b) decreased cortical grey matter attenuation with loss of normal grey white differentiation, c) bilateral basal ganglia attenuation and d) “Reversal” or “White cerebellum” sign which suggests severe injury and poor prognosis.

***MR findings***

These include extensive changes in the cortex and the deep grey matter present on diffusion-weighted imaging (DWI) and T2-weighted imaging within 6 days of the insult.

***T1W images***

Normal to very subtle abnormalities. Basal ganglia may show T1 hyperintensity and grey matter signal intensities may persist upto 2 weeks. Chronic stages will show cortical pseudo laminar necrosis.

***T2W images***

Normal to very subtle abnormalities in first 24 hours. There will be basal ganglia hyperintensity and chronic stages will show

residual basal ganglia hyperintensity. T2 FLAIR images will show symmetric hyperintensity in deep grey nuclei +/- cortex.

***DWI***

First imaging modality to become positive within hours after the ischemic event. There is increased signal in cerebellar hemispheres, basal ganglia and cerebral cortex. During the early sub-acute period, grey matter abnormalities are seen on diffusion-weighted images. During the late sub-acute period, diffusion-weighted images show mostly white matter abnormalities. During the chronic stage, the results of diffusion-weighted imaging are usually normal[4].

***ADC***

is reduced in hyper-acute phase due to influx of water from extra to intracellular space. Abnormal ADC values pseudo-normalize during the second week, whereas fractional isotropy values continue to decrease.

***DTI***

FA may be abnormal. Low FA may reflect breakdown in the white matter organisation[6]. Moderate basal ganglia/thalamic injury may result in atrophy but not overt infarct due to delayed apoptosis (may account for normal early ADC values)

***PWI***

In rat models, luxury perfusion immediately follows resuscitation from hypoxic ischemic injury-several hours of cortical and striatal, mild hypoperfusion followed by hyperemia. Quantitative DWI in comatose post cardiac arrest survivors holds promise as a prognostic adjunct [7].

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