

Neurological damage in heat stroke in a child: CT, MRI and SPECT appearances

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Citation

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Abstract

An 18-month old child suffered heat stroke after being locked in a car on a hot sunny day. He developed severe neurological dysfunction with status epilepticus, regression of milestones, cortical blindness and behavioral changes. We present the imaging findings of brain CT scan during the acute stage, and follow-up MRI and SPECT studies, all of which showed changes consistent with severe hypoxic brain damage.

CASE REPORT

An 18 months old child suffered a heat stroke after being accidentally locked in a parked car for about two hours. At presentation, the child was pale, cyanosed, dehydrated, tachycardiac and tachypnoeic with a core temperature of 39.2°C. He was drowsy, the pupils were dilated, but responding to light stimulus. There was no papilledema. Blood coagulation profile was normal. Liver function tests and renal function tests were borderline abnormal.

Over the next day, the child developed episodes of tonic-clonic seizures, involving the right side of the body, which were managed with IV diazepam and Midazolam. The seizures were not controlled with this regimen, increased in frequency and duration over the next 2 days, and progressed to vacant stares, refractory generalized seizures and deep coma. The seizures could finally be controlled with IV phenytoin, midazolam, Phenobarbital and mannitol. A CT scan of the brain was performed at this stage (4th day) (Fig 1).

Figure 1

Figure 1: Axial CT scan on the 4 day after heat stroke. Generalised cerebral edema with loss of gray-white matter differentiation, including indistinct basal ganglia margins. The relatively higher density of the cerebellum is apparent. Lateral ventricles and supratentorial cisterns were compressed. No parenchymal hemorrhages could be identified.

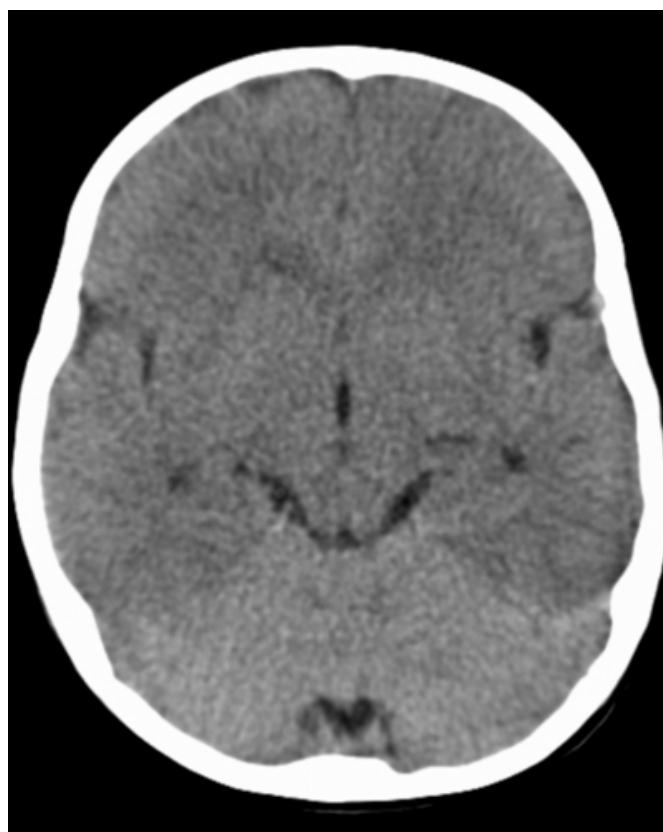


Figure 2

Figure 2: MRI Brain on Day 45. (a) Axial FLAIR image shows diffuse increase in the signal intensity of the subcortical and deep white matter with loss of gray-white differentiation. Partial sparing of the splenium of the corpus callosum and the medial occipital cortices (posterior circulation) is shown. Patchy high signal intensity of the parieto-occipital cortex at the middle cerebral \hat{A} - posterior cerebral arteries watershed zones (arrows).

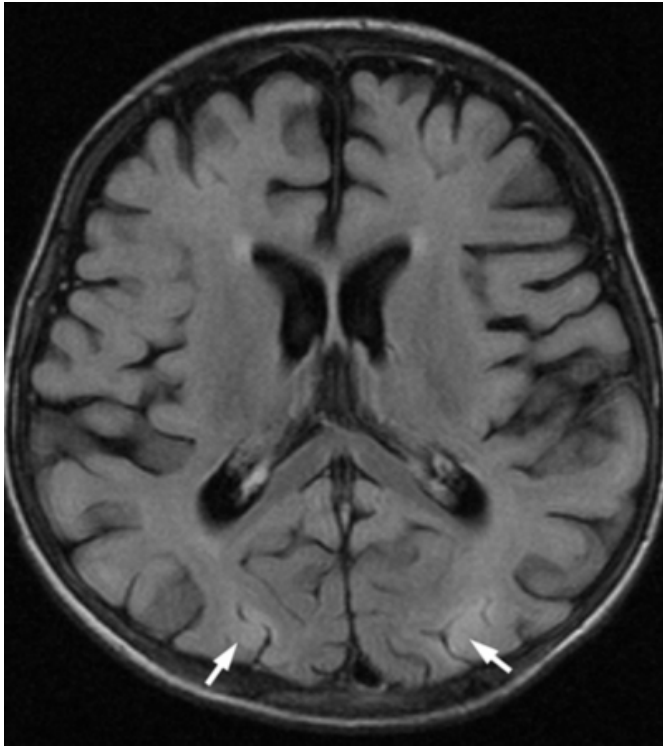
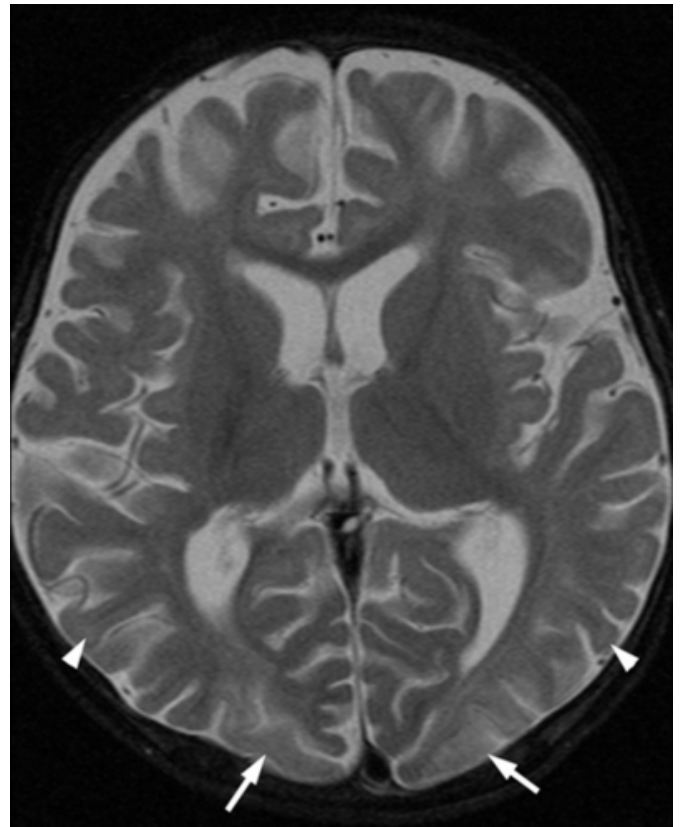


Figure 3

Figure 2: MRI Brain on Day 45. (b) Axial spin-echo T2 weighted image shows subcortical high signal intensity lines in both parietal lobes (arrowheads). Patchy high signal intensity is shown in the middle cerebral \hat{A} - posterior cerebral arteries watershed zones (arrows). There is a diffuse increase in the signal intensity of the basal ganglia structures. Note the relatively normal signal intensity of the medial occipital cortices. There is generalized atrophy of the cerebrum with enlargement of the CSF spaces.



On the ninth day of illness, the child was awake, but not responding to name or oral commands. He had lost head control, was unable to sit or stand, and was hypotonic (power in limbs 3/5), though there were active movements of the limbs. All deep tendon reflexes were brisk, with bilateral upgoing plantars. There were choreiform movements of all limbs. He had developed cortical blindness - there was no fixation, visual contact or following of light or objects. Pupils were 7mm diameter, and reacted to light. Visual evoked response study (LED goggles) demonstrated bilateral response at 125 and 123ms, right and left, respectively. Brainstem auditory evoked response revealed poorly formed waveforms with prolongation of wave I and subsequent waves.

Follow-up MRI (Fig 2) and SPECT (Fig 3) studies of the brain were performed 6 weeks after the onset of the illness to

evaluate the extent of neurological damage. At 9 months' follow-up, the child is on maintenance doses of Phenobarbital and sodium phenytoin. He is able to hold his head and sit with support, but he is unable to stand up or walk. He does not respond to commands and has persistent cortical blindness.

Figure 4

Figure 3: Brain SPECT (Tc-HMAPO) on Day 45. (a) Paramedian sagittal section shows marked hypoperfusion of the anterior circulation territory with patchy areas of fall-out, most prominent at the MCA-PCA watershed zone (arrow). Relatively preserved perfusion of the occipital lobes, cerebellum and thalamus.

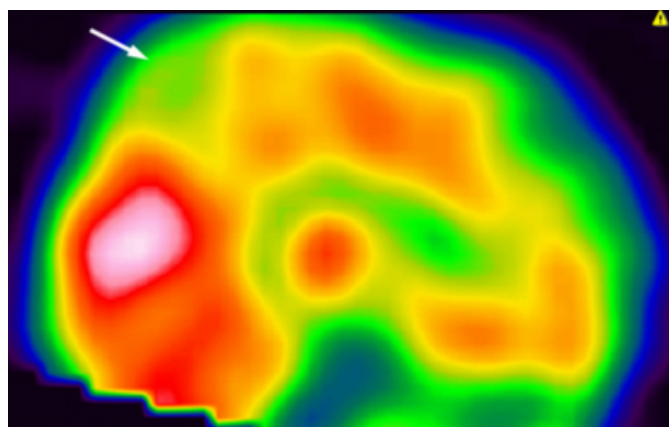
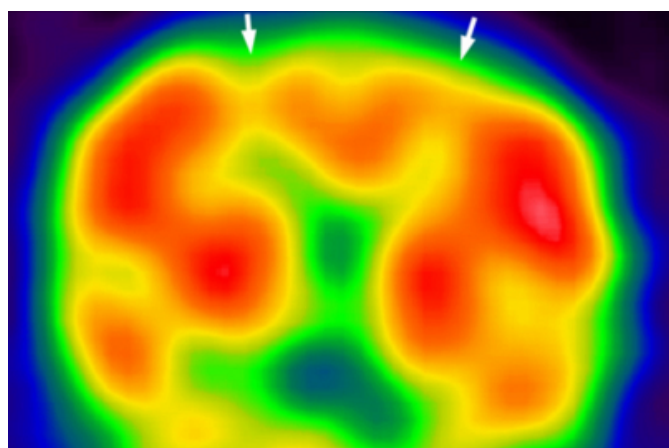


Figure 5

Figure 3: Brain SPECT (Tc-HMAPO) on Day 45. (b) Coronal section through the posterior parietal lobes shows marked hypoperfusion of the anterior cerebral artery territory and the temporal lobes. Lower perfusion is shown at the ACA-MCA watershed zone (arrows).



DISCUSSION

Heat stroke is a relatively common medical emergency [1,2,3]. It is associated with multi-organ dysfunction, is often fatal (10% to 21%), and, frequently results in neurological

disability [2, 4, 5] and delayed mortality [2]. Although, direct cellular damage from increased temperature presumably constitutes the initiating event in heatstroke [6], experimental observations have shown that endotoxemia, increased circulating levels of specific cytokines [6], failure of thermoregulation and hypovolaemic shock, all play a part in the clinical manifestations of heat stroke [7]. In animal models, hyperthermia, cerebral congestion and brain edema occur concurrently, resulting in elevated intracranial pressure – this together with a fall in the mean arterial pressure secondary to the peripheral vasodilatation causes a fall in the cerebral blood flow and cerebral hypoxia [8]. The result is widespread cell damage, with major involvement of the central nervous system, liver, kidneys and muscles.

Neurological involvement is the most catastrophic sequel, with patients presenting to the hospital in deep coma, flaccid paralysis, hyper-reflexia, seizures, and a high incidence of permanent neurological deficit [2,5,9].

The described imaging findings in heat stroke include early cerebral edema [9], loss of gray-white matter differentiation [1], patchy high signal intensity of the white matter of cerebral hemispheres and corpus striatum [10], central pontine myelinolysis [9], vascular boundary zone infarcts [11] and in later stages, diffuse cerebellar atrophy [10,12]. One recent report has documented T1 shortening, T2 hyperintensity and contrast enhancement of the external capsule and medial thalami, in addition to enhancement of cerebellar lesions [13].

Additional imaging findings (Fig 2), not described before in patients of heat-stroke, that were seen in our patient include extensive diffuse white matter hyperintensity with relative sparing of the splenium of the corpus callosum and posterior limbs of the internal capsules, with extensive sub-cortical laminar necrosis most prominent in the temporal and posterior parietal lobes. Patchy areas of increased cortical signal intensity were seen at the watershed zones in the paracentral posterior parietal – occipital lobes. There was moderate cerebral atrophy at six weeks. These findings correlate with the persistent motor deficit, psychological impairment and cortical blindness seen in our patient.

Most previous reports have highlighted a dominant involvement of the cerebellum and brainstem [4,14,15] and cerebellar atrophy [10,14]. In our patient there was relative sparing of the posterior circulation with predominant involvement of the anterior circulation, with clinical and imaging features of bilateral posterior border zone infarcts

[₁₆], namely, minimal responsiveness, cortical blindness and limb weakness, with cortical and underlying white matter changes in bilateral posterior border zones on MRI and SPECT studies (Figs 2 and 3).

Near drowning is a pathophysiological event similar to heat stroke with imaging findings of extensive cerebral ischemia - early cerebral edema, loss of gray-white matter differentiation, basal ganglia hyperintensity, patchy cortical hyperintensities and subcortical laminar necrosis. The findings of early brain edema, basal ganglia hyperintensity and cortical abnormalities correlate with poor neurological recovery [₁₇]. This is confirmed by the poor neurological recovery in our patient with residual cortical blindness, motor deficits and behavioral changes.

CONCLUSIONS

To conclude, heat stroke affects multiple organ systems, but the most debilitating consequences are due to irreversible damage to the central nervous system. The imaging findings of subcortical laminar necrosis, cortical and white matter signal intensity changes at the posterior border zones, multiple perfusion defects at SPECT, and limitation of the major abnormalities to a vascular territory (the anterior circulation) confirm that severe brain ischemia was the underlying cause for the neurological involvement in heat stroke in our patient.

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