Short report

Spinal cord lesions in heat stroke

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SUMMARY A clinical and pathological report of a patient who died 15 days after suffering a classic heat stroke is presented. The clinical picture was of a flaccid quadriplegia with anhidrosis and sphincter disturbance. The most severe pathological lesions were to be seen in the anterior and intermedio-lateral horns of the spinal cord. Hyperthermia was the only physiopathological mechanism demonstrated in the patient. It is suggested that the motor neurons and vegetative neurons of the spinal cord are specially sensitive to hyperthermia.

There is a considerable literature, as listed in recent reviews, on the epidemiology, pathogenesis, physiopathology and clinical features of heat stroke. There are few neuropathological studies, in spite of the fact that there are always characteristic signs and symptoms of neurological involvement. The basic neuropathological reference work is still that by Malamud et al in 1946, who studied 125 fatal cases of heat stroke induced by physical exercise and described characteristic lesions in the cerebrum, cerebellum and skeletal muscles. This paper presents a clinical and pathological study of a case of classic heat stroke, with no damage to the cerebrum but with severe neuronal lesions in the spinal cord.

Case report

A 66-year-old man was referred to the Hospital of Navarra; for the previous 3 hours he had remittent right-sided somatomotor partial seizures with secondary generalisation. The patient, who suffered from manic depressive psychosis, was a long term in-patient of a psychiatric hospital which had no air-conditioning. He had been receiving continuous mediation with amitryptiline (30 mg/day), imipramine (75 mg/day) and phenerganazine (6 mg/day). For the previous few days, the region had a heat wave with maximum temperatures of 38°C. The fits did not reappear after the administration of iv clonazepam 2 mg. The patient, who was very obese, had a rectal temperature of 42.5°C and his skin was hot and dry. Blood pressure was 120/70 mm Hg and pulse 120/min with normal sinus rhythm. There were no other abnormalities on physical examination. Neurological examination was normal except for a moderate drowsiness and mental confusion. Routine biological tests, including electrolytes, pH and clotting were normal except for a leucocytosis of 10,000 cells/mm³. CT scan and CSF were normal. The EEG recording was diffusely slowed (5 Hz). The rectal temperature was reduced to 38°C by physical means of cooling over the following 10 hours. Twelve hours after admission, the patient developed an acute respiratory insufficiency owing to weakness of the respiratory muscles and required endotracheal intubation and mechanical ventilation. At the same time he was found to be quadriplegic, with muscular hypotonia, absence of muscle stretch and skin reflexes, retention of urine and faecal incontinence. The cranial nerves, sensation and the level of consciousness were preserved. This neurological situation continued without change for 14 days, after which the patient suddenly died. During all this time, no sweating was seen, continuous physical cooling methods being required to maintain a normal body temperature. During the first 72 hours of the course of this illness, there was a rise in the skeletal isoenzyme of serum creatine kinase to 9-50 U/ml, which gradually became normal over the next few days. At no time was there severe hypoxia or circulatory collapse.

Pathological findings

The abnormal pathological findings were confined to skeletal muscle and the nervous system. The rest of study revealed only a moderately fatty liver. The cerebral hemispheres and the brain stem were macroscopically and histologically normal. In the cerebellum, there was almost total loss of Purkinje cells, without proliferation of Bergmann glia, and necrosis of the granular layer (fig 1, A). The dentate nucleus also showed neuronal loss without gliosis. With the myelin stains, a diffuse pallor of the cerebellar white matter could be seen, which was most marked in the hilus of the dentate nucleus (fig 1, B). Throughout the spinal cord there were marked neuronal abnormalities in the anterior and intermedio-lateral horns, with chromatolysis.

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and neuronophagia (fig 1, C). In the anterior roots numerous axons were seen to be interrupted, with abundant macrophages phagocytosing myelin. There were no lesions in the posterior horns and roots or in the spinal cord tracts. The sympathetic ganglia and the sural nerve were normal. Skeletal muscle showed severe rhabdomyolysis with regeneration (fig 1, D). Using histochemical techniques, the presence of angular fibres with inversion of the oxidative enzymatic pattern and numerous target fibres could be seen.

**Discussion**

There were many risk factors for heat stroke in this case: obesity, advanced age, institutionalisation, lack of air-conditioning and a heat wave at the beginning of the summer. The latter meteorological factor is associated with high morbidity and mortality due to heat stroke. One cannot exclude the possibility that the psychotropic drugs he was taking might have been contributors, because of their anticholinergic, alpha-adrenergic blocking and hypothalamic-suppressing actions.

Heat stroke runs its course with serious electrolyte disturbances, circulatory collapse and disseminated intravascular coagulation. This combination of multiple physiopathological factors makes it unlikely that in most cases hyperthermia per se is responsible for production of nervous system lesions. This present case is a rare instance of heat stroke with no other demonstrable physiopathological mechanism than hyperthermia. Rhabdomyolysis, unusual in classical heat stroke, but a constant finding in that induced by exercise, might in this case have been caused by convulsive seizures. Apart from this, the only pathological lesions were found in the nervous system, in the cerebellum and spinal cord. Generally it is assumed, in accord with Malamud et al that the neuronal lesions in the cere-
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The neuropathological findings in this unusual case suggest that the spinal cord, in addition to the cerebellum, is a region of the nervous system particularly sensitive to hyperthermia.

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References

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