MRI demonstration of the cerebellar damage in diffuse hypoxic-ischemic encephalopathy. Case report

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MRI demonstrated symmetric putaminal and cerebellar changes in a patient who survived a cardiorespiratory arrest due to acute idiopathic polyneuritis. Since similar aspects can be observed following cyanide or methanol poisoning differential diagnosis from these conditions has to rely on the combination of clinical and imaging findings.

Key Words: MRI — cerebellum — anoxic — ischemic encephalopathy

Introduction

Although it is known from pathological studies that the cerebellar cortex is with the cerebral cortex and basal ganglia one of the elective sites of damage in hypoxic-ischemic encephalopathy [1], computed tomography (CT) studies, presumably due to the limitations of the technique in investigating the posterior fossa, have focused mainly on post-anoxic supratentorial changes [4, 7]. We report the magnetic resonance imaging (MRI) pattern of the symmetric damage to the putamen and cerebellum that we observed in a patient who survived a cardiorespiratory arrest.

Case report

A 75 year old woman without relevant medical antecedents except a treated arterial hypertension, after several days of generalized weakness and malaise with increasing dyspnea had a respiratory arrest and lost consciousness while in her home. Within 4 minutes the patient was resuscitated and put on assisted ventilation. At admission to the emergency unit of a peripheral hospital the patient was stuporous. Blood pressure was 130/70 mmHg; ECG and chest X ray films were normal. Laboratory blood tests were unremarkable except for an arterial PO2 of 60 mmHg. Neurological examination revealed diffuse weakness, hypotonia, and areflexia. Lumbar puncture and CSF laboratory examination showed findings consistent with acute idiopathic polynueuritis (Guillain Barré syndrome) including slight hyperproteinemia (0.52 mg/dl), increased CSF albumin/serum albumin index (0.0118), and normal CSF cell count (1.8 cells ml). Thirty days after onset the patient appeared moderately confused with left arm weakness and persistent diffuse areflexia. Two weeks later ventilatory assistance began to be discontinued for increasingly longer periods of time. Four months after onset the patient was in spontaneous respiration, could sit in an armchair and talk with her relatives. Neurological examination revealed an oriented and cooperative patient, with elicitable deep tendon reflexes and persistent left arm hyperreflexic paresis. In addition, bursts of choreic movements of the right arm and leg which lasted approximately 3-4 minutes were noted. These in-
voluntary movements subsided during sleep and were controlled by intravenous diazepam. Plain CT showed widening of the basal cisterns and of the convexity subarachnoid spaces and bilateral hypodense areas in the basal ganglia. Cranial MRI examination 6 months after onset (Fig. 1) showed a bilateral spindle-shaped hyperintensity on spin-density and T2-weighted Spin-Echo images in the putamen associated with mild enlargement of the frontal horns of the lateral ventricles and symmetric hyperintensity of the cortex and white matter of both cerebellar hemispheres. Coronal T1-weighted Spin-Echo images centering on the signal changes in the posterior fossa showed no definite signal abnormality of the cerebellum (Fig. 2).

Discussion

After mechanisms of autoregulation of cerebral circulation have been overwhelmed by hypoxic-ischemic insults from various causes, such as suffocation, carbon monoxide poisoning, diseases which paralyze the respiratory apparatus as Guillian Barré syndrome or poliomylitis, or cardiovascular collapse, brain damage occurs, usually with a symmetric distribution, at sites where the metabolic rates are highest. These include border zones between major cerebral arteries, hippocampus, basal ganglia and cerebellum [1]. In a pathological study of 16 patients who died following anoxic-ischemic insults Sevestre et al. [6] found involvement of the basal ganglia in 14 cases and of the cerebellar cortex in 11 cases. They identified an elective involvement of the Purkinje cells and molecular layers with variable compensatory laminar proliferation of astrocytes [6].