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PRACTICAL PEARL

Crossed Cerebellar Diaschisis in Status Epilepticus

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Abstract
Background Crossed cerebellar diaschisis (CCD) has been reported on positron-emission tomography and single-photon emission computed tomography of stroke patients. Rarely it has been described with brain diffusion-weighted MRI (DWI) of status epilepticus (SE).
Methods Case report.
Results A 53-year-old woman was found unresponsive after cocaine use. A diagnostic electroencephalogram was consistent with ictal SE. A brain DWI showed reduced diffusion in the left temporo-parietal and occipital cortexes, the left thalamus and the right cerebellum. The DWI changes did not correspond to a vascular territory and were attributed to seizure activity and secondary CCD. A 2-week follow-up DWI showed interval near-complete resolution of the diffusion changes. CCD in SE may represent injury caused by excessive neuronal transmission from prolonged excitatory synaptic activity via the cortico-pontine-cerebellar pathway. Alternatively, it may be a result of interruption of the cortico-pontine-cerebellar pathway with loss of cortical inhibitory input.
Conclusion This case documents CCD during SE, providing further evidence of contralateral cerebellar involvement with a supratentorial epileptiform focus.

Keywords Crossed cerebellar diaschisis · Cerebellum · DWI · Status epilepticus

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Case Report

A 53-year-old woman was found unresponsive and incontinent of stool and urine after cocaine use. Upon arrival to the hospital her Glasgow coma scale score was 10 (E4 M5 V1). She was drowsy, had a left gaze preference and was able to localize to pain on the left, but her right arm and leg were plegic. A computed tomography scan of the brain was unremarkable. An electroencephalogram (EEG) showed periodic bursts of polymorphic delta and sharply contoured theta activity in the left hemisphere, and low amplitude polymorphic theta, admixed with alpha and delta frequencies in the right hemisphere, consistent with ictal status epilepticus (SE) (Fig. 1).

Brain diffusion-weighted MRI (DWI) showed widespread areas of reduced diffusion in the left temporo-parietal and occipital cortexes, the left thalamus, and the right cerebellum (Fig. 2, panel a). The DWI changes did not correspond to a vascular territory and were attributed to continuous seizure activity with crossed cerebellar diaschisis (CCD). After treatment with anticonvulsive agents, the EEG improved but continued to show asymmetry with slower frequencies over the left hemisphere. Two weeks later she was alert and awake, but a right hemiparesis and moderate aphasia persisted. A follow-up DWI at that time showed interval near-complete resolution of the diffusion changes (Fig. 2, panel b).

Comment

CCD has been primarily recognized in the stroke literature [1] and has been attributed to interruption of afferent cortico-ponto-cerebellar pathways resulting in decreased blood flow and metabolism in the cerebellar hemisphere

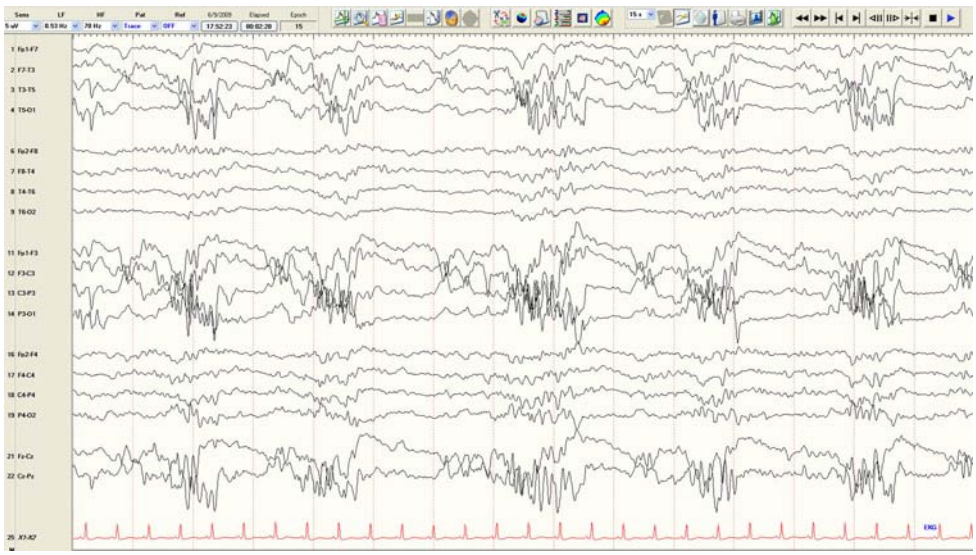

Fig. 1 EEG with high-amplitude burst discharges in the left hemisphere and low-amplitude activity in the right hemisphere. Pattern suggestive of ictal activity with a left hemispheric focus

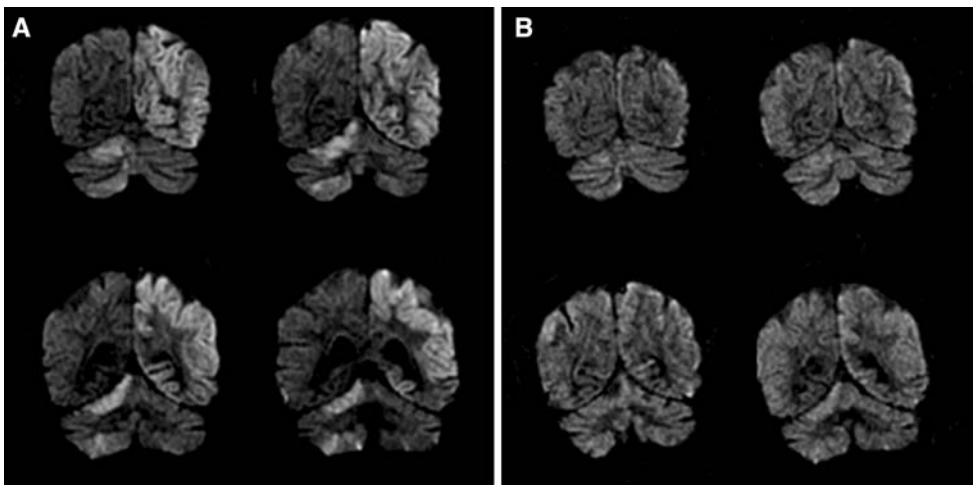

Fig. 2 Coronal diffusion-weighted MRI demonstrating extensive areas with reduced diffusion in the left temporo-parietal cortex and the right cerebellum (**a**). Interval near-complete resolution of seizure-related diffusion changes after 2 weeks (**b**)

contralateral to a supratentorial lesion [2]. The blood flow reduction seen in vascular diaschisis is thought to result from underlying decreased neuronal activity [3].

Increased excitatory synaptic activity, as seen during seizures, increases energy metabolism and cerebral blood flow (CBF) [4]. Prolonged seizure activity can cause hyperintense DWI signal [5, 6], probably due to uncoupling between metabolism and CBF resulting in tissue hypoxia, anaerobic glycolysis, sodium/potassium pump failure, and cytotoxic edema [7, 8].

CCD in SE may represent injury caused by excessive neuronal transmission from prolonged excitatory synaptic activity via the cortico-pontine-cerebellar pathways. Although structural damage of cortical areas and projecting fibers may account for the chronic effects of CCD, the acute process appears to be functional and may involve increased excitatory input from the cerebral cortex. A cortical focus of epileptiform discharges with transhemispheric diaschisis would suggest a functional mechanism for diaschisis during SE. Alternatively, as clinical neurophysiological studies as well as experimental work suggest [9, 10], increased neuronal excitability in remote areas affected by transcortical diaschisis may be a result of desinhibition of cortical neurons [3]. Both mechanisms may lead to induced cytotoxic injury evidenced by imaging studies.

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