

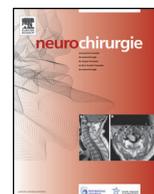


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## Letter to the editor

## Crossed cerebellar diaschisis in status epilepticus



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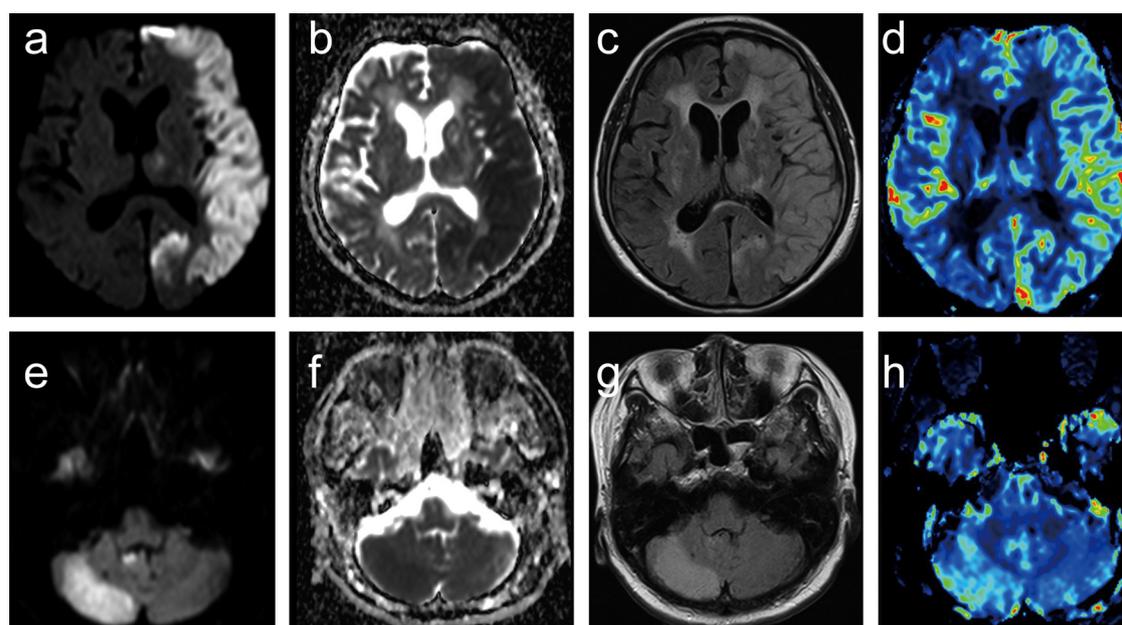
Crossed cerebellar diaschisis  
Status epilepticus  
Magnetic resonance imaging  
Cerebral blood flow

Dear editor,

A 76-year-old woman came to us with consciousness disturbance. She had a history of subcortical hemorrhage in the left parietal lobe, after which right hemiparesis remained and symptomatic epilepsy developed. The patient was being treated with the anticonvulsant valproate sodium. At admission, the Glasgow Coma Scale score was E3V1M5 and right hemiparesis with twitching on the right side of the face was noted. Intravenous administration of diazepam, glycerol, and the free-radical scavenger edaravone was initiated. Diffusion-weighted and fluid-attenuated inversion recovery MRI revealed extensive cortical signal changes, with decreased apparent diffusion coefficient values in the left cerebral hemisphere

and right cerebellum (Fig. 1a, b, c, e, f, g). Subsequent perfusion MRI showed increased blood flow in the corresponding lesions (Fig. 1d, h). In addition, video-electroencephalographic monitoring revealed spike waves in the left occipital lobe. We made a diagnosis of status epilepticus with crossed cerebellar diaschisis (CCD). Follow-up MRI 19 days later showed a reduction in signal changes along with improvement in laterality of blood flow in the corresponding lesions. Consciousness level showed gradual improvement, but without completely recovering previous status.

CCD has been described as contralateral cerebellar hypometabolism associated with reduced cerebellar blood flow in patients with stroke, and has notably been observed during the subacute-to-chronic stage following dense middle cerebral artery infarction. The proposed mechanism is transsynaptic neuronal degeneration secondary to inhibition of the cortico-pontine-cerebellar pathway. Furthermore, seizure-related hemispheric cortical signal abnormalities have been well documented in MRI studies [1]. However, cases described as CCD with contralateral cerebellar involvement have rarely been reported [2–6]. Status epilepticus causes an increase in cerebral blood flow (CBF), to deliver more oxygen and glucose in accordance with increased metabolic demand around the supratentorial epileptic focus [7]. In a later stage, decreased CBF leads to a mismatch in metabolic demand, resulting in irreversible neuronal damage. In acute CCD,



**Fig. 1.** MRI revealed extensive areas of cortical hyperintensity in the left hemisphere and contralateral cerebellum in diffusion-weighted (a, e) and fluid-attenuated inversion recovery (c, g) images. The corresponding lesions showed decreased values on apparent diffusion coefficient mapping (b, f) and increased blood flow on perfusion MRI (d, h).

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a cortico-pontine-cerebellar tract is considered to be involved in transmission of seizure activity to the contralateral cerebellum [5].

#### Disclosure of interest

The authors declare that they have no competing interest.

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