Left hemispheric status epilepticus with crossed cerebellar diaschisis

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Introduction*:* crossed cerebellar diaschisis (CCD) refers to the depression of blood flow and metabolism affecting the cerebellar hemisphere due to a contralateral cerebral injury, as a result of disruption of the cortico-ponto-cerebellar pathway. It has been described mostly in hemispheric and thalamic stroke, supratentorial tumours and infections and less often in epilepsy, in particular, status epilepticus (SE).

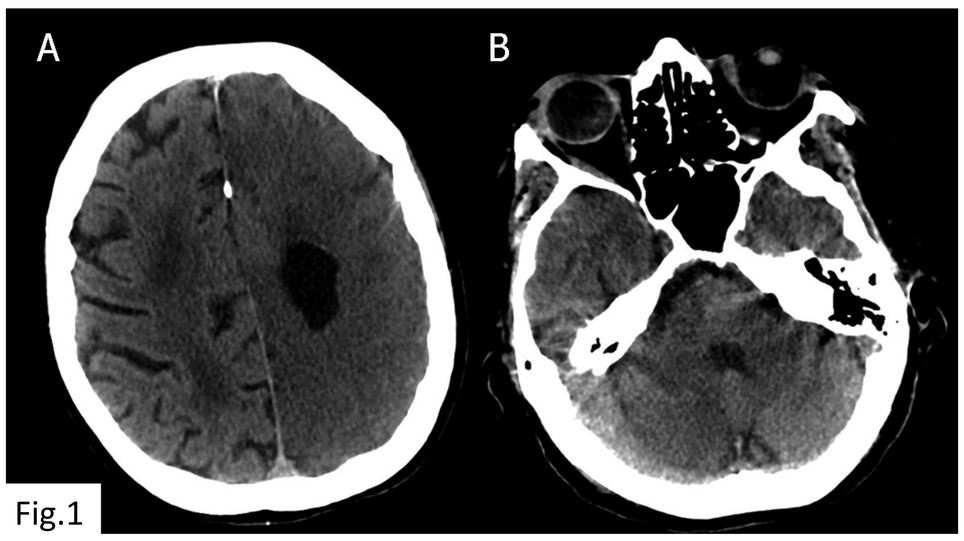

Fig.1

Case report: a 68-year-old woman referred to the emergency department for impaired consciousness and involuntary right-sided movements. She was affected by epilepsy secondary to cerebral palsy, and she presented partial seizures, occasionally followed by secondary generalisation, well controlled by drug treatment. *Neurological examination* showed right spastic hemiparesis as a result of cerebral palsy; clinical signs of cerebellar dysfunction were absent. The patient progressively recovered consciousness with intravenous valproic acid.

Diagnostic studies: *brain CT scan* (Fig.1), showed diffuse hypodensity of the left cerebral hemisphere and of the right cerebellar hemisphere. *Electroencephalography* (Fig.2) showed continuous epileptic discharges in the left hemisphere. *Brain MRI* (Fig.3), revealed gyriform reduced diffusion signal, T2-weighted and diffusion-weighted imaging (DWI) hyperintensity and swelling in the left cerebral hemisphere and in the contralateral right cerebellum; increased vascularisation in the left cerebral hemisphere was evident on MR angiography. *Routine blood tests* were normal except for high level of sodium. *Cerebrospinal fluid* examination was negative.

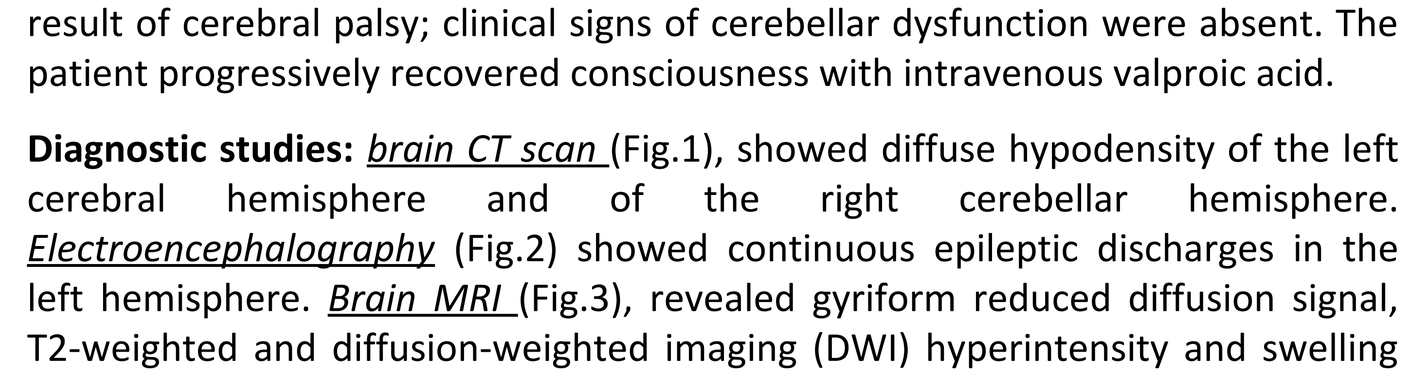

Fig.2

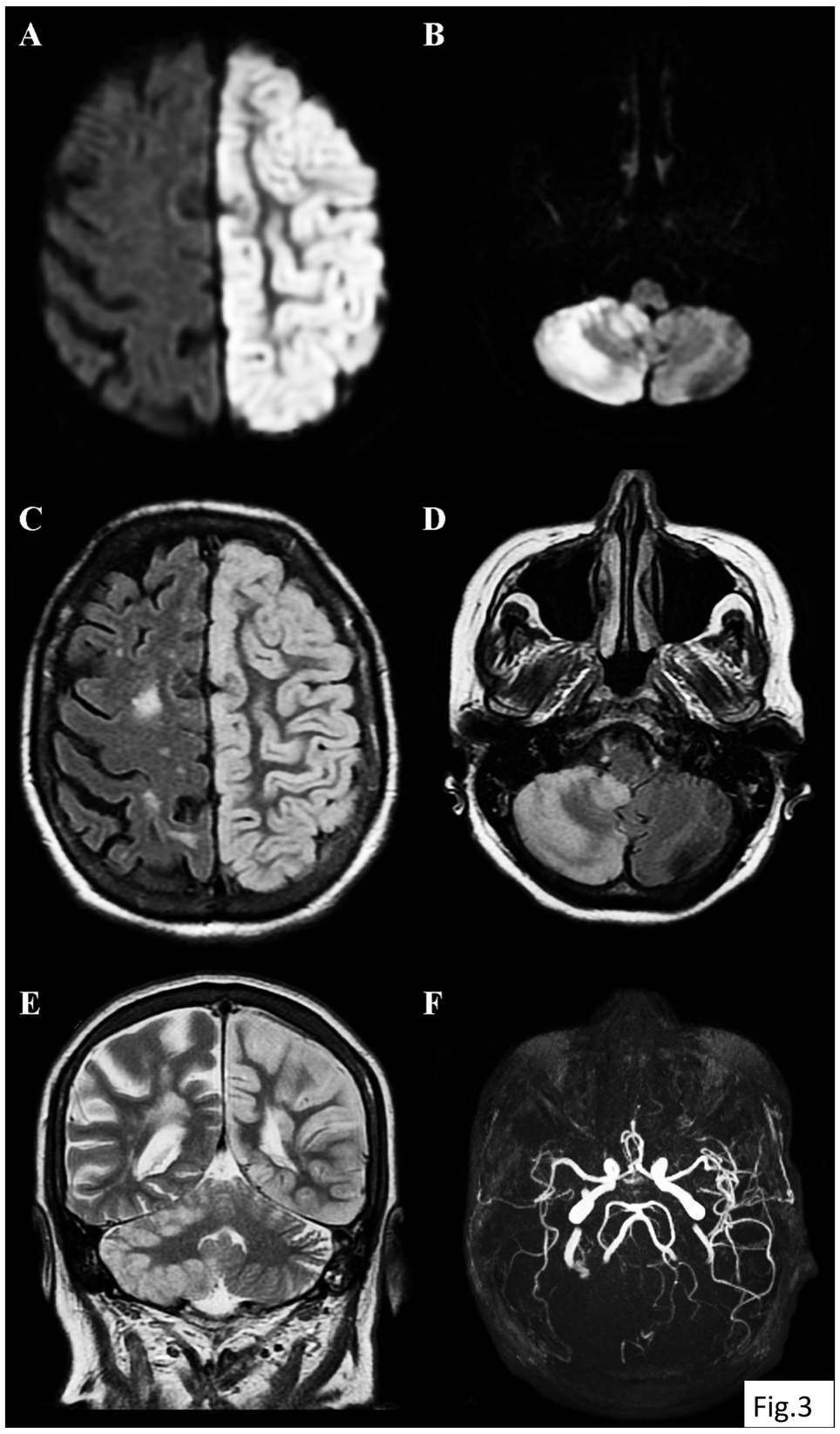

Fig.3

Discussion: these findings were consistent with left hemispheric SE with CCD. Seizures and SE can induce a variety of MRI abnormalities, including increase of signal intensity in T2-weighted imaging and in DWI. These changes support the idea that seizure activity results in vasogenic and/or cytotoxic oedema. In our case, the increased vascularization in the left cerebral hemisphere evident on MR angiography, the localization of the lesions and the resolution of symptoms rule out the vascular origin of the lesions. Moreover, the absence of blood brain barrier disruption and contrast enhancement in T1-weighted MRI make unlikely an infectious or inflammatory origin.

Conclusion: in patients affected by SE, glutamate is discharged continuously, and it induces desensitisation of gamma-aminobutyric acid type A receptor and, consequently, the excitement of neurons continues and cerebral metabolism and oxygen demand increase. In our case, we think that hypernatremia and dehydration could have favoured SE and subsequently CCD as the result of the excessive transmission of excitatory input from the seizing cortex to the contralateral cerebellum, via the cortico-ponto-cerebellar pathways, resulted in relative hypometabolism or hypoperfusion in the cerebellar hemisphere.

Reference: Ferilli MAN, Brunetti V, Costantini EM*, et al.* Left hemispheric status epilepticus with crossed cerebellar diaschisis. *J Neurol Neurosurg Psychiatry* Published Online First: 08 May 2017. doi: 10.1136/jnnp-2017-315930