Neuroimaging Highlight

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Epilepsy and Crossed Cerebellar Diaschisis with Persistent Cerebellar Syndrome

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A 24-year-old man with developmental delay and epilepsy since the age of 11 presented with one day history of secondarily generalized tonic clonic status epilepticus. He recently had focal motor seizures involving his left face and arm five times per day and generalized and complex partial seizures once per month despite two prior surgeries including a right temporal lobe resection. (Figures 1A, B) The pathology from his surgical resection revealed non specific gliosis. He had tried multiple anti epileptic drugs (AEDS) but his epilepsy remained intractable. He remained on divalproex sodium, clobazam and topiramate at the time of his admission. Upon admission to the emergency room (ER), he was intubated for airway protection and treated with intravenous phenytoin.

Computed tomogram head revealed known encephalomalacia in the right temporo-parieto-occipital lobe and a new area of hypodensity in the left cerebellar hemisphere. An initial magnetic resonance imaging (MRI) showed T2 hyperintensities in the right cerebral cortex and left cerebellar hemisphere consistent with crossed cerebellar diaschisis (CCD). (Figure 1C) After extubation, he had new left sided hemiparesis and left upper extremity dysmetria and dysdiadochokinesis. A second MRI two weeks later revealed CCD in similar appearance to the initial scan. He continued to have daily left sided focal motor seizures despite AED therapy. His deficits improved with daily physiotherapy but remained upon his discharge. A third MRI performed two months after the initial presentation demonstrated complete resolution of the increased fluid attenuation inversion recovery (FLAIR) signal in the cerebellum with no perfusion abnormalities. His left sided hemiparesis and cerebellar deficits remained however.

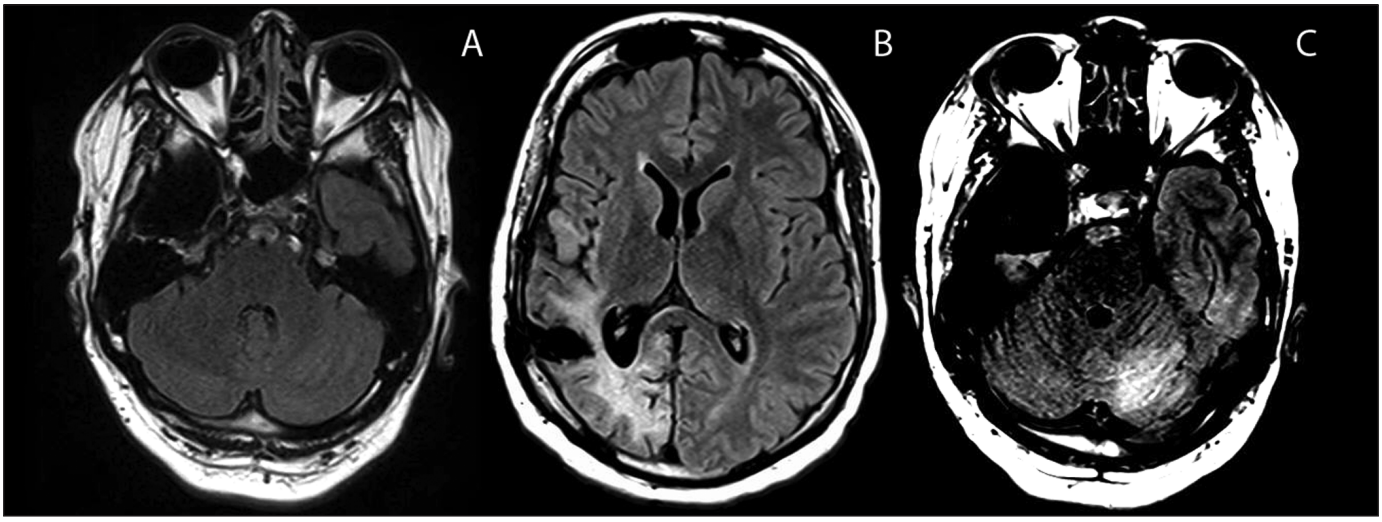

Figure 1: *CCD on MRI FLAIR sequences. A) Evidence of prior right temporal lobectomy is shown. B) Encephalomalacia and chronic right cortical signal change. C) Contralateral left cerebellar increased signal (CCD) which resolved on repeat MRI (not shown).*

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Comment

Diaschisis is a term coined by Von Monakow in the late 19th century to describe the depressive effects that damaged parts of the cerebrum could have on other interconnected areas at various levels.[1] Crossed cerebellar diaschisis (CCD) is a phenomenon of decreased metabolism and hypoperfusion of the cerebellar hemisphere due to damage of the contralateral cerebral hemisphere. This condition has been described in patients with ischemic stroke, status epilepticus and complex regional pain syndrome.[2-4] It is thought to be the result of de-afferentation due to loss of the contralateral cortico-ponto-cerebellar excitatory tracts resulting in cerebellar hypometabolism and decreased perfusion.[4]

Magnetic resonance imaging abnormalities are known to occur in the setting of status epilepticus however CCD has only rarely been documented on MRI FLAIR sequences, usually requiring dynamic sequences such as positron emission tomography, single-photon emission computed tomography and magnetic resonance imaging perfusion studies to assess the observed perfusion changes.[5-7] Typically CCD is an acute and transient phenomenon but patients may be left with potentially permanent neurological sequelae if the area of hypoperfusion is prolonged; persistent cerebellar syndrome following CCD has been previously described.[7] Our patient remained with a cerebellar syndrome despite the resolution of his MRI changes, supporting this hypothesis.

References

1. Finger S, Koehler P, Jagella C. The Monakow concept of diaschisis. Arch Neurol. 2004; 261(2):283-8.
2. Lin DDM, Kleinman JT, Wityk RJ, et al. Crossed cerebellar diaschisis in acute stroke detected by dynamic susceptibility contrast MR perfusion imaging. AJNR Am J Neuroradiol. 2009; 30(4):710-15.
3. Lai MH, Wang TY, Chang CC, et al. Cerebellar diaschisis and contralateral thalamus hyperperfusion in a stroke patient with complex regional pain syndrome. J Clin Neurosci. 2008; 15(10): 1166-8.
4. Lacout A, Marcy P-Y, Pelage J-P. Reverse crossed cerebellar diaschisis following refractory status epilepticus. Postgrad Med J. 2011; 87(1027):382-3. Epub 2011 Jan 17.
5. Huang YC, Weng HH, Tsai Y, et al. Periictal magnetic resonance imaging in status epilepticus. Epilepsy Res. 2009; 86(1):72-81.
6. Cianfoni A, Luigetti M, Bradshaw M, et al. MRI findings of crossed cerebellar diaschisis in a case of Rasmussen's encephalitis. J Neurol 2010; 257(10):1748-50.
7. Samaniego E, Stuckert E, Fischbein N, et al. Crossed cerebellar diaschisis in status epilepticus. Neurocrit Care. 2010; 12(1): 88-90.

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