



## Crossed Cerebellar Diaschisis

### Çapraz Serebellar Diaşizis

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#### Dear Editor,

A 33-year-old male who had been followed up for drug-resistant epilepsy since he was a child, was admitted to our neurology clinic with a seizure. Routine biochemical tests resulted as normal and a neurologic examination performed in the postictal period showed dysarthria, bilateral endpoint nystagmus, mild paresis of the right lower limb, and impaired cerebellar tests in the right side. A cranial magnetic resonance imaging (MRI) was performed because electroencephalography showed slowing in the background activity of left hemisphere and left frontotemporal epileptiform activity. Cranial MRI showed atrophy of left cerebral and right cerebellar hemisphere (Figure 1, 2, 3). Neuroradiologic findings were suggestive of crossed cerebellar diaschisis (CCD).

CCD is characterized by impairment of the corticopontocerebellar tract secondary to pathology in supratentorial neural parenchyma resulting in a decrease in blood flow and functional metabolism in the contralateral cerebellar hemisphere in the acute period and atrophy in the chronic period (1). It is usually asymptomatic, unlike the primary pathology. Diaschisis was first defined by von Monakow as “abolition of excitability” and “functional standstill” in the 1870’s, and Baron et al. (2) defined this phenomenon as CCD in 1981. Damage of corticopontocerebellar fibers is usually secondary to cerebral hypoperfusion, but it can also be related with supratentorial hyperperfusion following

revascularization (3). Scientific data show that disruption in corticopontocerebellar fibers causes functional deactivation by leading decrease in excitatory input and a decrease in cerebellar blood flow (4).

CCD mostly accompanies middle cerebral artery infarctions. Sommer et al. (4) found CCD in 35.3% of patients with middle cerebral artery infarction. In particular, it was found to be associated with hypoperfusion involving the left hemisphere, frontal lobe, and thalamus. Also, Förster et al. (5) found CCD in 20% of patients with isolated thalamic infarction. Beyond infarction, CCD can be observed with supratentorial pathologies including Rasmussen’s encephalitis, trauma, status epilepticus, neoplasms, surgery, migraine, and Dyke-Davidoff-Masson syndrome (1). The cerebellar pathology improves in most patients, but cerebellar atrophy develops in 20% of patients. Cerebellar atrophy can occur many years later. Nuclear medicine techniques such as positron emission tomography, single photon emission tomography, and xenon-computed tomography (CT) is used in imaging of CCD. CT/MR perfusion can be used to detect hemodynamic changes. Our patient was a chronic case detected in a routine cranial MRI. CCD should always be kept in mind in the differential diagnosis of unilateral cerebellar atrophy shown in cranial MRI, and a primary pathology should be investigated in such patients.

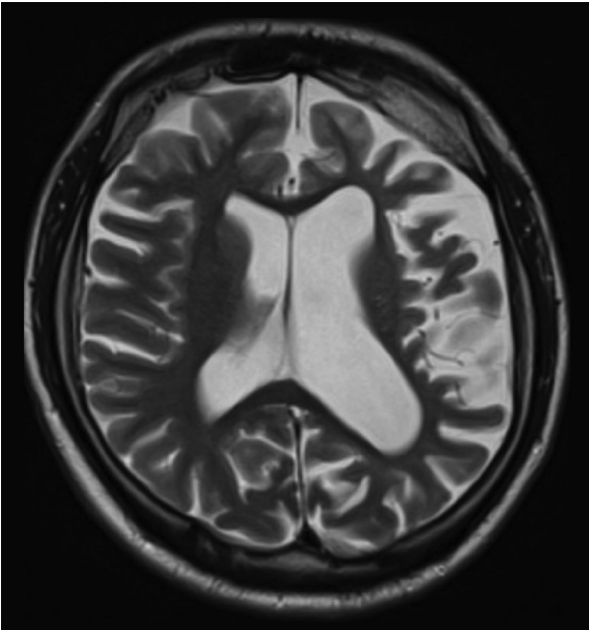
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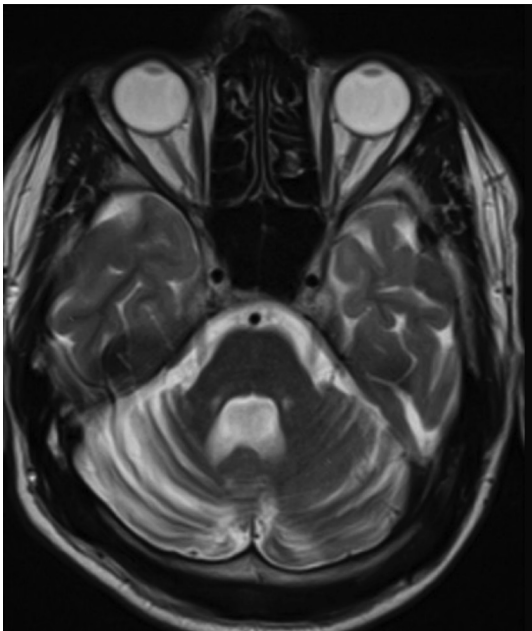
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**Figure 1.** Left cerebral hemispheric atrophy in axial T2-weighted images.

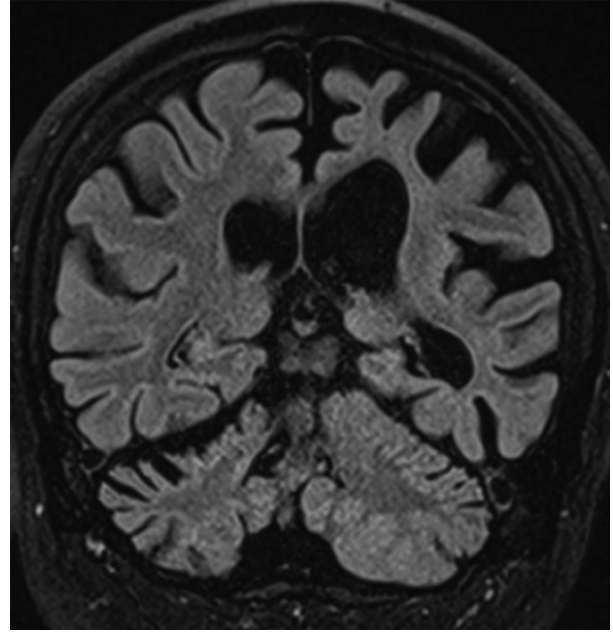


**Figure 2.** Right cerebellar hemispheric atrophy in axial T2-weighted images.

#### Ethics

**Informed Consent:** It was not taken.

**Peer-review:** Internally peer-reviewed.



**Figure 3.** Left cerebral and right cerebellar atrophy are shown in the same section as crossed in coronal fluid-attenuated inversion recovery-weighted images, which are suggestive of crossed cerebellar diaschisis.

#### Authorship Contributions

Surgical and Medical Practices: B.Y., N.K., Concept: B.Y., Design: B.Y., Data Collection or Processing: : M.H.A., B.Y., H.Ç., Analysis or Interpretation: B.Y., N.K., Literature Search: B.Y., N.K., Ö.K.Y., Writing: B.Y., N.K.

**Conflict of Interest:** No conflict of interest was declared by the authors.

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