Crossed cerebellar atrophy: Update

Atrophy of the cerebellum contralateral to a hemispheric supratentorial lesion, or crossed cerebellar atrophy (CCA), has been recognized by neuropathologist for more than 100 years. Diaschisis in neurology signifies reduction of function of a part of the brain following the interruption at a remote site of an afferent pathway, which normally supplies background excitation to the neurons in that part, keeping them in a state of low activity.[1] Crossed cerebellar diaschisis (CCD) is a phenomenon that has been identified on positron emission tomographic (PET) scans in which cerebellar hypometabolism is ascribed to functional disconnection of the contralateral hemisphere from the cerebral cortex. This phenomenon was reported by Baron *et al*.,[2] who observed a parallel reduction in blood flow and oxygen uptake in the cerebellar hemisphere contralateral to the side of supratentorial ischemic infarction.

Crossed cerebellar diaschisis has been observed in supratentorial ischemic strokes, and a variety of brain diseases such as progressively enlarging tumors, arteriovenous malformations, hemorrhages and encephalitis.[3]

The principal mechanism of the transneuronal cerebellar metabolic depression in CCD is thought to be interruption of cerebrocerebellar pathways. This interruption is probable due to damage of the predominantly excitatory corticopontine-cerebellar projections, most of which originate from the frontal and parietal cortices. Although, the CCD may persist or even worsen, it often resolves with time. The mechanism by which cerebellar metabolism returns to normal is unknown.

Crossed cerebellar atrophy has been reported in a few cases after CCD. It has been well accepted that CCD and CCA constitute a spectrum of the same biological process, but the factors that determine a reversible and functional phenomena to become an irreversible structural change are still not fully understood.

Access this article online	
Quick Response Code:	Website: www.ruralneuropractice.com
	DOI: 10.4103/0976-3147.102588

Verhaart observed that CCA tends to be associated with long-standing, extensive unilateral lesions of the cerebral hemisphere, usually originating in the infancy or early childhood. Tien *et al*.[4] were the first to report the possible connections between CCD and CCA. They found that 8 of 26 patients with CCD also had CCA. They suggested that CCA may often be associated with long-term disease such as hemispheric atrophy with seizures. Others authors[5] have suggested that repetitive seizures and status epilepticus could be a main pathogenic factor in CCA.

Niimura *et al*.[6] demonstrated increased glucose utilization and increased binding of benzodiazepine receptors in the contralateral cerebellar hemisphere, when brain injury occurred early in life before one year of age but after the first 4 weeks. Naturally, there is difference in cerebellar functional reorganization pattern following contralateral cerebral injury between developing and developed brains. In children, abnormalities in cerebellar metabolism after cerebral injury are more likely to progress to cerebral atrophy than adults because the immature cerebellum is probably more dependent on the excitatory pathways for normal growth.

Chakravarty[7] suggested that diaschisis presents itself as a different form (ipsilateral or contralateral) depending upon the age of cerebral insult. Early insults would be likely to produce ipsilateral and later ones to produce crossed cerebellar diaschisis. Others workers[8] also commented on this difference in side of cerebellar diaschisis depending upon the age of onset of the cerebral injury. It has been suggested that the production of remote effects, such as crossed and uncrossed cerebellar diaschisis, could be closely related to maturation matruation of the cortico-ponto-cerebellar tract in the developing brain during childhood.

However, in spite of marked cerebellar volume loss in one hemisphere, none of the cases showed any clinical evidence of cerebellar disease. The significant motor deficit and spasticity undoubtedly marked any cerebellar component in the clinical evaluation.

Pedro E Jiménez-Caballero
Department of Neurology,
San Pedro de Alcántara Hospital,
Cáceres, Spain.

Address for correspondence:
Dr. Pedro Enrique Jiménez Caballero,
Neurologist, Calle Dionisio Acedo nº 9,
Portal 7, 4-1ª, 10001, Cáceres, Spain.
E-mail: pjimenez1010j@yahoo.es

References

1. Feeny DM, Baron JC. Diaschisis. Stroke 1986;17:817-30.
2. Baron JC, Bousser MG, Comar D, Castaigne P. Crossed cerebellar diaschisis in human supratentorial infarction. Ann Neurol 1980;8:128.
3. Menon B. Crossed cerebellar atrophy. J Neurosci Rural Pract.2012;3:422-3.
4. Tien RD, Ashdown BC. Crossed cerebellar diaschisis and crossed cerebellar atrophy. Correlation of MR findings, clinical sumptoms, and supratentorial diaschisis in 26 patients. AJR Am J Roentgenol 1992;42:2267-73.
5. Teixeira RA, Li LM, Santos SL, Zanardi VA, Gerreiro CA, Cendes F. Crossed cerebellar atrophy in patients with precocious destructive brain insults. Arch Neurol 2002;59:843-7.
6. Niimura K, Chugani DC, Muzik O, Chugani HT. Cerebellar reorganization following cortical injury in humans. Effect of lesion size and age. Neurology 1999;52:792-7.
7. Chakravarty A. MR evaluation of crossed and uncrossed cerebral-cerebellar diaschisis. Acta Neurol Scand 2003;108:60-5.
8. Sztriha L, Al Suhaili AR, Paris V, Nuk M. Regional cerebral blood perfusion in children with hemiplegia. A SPECT study. Neuropediatrics 1996;27:178-83.

How to cite this article: Jiménez-Caballero PE. Crossed cerebellar atrophy: Update. J Neurosci Rural Pract 2012;3:235-6.
Source of Support: Nil. **Conflict of Interest:** None declared.