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.

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 maturation 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.
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