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Case Report

Cerebellar Cognitive Affective Syndrome in the Context of Crossed Cerebellar Diaschisis Post Pontine Infarction

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Abstract

Crossed cerebellar diaschisis (CCD) is characterized by reduced blood flow and hypometabolism in the cerebellum secondary to a supratentorial lesion in the contralateral cerebral hemisphere. Although CCD due to an infratentorial lesion, particularly a pontine infarction, is rare, it has been previously described. We report a case of a middle-aged man with left paramedian pontine infarction who developed new-onset cognitive dysfunction. Neuropsychological assessment revealed deficits consistent with cerebellar cognitive affective syndrome (CCAS). Brain single-photon emission computed tomography showed hypometabolism in the posterior right cerebellar hemisphere, consistent with CCD. We hypothesize that the cognitive dysfunction, compatible with CCAS following the left pontine infarction, can be explained by CCD-related hypometabolism in the right cerebellum. This case contributes to the limited literature on CCD from infratentorial lesions, emphasizing the potential for CCAS to arise from pontine infarction.

Keywords: *Crossed cerebellar diaschisis; Cerebellar cognitive affective syndrome; Pontine infarction.*

Introduction

Diaschisis refers to the functional and neurophysiological changes that occur in a localized brain area distant from but interconnected with a site of injury. Von Monakow first described it in 1914 (1).

Various forms have been identified, including cortical repercussions of ipsilateral basal ganglia lesions, trans-hemispheric effects of cortical or subcortical lesions, and changes in the contralateral cerebellar hemisphere due to supratentorial lesions (1-3). The latter, known as crossed cerebellar diaschisis (CCD), was first defined in 1981 by Baron et al (4).

CCD is primarily associated with supratentorial lesions, such as cerebral infarctions, causing reduced blood flow and hypometabolism in the contralateral cerebellum. In contrast, CCD due to infratentorial lesions, such as pontine infarctions, is notably rarer, although it has been documented in some cases (8).

Positron-emission tomography (PET) and single-photon emission computed tomography (SPECT) are commonly used to document changes in cerebral blood flow and metabolism in the contralateral cerebellum (4).

Beyond motor coordination, the cerebellum has a significant influence on cognitive and emotional processes. Cerebellar cognitive affective syndrome (CCAS), described by Schmahmann and Sherman in 1998, leads to impairments in executive function, spatial cognition, language, and emotional regulation due to cerebellar damage. Patients may exhibit deficits in planning and memory, emphasizing the cerebellum's broader role in cognitive and affective functioning (5). Understanding these mechanisms is critical for clinical diagnosis and treatment.

In this case report, we describe a rare instance of CCD secondary to an infratentorial lesion, specifically a left pontine infarction, which presented clinically as CCAS.

Case Presentation

A 51-year-old man with no significant medical history presented at the emergency department with severe left-sided facio-brachio-crural hemiparesis and dysarthria. He also reported right-sided neck pain that had been present for two days before admission. Computed tomography angiography of the head and neck revealed a dissection of the right vertebral artery. The patient received intravenous thrombolysis and was subsequently transferred to our institution for further workup and rehabilitation. Brain magnetic resonance imaging (MRI) revealed a left paramedian pontine infarction (Figure 1).

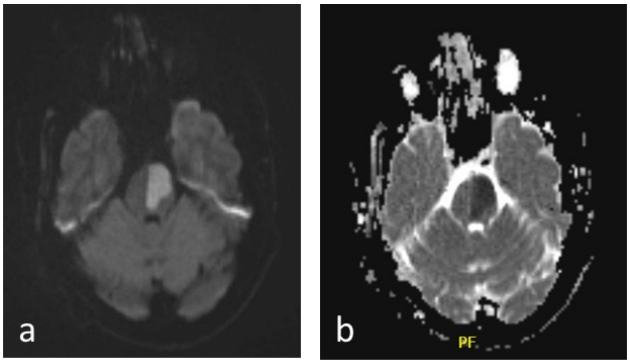

Figure 1. *Brain MRI.*
Axial diffusion-weighted imaging ***(a)*** *and apparent diffusion coefficient map* ***(b)****, performed two days post-stroke, revealed a recent left paramedian pontine infarction.*

During hospitalization, right leg strength improved, but the arm remained weak and non-functional. Unexpectedly, the patient developed new-onset cognitive dysfunction. Neuropsychological assessment at one-month post-stroke revealed slowed processing speed, short-term memory deficits, marked executive dysfunction (including cognitive flexibility, inhibition and lexical fluency), major deficits in selective and visual attention, and significant visuoconstructive impairment.

Brain SPECT demonstrated the known left paramedian pontine infarction and moderate hypometabolism in the posterior right cerebellar hemisphere, consistent with CCD, with no evidence of neurodegenerative disease (Figure 2). Cerebrospinal fluid (CSF) analysis was normal, including Aβ peptide 1-42, tau, and phosphorylated tau levels. Autoimmune and infectious workup was negative. No flow reduction was detected in either the ipsilateral or contralateral frontoparietal cerebral cortex.

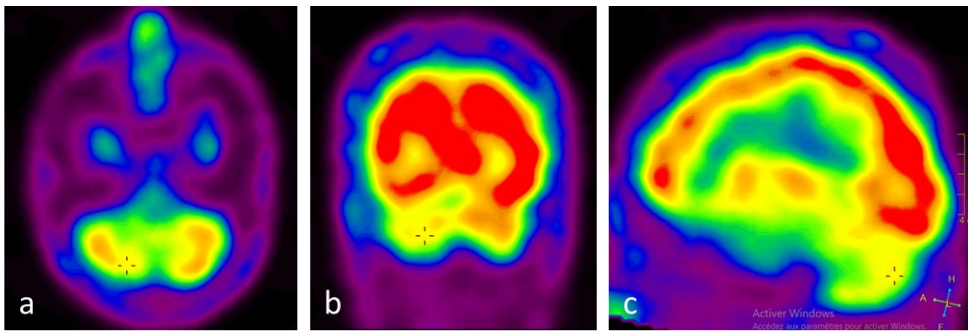

Figure 2. *Brain SPECT.*
Hypometabolism in the posterior right cerebellar hemisphere in axial ***(a)****, coronal* ***(b)*** *and sagittal* ***(c)*** *planes.*

Based on clinical, neuropsychological, brain imaging, and CSF findings, a diagnosis of CCAS was made.

At two months post-stroke, neuropsychological assessment showed some improvement in processing speed, calculation ability, episodic memory, visual selective attention, and task integration. However, significant deficits persisted in executive functions (working memory, flexibility, inhibition), visuoconstructive abilities, and lexical fluency.

During hospitalisation, the patient developed inappetence and depression, for which mirtazapine and paroxetine were initiated, respectively, with a good clinical response.

We hypothesize that the cognitive dysfunction in this case, consistent with CCAS, resulted from CCD-related hypometabolism in the right cerebellum following the left pontine infarction.

Discussion

Like supratentorial strokes, pontine strokes can result in remote disconnections, likely due to damage to the corticopontocerebellar pathways. These pathways involve the pons receiving ipsilateral cortical afferent fibers, which cross the midline to enter the cerebellum via the middle cerebellar peduncle. Efferent cerebellar fibers also cross the midline before projecting to the thalamus through the superior cerebellar peduncle (6).

Rousseaux et al. described asymmetrical cerebellar perfusion characterized by lower activity in the contralateral, and less commonly in the ipsilateral, cerebellar lobe (7). Using the cerebral perfusion tracer ^{99m}Tc-hexamethylpropyleneamine oxime (^{99m}Tc-HMPAO), they reported a case of paramedian pontine infarction associated with reduced tracer uptake in the contralateral cerebellum.

Cognitive impairment after stroke varies depending on lesion location. The impact of brainstem damage on cognition remains underreported. A recent systematic review highlighted a spectrum of cognitive deficits following pontine injury, particularly executive dysfunction, in patients aged 63 to 86 years. The authors suggest that cognitive decline after a pontine stroke may result from frontopontocerebellar diaschisis (7).

For many years, the cerebellum was regarded primarily as a structure involved in motor control. However, research over the past few decades has increasingly demonstrated the cerebellum's critical role in cognitive and affective functions. Lesions of the cerebellar posterior lobe are now known to result in CCAS, a syndrome characterized by deficits in executive functioning, visuospatial processing, language skills, and affect regulation (5).

The functional distinction between the sensorimotor cerebellum, located in the anterior lobe, and the cognitive-limbic cerebellum, primarily situated in the posterior lobe, helps explain the diversity of symptoms seen in cerebellar dysfunction. Executive function impairment is a prominent feature of CCAS, followed by language, visuospatial, and affective changes (8, 9).

In our case, the patient exhibited clear signs of cognitive dysfunction consistent with CCAS following his pontine infarction. Neuroimaging, particularly SPECT, demonstrated hypometabolism in the right cerebellum contralateral to the pontine lesion, confirming the presence of CCD. Although a formal assessment using the CCAS scale was not performed, neuropsychological evaluation revealed significant deficits in executive functioning, attention, memory, and emotional regulation. It is important to note that not all features of CCAS (executive, linguistic, visual spatial, affective) are necessarily present in every patient with damage to the cognitive/limbic cerebellum (8).

Unfortunately, the patient was lost to follow-up, limiting our ability to track the long-term progression of his cognitive symptoms and imaging control.

Conclusion

In summary, this case highlights the importance of recognizing non-motor impairments in patients with cerebellar disorders, particularly those involving the posterior cerebellum. Cognitive dysfunction following a pontine infarction, especially when consistent with CCAS, should prompt further investigation for possible CCD. Early identification and comprehensive neuropsychological assessment are crucial for diagnosing CCD and guiding appropriate management strategies.

Disclosure Statement

No potential conflict of interest was reported by the author(s).

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None.

Data Availability Statement

Because this is a case report, there are no associated research data to be shared.

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