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Crossed cerebellar diaschisis after stroke identified noninvasively with cerebral blood flow-weighted arterial spin labeling MRI☆

Megan K. Strother[a], Cari Buckingham[a], Carlos C. Faraco[a], Daniel F. Arteaga[a], Pengcheng Lu[b], Yaomin Xu[b], Manus J. Donahue[a,*]

[a] Department of Radiology and Radiological Sciences, Vanderbilt Medical Center, Nashville, TN, USA
[b] Center for Quantitative Sciences, Vanderbilt Medical Center, Nashville, TN, USA

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ABSTRACT

Background and purpose: Crossed cerebellar diaschisis (CCD) is most commonly investigated using hemodynamic PET and SPECT imaging. However, noninvasive MRI offers advantages of improved spatial resolution, allowing hemodynamic changes to be compared directly with structural findings and without concerns related to ionizing radiation exposure. The aim of this study was to evaluate relationships between CCD identified from cerebral blood flow (CBF)-weighted arterial spin labeling (ASL) MRI with cerebrovascular reactivity (CVR)-weighted blood oxygenation level dependent (BOLD) MRI, Wallerian degeneration, clinical motor impairment, and corticospinal tract involvement.
Methods: Subjects ($n = 74$) enrolled in an ongoing observational stroke trial underwent CBF-weighted ASL and hypercapnic CVR-weighted BOLD MRI. Hemispheric asymmetry indices for basal cerebellar CBF, cerebellar CVR, and cerebral peduncular area were compared between subjects with unilateral supratentorial infarcts ($n = 18$) and control subjects without infarcts ($n = 16$). CCD required (1) supratentorial infarct and (2) asymmetric cerebellar CBF (>95% confidence interval relative to controls).
Results: In CCD subjects ($n = 9$), CVR ($p = 0.04$) and cerebral peduncular area ($p < 0.01$) were significantly asymmetric compared to controls. Compared to infarct subjects not meeting CCD criteria ($n = 9$), CCD subjects had no difference in corticospinal tract location for infarct ($p = 1.0$) or motor impairment ($p = 0.08$).
Conclusions: CCD correlated with cerebellar CVR asymmetry and Wallerian degeneration. These findings suggest that noninvasive MRI may be a useful alternative to PET or SPECT to study structural correlates and clinical consequences of CCD following supratentorial stroke.
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1. Introduction

Crossed cerebellar diaschisis (CCD) refers to a depression of CBF and metabolism affecting the cerebellar hemisphere contralateral to supratentorial lesions. CCD manifests as decreased cerebellar CBF and CBV, which is thought to be secondary to arterial vasoconstriction but with retained cerebrovascular reactivity (CVR) capacity [1,2]. CCD has been predominantly studied through hemodynamic PET [3] and SPECT [4] imaging. While these imaging modalities have greatly improved our understanding of CCD, the radioactive tracers required limit their longitudinal monitoring potential, a characteristic that is necessary to understand the temporal progression and pathophysiology of CCD. Further, PET and SPECT lack the spatial resolution achieved with MRI, which is necessary to characterize structural findings that precipitate or result from CCD. Perfusion MRI utilizing exogenous contrast agents has had mixed results to date, is contraindicated in some patients, and in cases of ischemia remains largely qualitative in nature owing to difficulties in accurately quantifying regional arterial input functions [5]. Alternatively, noninvasive MRI methods for characterizing CCD would have few patient restrictions and thus are of particular interest for more readily evaluating novel stroke rehabilitation techniques [6] and functional outcomes [7,8].

Yamada et al. first characterized CCD with hemodynamic MRI utilizing DSC-MRI [7]. While this remains the most frequently reported MRI metric for CCD, some studies report DSC-MRI lacks sensitivity compared to PET [9]. When evaluating the body of work on CCD across all imaging modalities, two important limitations should be considered. First, posterior vascular stenosis has not always been considered [4,7,10]. Therefore, flow changes due to

☆ All work was carried out at the Vanderbilt Medical Center, Nashville, TN, USA.
* Corresponding author at: Vanderbilt University Institute of Imaging Science, Rm. AAA-3115, 1161 21st Ave South, Nashville, TN 37232, USA. Fax: +1 615 322 0734.
E-mail address: mj.donahue@vanderbilt.edu (M.J. Donahue).

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vertebral artery stenosis could impact the CCD cohort. Second, as most CCD studies have focused on hemodynamic findings identified with PET and SPECT, hemodynamic sequelae of CCD have been studied more thoroughly than the structural changes which cause or are associated with it, which are less well understood [1,10,11].

In a case study, noninvasive arterial spin labeling (ASL) MRI identified asymmetric CBF consistent with CCD [12]. Alternatively, CVR-weighting can readily be obtained using the well-established blood oxygenation level-dependent (BOLD) MRI approach in conjunction with a hypercapnic gas stimulus. To our knowledge, only one prior report used a noninvasive gas challenge with fMRI to study CCD, reporting cerebellar asymmetry with reduced CBV and CBF in acute MCA stroke subjects following a hyperoxic respiratory challenge [13].

The aims of the present study were to understand to what extent CCD correlated with (1) cerebellar CVR asymmetry from hypercapnic BOLD MRI (2) motor impairment (3) infarct location involving the corticospinal tract and (4) Wallerian degeneration. Our working hypothesis for using BOLD MRI in CCD is that reduced baseline CBF contralateral to the hemisphere with supratentorial infarcts will translate to a smaller vasoreactive response. In addition, we hypothesize CVR should be asymmetric between cerebellar hemispheres in a manner that reflects baseline hemodynamic tone in the face of preserved CVR capacity.

2. Materials and methods

The goal of this work was to stratify patients with supratentorial infarcts into groups with vs. without cerebellar hemispheric asymmetry in CBF. For these two groups, we evaluated mean differences in (1) cerebellar CVR asymmetry (2) motor impairment (3) infarct location involving the corticospinal tract and (4) Wallerian degeneration. A control group without infarct was also included for comparison. Below we outline the details for this set of experiments.

2.1. Ethical considerations

The local Institutional Review Board approved this study and work was carried out in accordance with The Code of Ethics of the World Medical Association (Declaration of Helsinki). Subjects ($n = 74$) were enrolled between January 2010 and June 2014 as part of the Assessment of Multimodal MRI in Patients at Risk for stroke with Intracranial Stenosis trial and provided informed, written consent.

2.2. MRI acquisition

All participants were scanned on the same scanner. All subjects underwent MRI at 3.0 T with ASL, hypercapnic BOLD, and structural imaging; data were acquired with body coil radiofrequency transmission and a 16-channel multi-array SENSitivity Encoding (SENSE) head coil for reception.

For baseline CBF assessment, a pseudocontinuous ASL (pCASL) sequence was used with a 1.6 s labeling pulse train consisting of Hanning-windowed 0.7 ms pulses, followed by a post-labeling delay of 1.525 s (TR = 4 s; TE = 13 ms; spatial resolution=$3 \times 3 \times 7\ mm^3$). A separate equilibrium magnetization image was acquired with identical geometry but spin labeling preparation removed for M_0 calculation.

For CVR assessment, BOLD hypercapnic-hyperoxic (carbogen; 5% CO_2, 95% O_2) CVR-weighted scans were performed using a standard T_2^*-weighted echo-planar imaging sequence (TR/TE = 2000/35 ms; spatial resolution = 3.5 mm isotropic) in a block-design paradigm. The stimulus paradigm consisted of two blocks of 3 min hypercapnic hyperoxic administration interleaved with medical air (~21% O_2/~79% N_2/<1% trace elements), all delivered at 12 L/min through a customized facemask setup. Physiological monitoring was achieved using an In Vivo Research Inc. device and Millennia Vital Systems Monitoring System (3155 MVS). Monitored parameters included heart rate, blood pressure, arterial oxygen saturation fraction (Y_a), and $EtCO_2$ (Salter Labs; Ref: 4000F).

For structural imaging, whole-brain T_1-weighted MPRAGE images with TR/TE = 8.9/4.6 ms, flip angle = 8°, spatial resolution = 1 mm isotropic; 2D turbo-inversion-recovery T_2-FLAIR with TR/TE = 11000/120, flip angle = 90°, spatial resolution = $0.9 \times 1.0 \times 5$ mm; and 3D T_1-weighted gradient echo time-of-flight angiography with TR/TE = 13.9/1.9 ms, flip angle = 20°, spatial resolution = $0.8 \times 0.8 \times 1.0$ mm were acquired.

2.3. MRI analysis

For basal CBF, ASL data were processed by an imaging physicist blinded to the structural analysis, surround-subtracted [14] and normalized by M_0 to generate CBF-weighted maps. CBF was quantified via application of the solution of the flow-modified Bloch equation [15]. To allow for comparison between subjects, all images were co-registered to a standard 4 mm T_1-weighted Montreal Neurological Institute atlas. Mean CBF for each cerebellar hemisphere was calculated within the Harvard-Oxford cerebellar atlas (region-of-interest shown in Results). Note that regions-of-interest were defined from standard cerebellar atlases for CBF analysis and the same regions were used in all subjects.

For CVR analysis, the same imaging physicist, blinded to structural imaging findings, analyzed the BOLD data, which were corrected for motion and baseline drift using standard affine correction algorithms. CVR was calculated as the z-statistic of the BOLD time course, which is defined as the magnitude of the signal change with hypercapnia normalized by the standard deviation of the baseline signal [16].

Infarct was determined by a board-certified neuroradiologist with criteria being lesion on FLAIR greater than 3 mm with confirming T_1 encephalopathy. For some patients, the image shown is only one lesion in a string of infarcts. The single punctate T_2 change may not meet 3 mm criteria alone but were included as infarct if in the setting of a string of T_2 changes in deep or subcortical white matter which are characteristic of watershed infarcts. The watershed pattern of infarcts was common in patients with Moyamoya disease. This pattern is distinct from periventricular white matter changes, which may not represent lacunar infarcts.

For Wallerian degeneration analysis, the area of the cerebral peduncles was measured from the T_1-weighted images separately by two neuroradiologists, blinded to clinical history and supratentorial findings, and CBF and CVR analysis outlined above. Peduncular area was manually drawn on the T_1-weighted axial images using the method of Mark et al. [17]. Using Medical Image Processing, Analysis & Visualization v7.0.1, a region-of-interest was drawn on the axial image with maximum peduncular width using a straight line to connect the oculomotor nerve sulcus in the prepontine cistern to the lateral sulcus of the midbrain, then filling in the peduncle and calculating the cross-sectional area (Fig. 1).

Vertebrobasilar stenosis was classified by a neuroradiologist from angiography performed within 30 days of BOLD imaging (DSA, CTA, or MRA). Stenosis degree was classified as the ratio of the width of the stenosed lumen (measured in the plane with the most severe stenosis) to the width of the normal distal vessel.

2.4. Motor impairment determination

Clinical motor function was assessed retrospectively by a neurologist blinded to MRI findings using electronic medical records to determine motor function surrounding the time of the BOLD

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