

Mixed Aphasia Caused by Bilateral Cerebellar Infarcts: A Case Report

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Case Report

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Abstract

Although neuroanatomical and physiological understanding of the cerebellum has evolved over recent decades and continues to develop, there is much that remains to be expounded upon, especially with regards to nonmotor roles. Neurocognitive and language processing is one area where involvement of the cerebellum is no longer in question, but the extent and mechanism of this relationship has yet to be defined. For example, which of the cerebellar hemispheres is involved continues to be debated. We present a case wherein a thrombus in the basilar artery led to bihemispheric cerebellar strokes with profound mixed effects on the patient's language and cognition. To the authors' knowledge this is the first published case of bilateral cerebellar strokes resulting in a mixed aphasia reported in scientific literature. This demonstrates the importance of continued research into a model for cerebellar function and the clinical impact of lesions to various cerebellar regions.

Introduction:

The conventional model of cerebellar function suggests that its neurophysiologic role involves somatic and autonomic motor coordination, but understanding of its nonmotor functions has expanded over recent decades. Due to interconnectivity between cerebellar structures and cerebral association areas as well as multiple vascular supplies, localization of function within the cerebellum is a complex topic; however, assessment of patients with cerebellar lesions and the advent of functional magnetic resonance imaging (fMRI) have improved our understanding of the topography of cerebellar function¹⁻³. Because nonmotor deficits from cerebellar lesions are often not as severe or reproducible as from lesions to a cerebral counterpart, and due to poor localization, these deficits are often missed^{1,4}.

Whether due to the subtlety of deficits caused by cerebellar lesions or because of heterogeneity of cerebellar circuitry between individuals, various models and theories of cerebellar function with regards to cognition and speech have been proposed, but there is lack of conformity between experts on the localization of various nonmotor cerebellar functions^{2,4}. This becomes increasingly important in an era when post-stroke management is introducing modalities such as transcranial direct current stimulation (tDCS) that can be targeted to certain areas of the brain to potentially improve recovery⁵⁻⁷. Formal correlation of cerebellar regions to elements of speech and neurocognition could have an exponential impact on the future management of stroke and other cerebellar lesions. We present a case of mixed aphasia and impaired cognition caused by bilateral cerebellar infarcts, which appears to be the first published case involving this presentation, for the purpose of furthering understanding about this underrepresented pathology.

Patient and Methods:

A 75-year-old right-handed Caucasian male with a past medical history significant for rectal adenocarcinoma two years status-post resection as well as hypertension, hyperlipidemia, and former tobacco use presented to the emergency department after developing left sided weakness and difficulty

with speech beginning more than twelve hours prior. A stat noncontrast computed tomography (CT) of the head was unremarkable, but CT perfusion study revealed ischemic penumbrae in bilateral cerebellar hemispheres. CT angiography was positive for a distal basilar artery thrombus at the junction of the posterior cerebral arteries [Fig. 1] as well as calcified atherosclerotic plaques without significant stenosis throughout the carotid and vertebral arteries. On arrival, blood pressure was 107/61 mmHg, pulse was 70, respiratory rate was 16, and temperature was 36.8°C. Routine laboratory results were unremarkable and serum glucose was 154 mg/dL.

An intravenous (IV) heparin drip was started before the patient was transferred to a comprehensive stroke center for potential thrombectomy. Ultimately, a thrombectomy was deferred by the interventional radiologist due to high-risk location of the thrombus. Upon evaluation by the neurology service, the patient was found to have incomplete deficits in both fluency and comprehension. He had intermittent deficiencies in naming and was able to repeat words and short phrases but had intermittent difficulty repeating complex sentences. These findings remained consistent on subsequent speech therapy evaluations. He was found to have dysdiadochokinesia and dysmetria in bilateral upper extremities and was no longer found to have lateralizing weakness. No visuospatial or cranial nerve deficits or nystagmus were noted.

Later comprehensive evaluation of the patient's cognition and language found that he was oriented to self, place, time, and situation, but intermittently became confused with other simple questions. He had difficulty articulating abstract concepts and understanding illustrated pictures. He had mild deficits naming objects and pictures, with 75% accuracy. He could repeat single words with 80% accuracy, short phrases with 60% accuracy, and complete sentences with 40% accuracy. He could declare opposites with 80% accuracy. His responses to questions with single-word answers had approximately 70% accuracy, whereas he correctly answered yes/no questions with 80% accuracy. He followed single-step motor commands with 100% accuracy, but with two-step commands had 66% accuracy, and he occasionally perseverated with commands. He could read short phrases with 100% accuracy, and sentences with 75% accuracy. Handwriting fluency had 0% accuracy, although with legible letters. The patient had difficulty with simple math problems, but could correctly write the numbers on a clock. His immediate and delayed recall of wordsets was impaired with overall 15% accuracy. His speech deficits were characteristic of verbal apraxia and word-finding difficulty without dysarthria, and he was noted to have frequent paraphasias involving consonants and categorically similar words. The patient was briefly suspected of having underlying dementia, but further conversation with his family confirmed that these abnormalities were not previously present.

Contrast enhanced magnetic resonance imaging (MRI) of the brain was positive for embolic-appearing small scattered infarcts in the superior aspect of bilateral cerebellar hemispheres, with no abnormal enhancement to suggest any metastatic disease [Fig. 2-3]. A transthoracic echocardiogram (TTE) initially reported a thrombus in the ventricular apex, however this was no longer seen on a repeat TTE with Definity contrast two days later. The cardiology service recommended transition from IV heparin to warfarin, however frank blood was noted in the patient's stool and antithrombotics were held. The

gastroenterology service was consulted and colonoscopy revealed multiple fungating, irregular, friable masses in the rectum, eventually shown on biopsy to be tubulovillous adenomas with high grade dysplasia.

Because anticoagulation was suspended for gastrointestinal blood losses, and because of the patient's persistent speech deficits, a repeat MRI of the brain was obtained eight days after initial presentation, with close scrutiny given to the frontotemporal lobes. No additional infarcts were found. Metabolic and infectious workup was negative for other underlying causes of changes in mentation. The strokes were suspected to be caused by cardioembolism to the basilar artery due to hypercoagulability in the setting of rectal cancer. Ultimately, the patient was started on warfarin and temporarily bridged with enoxaparin, and was discharged to acute rehabilitation with some mild improvements in his speech noted after repeated speech therapy.

Discussion:

Although cases associating cerebellar infarcts with deficits in language and cognition have been reported for nearly three decades, a review of available literature found no other reported cases of bihemispheric cerebellar strokes in this context. Case reports of cerebellar infarcts associated with language deficits appear to favor the right more often than left hemisphere⁸⁻¹⁰. Infarcts involving the distribution of the superior cerebellar artery also appear to be common, as was found in our patient. Due to fMRI studies showing a functional connection between the right cerebellar hemisphere and the left temporal, frontal, and parietal lobes, until recently it was thought that the left cerebellar hemisphere is not involved in language processing^{4,11-12}. This has impacted not only models for cerebellar neurophysiology, but also guidelines for treatment using neuromodulation⁵. Although our hospital is not equipped to perform single-photon emission computerized tomography (SPECT) or fMRI for further evaluation of subsequent language function, having a second MRI more than one week afterwards showing no additional lesions gives us reasonable confidence in the proximate cause.

The effect of bilateral cerebellar infarcts on language and cognition is relevant in the setting of the supposition that diaschisis is responsible for changing the function of the cerebral language areas after the loss of cerebellar input^{2,4,11,13}. Given the interconnectivity of the cerebellar circuitry, it is reasonable to conjecture that impairment of more than one cerebellar hemisphere may impact language differently than a single affected hemisphere. This may help explain why deficits experienced after a cerebellar infarct do not always match cerebellar involvement in tasks seen on the fMRI of healthy volunteers¹⁴. It is noteworthy that in our case, the patient demonstrated deficits in both language and cognition, which have been intrinsically related in prior research¹⁵. The relatively small infarcts resulting in deficits in language, cognition, and motor coordination demonstrate that the structures involved in these functions are likely closely interrelated. This patient also had only partial deficits in several language domains, including semantic fluency, comprehension, naming, repetition, reading, and writing, which implies that language processing could be integrated throughout various cerebellar structures rather than spatially correlated to

discrete topographical regions. Interestingly, no dysarthria was noted, although this is commonly reported among other cases of language deficits related to cerebellar infarct^{1,2,4,11}.

Conclusion:

The pathophysiology of language and cognitive deficits in the setting of cerebellar infarcts remains an evolving subject that will benefit from further investigation. Although the importance of the cerebellum to language and neurocognition is established, wider recognition of this phenomenon will improve understanding of the mechanism of these deficits, as some heterogeneity is noted among reported cases. Although verbal apraxia and dysarthria are frequently reported sequelae of unilateral cerebellar infarcts, in this case the patient developed verbal apraxia, word finding difficulty, mixed aphasia, and cognitive impairment along with motor ataxia following bilateral superior cerebellar infarcts. It is possible that post-stroke changes in neurophysiology due to diaschisis may cause substantial phenotypic variation. The neuroanatomical correlation of these deficits is clinically relevant for evolving neurostimulation techniques that may improve recovery from debilitating strokes.

Declarations:

Ethical approval: All information included within this manuscript was obtained in accordance with the ethical standards of the institutional review board at Roseman University and as outlined by the CARE guidelines for case reports.

Consent to publish: Informed consent was obtained in accordance with the CARE guidelines.

Competing interests: The authors have no relevant financial or non-financial interests to disclose.

Authors' contributions: T.C. wrote the manuscript text, performed a literature review, and prepared figures 1-2. C.D. performed a literature review. S.V. provided images from the case. P.J. and A.V. provided editing for the manuscript. A.V. prepared figure 3. All authors reviewed the manuscript before submission for publication.

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Figures



Figure 1

Coronal view of computerized tomography angiogram (CTA) of the head and neck indicating distal basilar artery thrombus at the junction of the posterior cerebral arteries (red arrowhead).

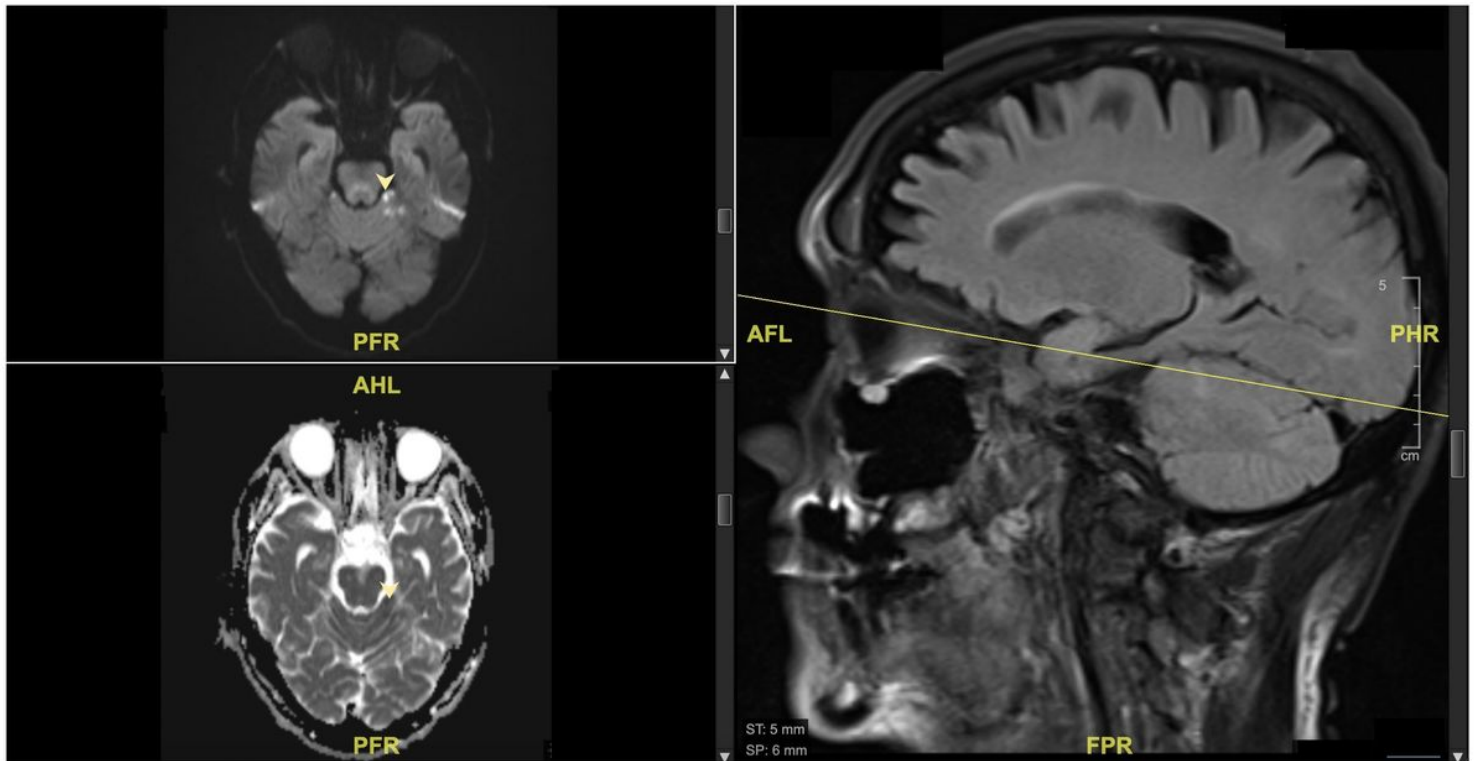


Figure 2

Axial diffusion-weighted imaging (DWI) and apparent diffusion coefficient (ADC) sequences indicating superior-most cerebellar infarcts (yellow arrowheads). Sagittal fluid attenuated inversion recovery (FLAIR) sequence demonstrates infarcts are within superior cerebellar artery vascular territory (yellow line).

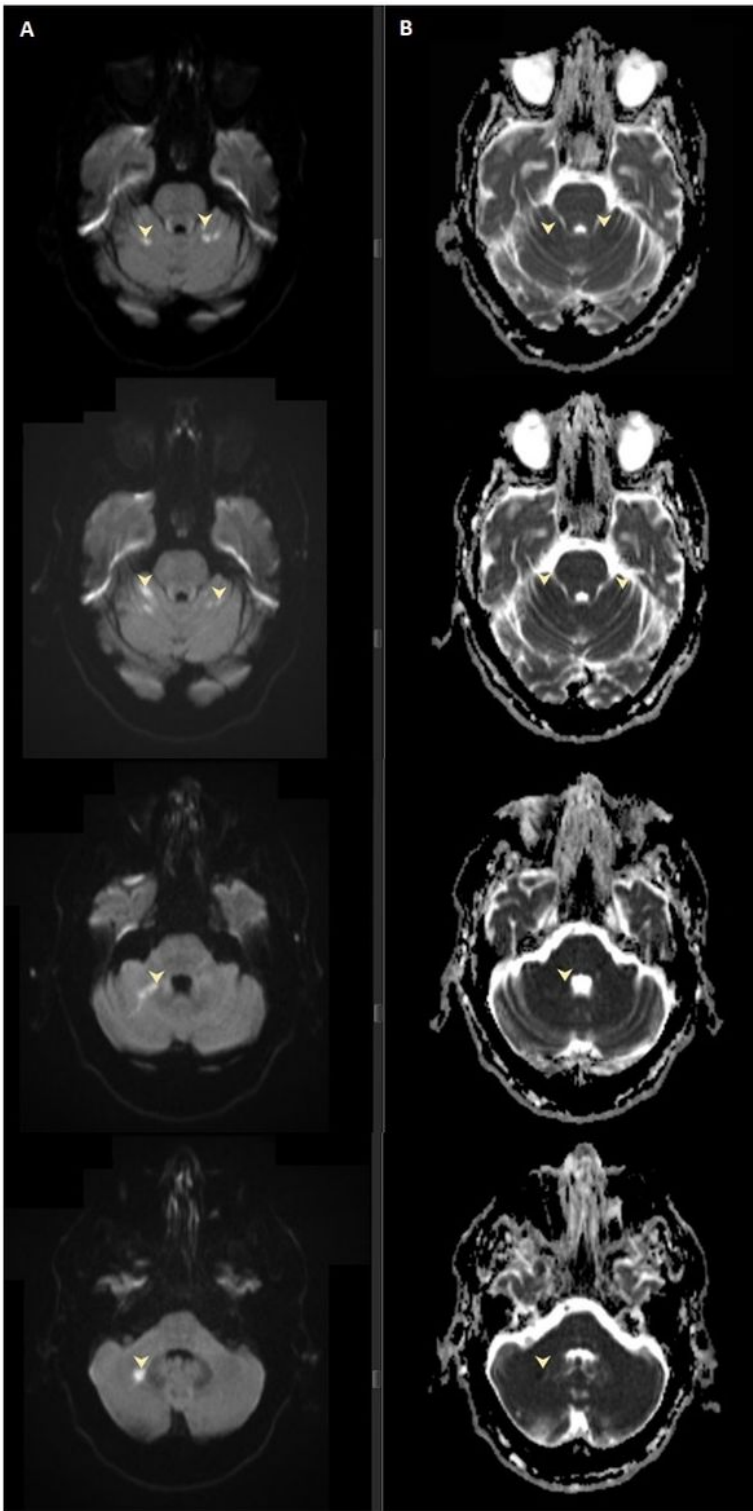


Figure 3

Axial DWI (Panel A) and ADC (Panel B) sequences indicating multiple bihemispheric cerebellar infarcts (yellow arrowheads).