

Higher Visual Function Deficits Post Stroke

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A 62-year-old right-handed woman with history of type 2 diabetes mellitus and hypertension noticed sudden loss of vision in the temporal field of the right eye along with paresthesias in the right side of the face. Noncontrast CT head in the emergency department showed acute infarction. MRI brain confirmed diffusion restriction in the territory of left posterior cerebral artery (Figure 1A,B). On examination, vision was 20/30 and 20/40 with mild right relative afferent pupillary defect (RAPD). Ocular motility was full. Ophthalmoscopy revealed mild diabetic retinopathy in each eye. Automated perimetry demonstrated right homonymous hemianopia (Figure 1C). She struggled reading Ishihara plates and was unable to identify the control plate, reading the numerical figures as letters; however, she was able to correctly identify the colors of the objects

presented to her. She was able to correctly describe the Boston Cookie theft picture.¹

This patient's ability to recognize objects' color but inability to identify the control Ishihara plate indicated that her problem was not due to color vision deficit. Her ability to correctly describe Boston Cookie theft picture confirmed that she did not suffer from simultanagnosia. When asked to write a sentence, she was able to do so easily; however, she could not read it back 15 seconds later, indicating the presence of pure alexia or alexia without agraphia and localizing the lesion to the occipital lobe of the dominant hemisphere as well as splenium of the corpus callosum (Figure 1B). The subtle right RAPD indicated extension of the lesion to also involve the left optic tract as it enters the thalamus.

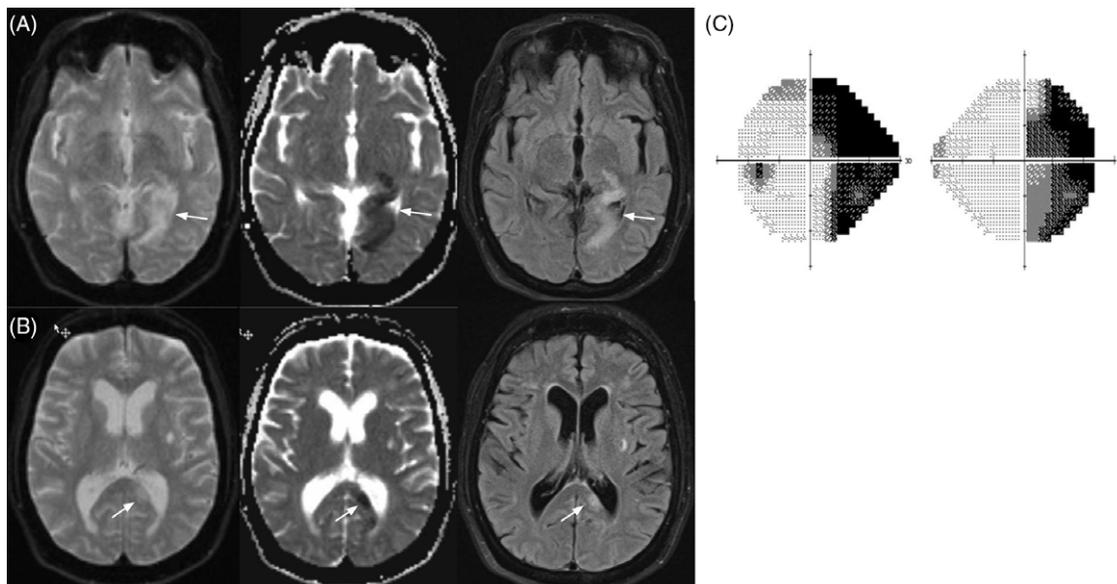


Figure 1: Axial MR images showing diffusion restriction (left) and associated decreased diffusion coefficient (ADC, center) involving the left thalamus, occipital-parietal lobe (A) and splenium of corpus callosum (B) that is hyperintense on T2/FLAIR (right). (C) Humphrey 24-2 visual field testing showed a near complete right homonymous hemianopia.

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Alexia without agraphia is a cortical disconnection syndrome which occurs when the visual information cannot reach the angular gyrus, a region in the parietal lobe of the dominant lobe which is involved in language processing and semantic memory critical for reading.¹ Input from the occipital cortex of the dominant hemisphere is unavailable as it is directly damaged, producing homonymous hemianopia, while the intact contralateral occipital cortex cannot communicate with the angular gyrus due to the lesioned splenium of the corpus callosum which carries these decussating fibers to the contralateral hemisphere. Thus, the patient is able to write but is unable to read a sentence they have just written (Video 1).² Letter identification may also be impaired, although the patient can match or draw the shape of the letter (Video 2). Spelling, language comprehension, and ability to hold a conversation are unaffected.³

The differential diagnosis for reading difficulty is wide. The explanation may be simple, such as age-related presbyopia, dry ocular surface, or convergence insufficiency. In patients with the history of stroke, however, disorders of higher visual processing should be suspected as they can be difficult to pinpoint unless specifically thought of. Visual field testing is also an important part of the examination as homonymous hemianopia, particularly right-sided, interferes with normal saccadic patterns in reading and can produce a word-length effect similar to that seen in alexia.⁴

Recognition of alexia allows for better understanding of the patient's experience and allows for the adoption of management strategies such as using pictograms instead of text for shopping lists and use of apps to convert text to speech. Rehabilitation strategies including listening to repetitive word reading with simultaneous display of the text and tactile-kinesthetic training where patients trace letter outlines on their own skin to improve letter recognition have also shown to be beneficial.⁵

KEY POINTS:

1. Disorders of higher visual processing should be thought of in all patients who experience reading difficulties after stroke.
2. Asking a patient to read the sentence that they have written can allow quick recognition of alexia with or without agraphia which localizes the lesion to dominant occipital lobe as well as splenium of corpus callosum.
3. Ability to synthesize and process visual information is dependent on occipitoparietal and occipitotemporal connections (the so-called “where” and “what” pathways).

DISCLOSURES

The authors have no conflicts of interest to declare.

SUPPLEMENTARY MATERIAL

For supplementary material accompanying this paper visit <https://doi.org/10.1017/cjn.2021.211>

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