Expressive Language Impairment in the Visual Input Modality: A Case Report

Le trouble du langage expressif qui se manifeste dans les tâches utilisant des stimuli visuels : une étude de cas

KEYWORDS

APHASIA

CASE STUDY

POSTERIOR CEREBRAL ARTERY STROKE

ALEXIA WITHOUT AGRAPHIA

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Abstract

A limited number of cases have previously been described following a stroke involving the left posterior circulation in which patients exhibit a selective deficit in naming objects presented visually in the absence of overt agnosia. We present language and cognitive assessment results for a patient with a similar profile following a left posterior cerebral artery territory ischemic infarct by employing the Western Aphasia Battery–Revised, Cognitive Linguistic Quick Test–Plus, subtests of the Boston Diagnostic Aphasia Examination–Third Edition, and other informal assessment tasks. The patient demonstrated specific impairment in tasks of expressive language involving visual input (i.e., verbal or written object/picture naming and picture description) compared to those involving other modalities (i.e., tactile input, auditory input) or based in spontaneous expression. This was in addition to alexia without agraphia, right-sided homonymous hemianopsia, and impaired colour naming. This case report is unique in that it provides a full characterization of the language impairment profile for a patient with this atypical presentation within the acute post-stroke period. We discuss how the patient's impairment profile may align with a model proposing incomplete access to semantic representations from visual input in the context of intact structural representations. By adding to the literature in this area, this case report may aid clinicians in recognizing similar atypical patient presentations and subsequently designing appropriate rehabilitation interventions.

Abrégé

À ce jour, on recense peu d'études de cas décrivant des patients chez qui, à la suite d'un accident vasculaire cérébral dans les artères irriguant la portion postérieure gauche du cortex, on observe un déficit touchant spécifiquement la dénomination d'objets lorsqu'ils leur sont présentés de façon visuelle, et ce, en l'absence de signes apparents d'agnosie. Le présent article présente les résultats d'une évaluation des fonctions cognitives et langagières réalisée auprès d'un patient ayant un profil similaire à la suite d'un accident vasculaire ischémique dans le territoire irrigué par l'artère cérébrale postérieure gauche. Plus précisément, les fonctions cognitives et langagières du patient ont été évaluées à l'aide de la *Western Aphasia Battery–Revised, du Cognitive Linguistic Quick Test–Plus,* de sous-tests de la *Boston Diagnostic Aphasia Examination–Third Edition*, ainsi que d'autres tâches d'évaluation informelles. Le trouble du langage du patient se caractérisait spécifiquement par de faibles performances dans les tâches qui évaluaient le langage expressif en utilisant des stimuli visuels (p. ex. images ou mots écrits d'objets; tâches de dénomination ou de description d'objets) et de fortes performances en discours spontané et dans des tâches qui évaluaient le langage expressif en utilisant des stimuli présentés dans des modalités autres que visuelles (p. ex. tactiles ou auditives). À cela s'ajoutait une alexie sans agraphie concomitante, une hémianopsie homonyme droite et une difficulté à nommer les couleurs. Cette étude de cas est unique du fait qu'elle fournit une description détaillée du profil langagier en phase aiguë d'un patient ayant un tableau clinique atypique à la suite d'un accident vasculaire cérébral. Dans cet article, nous discutons de la façon dont le profil du patient semble cohérent avec un modèle qui propose un accès limité aux représentations sémantiques des stimuli visuels et dans lequel les fonctions de reconnaissance des formes demeurent intactes. Par son apport à la littérature, cette étude de cas pourrait aider les cliniciens à reconnaître les tableaux cliniques atypiques similaires chez leurs patients en vue d'établir un plan d'intervention approprié.

Accurate recognition of the characteristics of visual and language impairments from acute stroke are important in the early phase to assist in determining appropriate rehabilitation treatment modalities. A limited number of cases have been described in which patients exhibit a selective deficit in naming objects presented visually, with a relatively preserved ability to name them when presented through another sensory modality (Campbell & Manning, 1996; Coslett & Saffran, 1992; Ferreira et al., 1997; Hillis & Caramazza, 1995; Kwon & Lee, 2006; Lhermitte & Beauvois, 1973; Marsh & Hillis, 2005; Plaut, 2002; Rodrigues et al., 2008). Unlike agnosia, patients can demonstrate recognition of the objects presented. Such impairment typically follows a stroke involving the left posterior cerebral artery (PCA) territory, with involvement of the dominant left occipito-temporal region and splenium of the corpus callosum (Ferreira et al., 1997). In the first described case, the term "optic aphasia" was coined for this presentation (Freund, 1889). Past reports have typically presented patients in the subacute or chronic period post-stroke and have focused their assessments on verbal naming skills rather than describing the overall language impairment profile.

Several models of semantics have been used to explain this selective deficit in naming from a visual input modality (Rodrigues et al., 2008). These models acknowledge that visual processing occurs in the right hemisphere of these patients, given their left occipital lobe lesion(s). To varying degrees, they also factor in damage to the splenium of the corpus callosum, which impairs information transmission between hemispheres. Beauvois (1982) proposed a disconnection between modality-specific semantic systems (visual and verbal), resulting in intact access of visual input to visual semantics, but of only non-visual input to the verbal semantic system involved in naming. Coslett and Saffran (1989, 1992) described distinct left and right hemisphere semantic systems, whereby left hemisphere semantics supports naming but is disconnected from visual input, with right hemisphere semantics supporting non-verbal responses to visual input. Hillis and Caramazza (1995) proposed impairment in access to a unitary semantics system located in the left hemisphere—an impaired connection between visual information in the right hemisphere and language output in the left hemisphere (Hillis, 2007).

This case report describes a patient admitted to hospital with an acute left PCA territory infarct who demonstrated atypical impairments specific to tasks of expressive language involving visual input (i.e., verbal or written object/picture naming and picture description) compared to tasks involving other modalities (i.e., tactile or auditory input) or based in spontaneous expression. The objectives of this case report were (a) to provide illustrative language and cognitive assessment results for this patient within the 3-week acute post-stroke period and (b) to employ a particular model of semantics to propose a unified explanation for his impairment profile.

Method

Clinical Presentation

JDM, a 66-year-old right-hand dominant male, was admitted to the acute stroke ward after presenting to the emergency room with acute confusion upon waking and calling his son. Upon arrival in the emergency room, he was documented to have expressive aphasia, right-sided homonymous hemianopsia, right hemifacial weakness, right-sided hemiparesis, and bilateral upper extremity dysmetria. Past medical history included coronary artery disease with two prior myocardial infarctions, hypertension, hyperlipidemia, and type II diabetes mellitus. He wore reading glasses. He and his daughter denied any history of communication or cognitive impairment. He reported a Grade 12 education and being retired from a career as a network specialist. JDM reported being generally confused and noticing difficulties with his memory following the stroke.

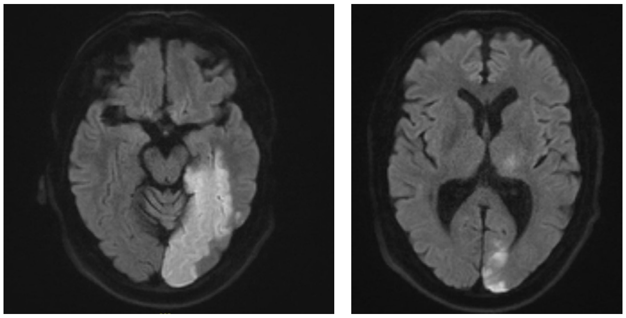
Medical Intervention and Imaging

JDM presented outside the therapeutic window for intravenous thrombolysis, and fortunately experienced early spontaneous improvement of his right-sided hemiparesis and conversational expressive language. The initial computed tomography scan of the head showed multifocal infarcts suspicious for cardioembolism, which was later confirmed by echocardiogram demonstrating multiple intracardiac thrombi. Computed tomography angiogram showed no evidence of proximal large vessel occlusion, and he was thus not a candidate for endovascular treatment. Acuity of the left PCA territory cortical and subcortical infarcts was confirmed with magnetic resonance diffusion-weighted sequences of the brain (see **Figure 1A** and **Figure 1B**). There was no obvious lesion of the corpus callosum on these early images. However, detailed structural magnetic resonance imaging was not performed and thus it is difficult to rule out corpus callosum involvement as the large medial occipital lesion extended near the splenium. A follow-up computed tomography scan on Day 3 post-stroke demonstrated medium to large territory cortical lesions of his left posteromedial temporal and left medial occipital lobes as well as deeper infarcts of the left thalamus (see **Figure 2A** and **Figure 2B**).

Neurological Examination

JDM was first examined by the physiatry consultants (JK, JY) on Day 9 post-stroke. His neurological examination

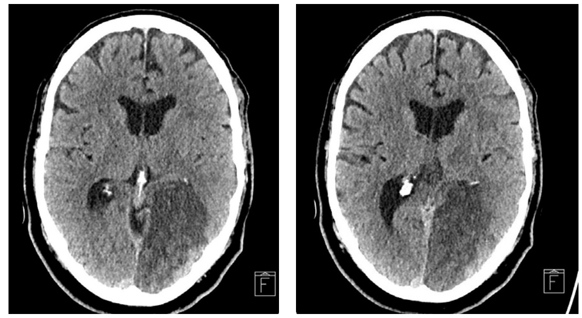
Figure 1



A B

Magnetic resonance diffusion-weighted sequences of the brain at the time of stroke presentation. Panel A: Restricted diffusion in the medial aspect of the left occipital lobe, extending into the temporal lobe, consistent with recent left PCA territory ischemic infarct. Panel B: Restricted diffusion of the medial occipital lobe and subtle/ill-defined restricted diffusion in the left thalamus.

Figure 2



A B

Computed tomography imaging of the brain at 3 days post-stroke. Panel A: Extensive infarction of the left occipital lobe extending into the temporal lobe. Panel B: Extensive infarction of the left occipital lobe and subtle hypointensity of the left thalamus.

was significant for a persistent right-sided homonymous hemianopsia, altered sensation to his right side, resolution of prior motor deficits with normal motor power and tone, and mild residual dysmetria of the right arm and leg. On bedside visuospatial testing, he was able to produce intact and complete drawings of a clock and house on verbal command, as well as identify the midpoint of horizontal lines. He was noted to move his head to various angles throughout reading and writing tasks. Praxis appeared intact as he was able to demonstrate brushing his teeth, waving goodbye, and swinging a baseball bat, and was able to follow motor and coordination testing without difficulty. No obvious oral motor or verbal apraxia was noted. Most striking during his bedside clinical examination were features of difficulty with colour and object naming. He often responded with "brown" for colour naming and, while he could not name objects presented visually, was able to provide a clear description of function.

Language and Cognitive Assessment Procedures

A registered speech-language pathologist (CI) administered and scored multiple language and cognitive assessment tasks. The bedside screening version of the Western Aphasia Battery–Revised (WAB-R; Kertesz, 2007) was administered on Day 3 post-stroke, and the full WAB-R (Kertesz, 2007) was administered on Day 14 post-stroke as a more comprehensive battery to evaluate language function and to determine the presence, severity, and type of aphasia. For the object naming subtest, he was provided visual input only (not allowed to touch the object). For his unsuccessful responses he was asked to describe the object and pantomime its use (objects that lent themselves to pantomime). As a further extension of the object naming subtest, JDM was asked to name 17 of the original 20 objects with tactile input only (three objects did not lend themselves to tactile naming). Specifically, after an approximately 30-minute interval post-visual naming during which he completed other non-naming related assessment tasks, he was asked to close his eyes, then permitted to manipulate the objects with one or both hands and to identify them. The same protocol was repeated in the opposite order—tactile naming followed by visual naming after a filled interval—on Day 17 post-stroke.

The Cognitive Linguistic Quick Test–Plus (CLQT+; Helm-Estabrooks, 2017) was administered on Day 17 post-stroke to assess cognitive domains of attention, memory (episodic, procedural, and working memory; both visual and verbal memory), executive functions (e.g., planning, mental flexibility, inhibition, and self-monitoring), language, and visuospatial skills. The tasks involved in this assessment (and the domains they assessed) included answering questions related to personal facts (language, episodic memory), symbol cancellation (visual attention, perceptual skills), confrontation naming (language), story retelling (attention, verbal working memory, language), symbol trails (attention, executive functions, visual perception), generative naming (working memory, language, executive functions), design memory (visual attention, visual memory, visuospatial skills), mazes and design generation (each addressing attention, executive functions, visuospatial skills), and clock drawing (addressing all five domains). Additional verbal and written expression assessment tasks, subtests of the Boston Diagnostic Aphasia Examination—Third Edition (Goodglass et al., 2001) and informal writing tasks, were administered on Days 14 and 17 post-stroke.

Rehabilitative Intervention

On Day 21 post-stroke and shortly after the completion of the above assessments, JDM was transferred to a tertiary inpatient rehabilitation program at another facility to continue with intensive speech therapy and other rehabilitation.

Ethics and Participant Consent

Individual case reports fall outside the scope of our institutional research ethics board and are exempt from formal ethics board review. The objectives of publication and potential audience of this case report were discussed verbally with the patient and the written consent form was read to him. He provided verbal and written consent to share details of his clinical presentation and investigations.

Results

Results of the language and cognitive testing performed in acute care are detailed below. JDM achieved an Aphasia Quotient of 81/100 on the full WAB-R, with a classification of mild anomic aphasia (see **Table 1**). These results were very similar to his performance on the bedside screening version of the WAB-R administered earlier in his admission. Improvement was noted in the spacing of his written output and in the helpfulness of phonemic cueing. JDM demonstrated intact repetition and strong auditory comprehension, including in conversation. Difficulty was noted with word fluency/generative naming tasks on the WAB-R and CLQT+ (WAB-R word fluency score: 5, maximum possible score: 20; CLQT+ generative naming score: 2, cut score: 5). Expressive language tasks based on auditory input, such as responsive naming and sentence completion, were relative strengths (see **Table 1** and **Table 2**).

JDM demonstrated fluent expressive language in conversation, characterized by occasional hesitations and infrequent word-finding difficulty. Fluency declined in a picture description task, with increased, unresolved instances of word-finding difficulty, typically for nouns (e.g., for "kite," "sand," and "sailboat;" see **Table 1**). JDM's score on visual object naming reflects correct responses for 3/20 (15%) objects without cueing (see **Table 1**). For his unsuccessful responses, he was able to describe the object with fluent verbal output (function, characteristics, etc.) on 13/15 (87%) attempts and pantomime the use of the object on 8/9 (89%) attempts. JDM scored 9/17 (53%) for tactile naming. When repeated on Day 17 in the opposite order, his scores were nearly identical: 4/20 (20%) for visual input and 9/17 (53%) for tactile input. His naming errors were often perseverations on the label "pencil," including before the pencil stimulus was presented and across both testing dates.

JDM was noted to have difficulty naming colours. While he was able to point to 6/6 colours without hesitation during the WAB-R auditory word recognition subtest, he had difficulty naming colours not only on visual presentation (1/3; stating an incorrect colour), but also during sentence completion and responsive naming tasks.

During attempts at oral reading, JDM employed an oral letter-by-letter reading approach which was, unfortunately, typically unsuccessful (e.g., "l-e-n-g-i-l" for "pencil"). This finding contrasted with being able to point to 6/6 letters presented in an auditory letter discrimination task and to copy written words (see **Table 1**). The strategy of tracing the letters of a word (kinesthetic input) was determined by JDM's occupational therapist to increase his success with naming graphemes. However, when he was successful in spelling the word aloud, he often mispronounced it (e.g., "racy" after correctly spelling "r-a-i-s-e"). When relying only on the auditory pathway, he could name orally spelled words (one similar error, whereby he stated "nosey" for "n-o-s-e") and orally spell given words with a high level of accuracy (see **Table 1**). Similar to his difficulties with verbal expression, JDM demonstrated disproportionate difficulty with tasks of written expression involving visual input (i.e., written object/picture naming and written picture description) in comparison to auditory input (i.e., writing to dictation) and spontaneous written expression (see **Table 1** and **Table 2** for details).

Results from the CLQT+ (see **Table 3** and **Table 4**) indicated that JDM's attention, executive functions, and visuospatial skills were mildly impaired. His memory was found to be moderately impaired and language skills severely impaired. The language domain's score was influenced by his poor performance on the confrontation naming subtest, which was based on visual input. JDM's composite severity rating corresponded to moderate cognitive impairment (on the cusp of mild impairment). Overall, his language function was more impaired than his non-linguistic cognition.

With respect to visuospatial function, he earned full points on a complex symbol trails task involving drawing lines to connect circles and triangles in an alternating fashion, and in order of increasing size. He also demonstrated strong performance on a design memory task (5/6 correct, cut score: 5) requiring recall and

Table 1

Western Aphasia Battery–Revised (WAB-R)

WAB-R subtest	Score
Spontaneous speech	17/20
Information content	8/10
Fluency, grammatical competence, and paraphasias	9/10
Auditory verbal comprehension	190/200
Yes/No questions	60/60
Auditory word recognition	54/60
Sequential commands	76/80
Repetition	100/100
Naming and word finding	41/100
Object naming	17/60[a]
Word fluency	5/20
Sentence completion	9/10
Responsive speech	10/10
Aphasia quotient	81/100[b]
Aphasia classification	Anomic (mild)
Reading	- /100[c]
Written word-Object choice matching	0/6
Letter discrimination	6/6
Spelled word recognition	5/6
Spelling	5/6
Writing	75/100
Writing upon request (name, address)	6/6
Writing output (written picture description)	17/34[d]
Writing to dictation (complex sentence)	8.5/10
Writing dictated words	10/10
Alphabet and numbers	22.5/22.5
Dictated letters and numbers	7.5/7.5
Copying a sentence	3.5/10[e]

[a] Score reflects correct responses for 3/20 objects without cueing. Task repeated on Day 17 post-stroke (see results in body of text). [b] Aphasia quotient severity: > 76 (mild), 51–75 (moderate), 26–50 (severe), 0–25 (very severe). [c] Discontinued attempts at additional subtests due to severity of deficits (i.e., unable to complete tasks at the single word level). Unable to calculate total score. [d] Writing Output sample: *Printed in all caps. Errors underlined.* "The boy is walking with his top (*dog*) while (*expressed word-finding difficulty related to the kite*). The man is fishing ad has cau (*followed by two improperly formed letters*) a hish. The man and womarn areing an afternoon." [e] JDM lost his place halfway through the sentence but demonstrated accurate output up until that point.

identification of two intricate target designs from a set of six over three trials. He verbalized difficulty on the more complex of two maze tasks, stating "it would be helpful if I could see the whole page at the same time" (seemingly describing his visual field cut); he was nearly successful but exceeded the time limit.

Table 2

Additional Verbal and Written Expression Assessment Tasks

Subtest/task	Score
Boston Diagnostic Aphasia Examination–Third Edition	
Responsive naming	8/10[a]
Writing - Basic encoding skills – Dictated words	
Primer word vocabulary	4/4
Regular phonics	2/2
Common irregular forms	3/5[b]
Informal assessment tasks	
Written object naming	0/3
Written picture naming	2/5
Spontaneous written narrative	N/A[c]

[a] Modified scoring. Not based on time taken to respond, as in protocol. [b] 60th percentile. Unable to make comparisons to normative data for remaining assessment tasks due to modified scoring, use of short-form versions, or informal nature of task. [c] Spontaneous written narrative sample: *Mixture of capital and lower-case letters throughout. Errors underlined.* "Today I was doing tord exercises and had to stop for awhile. We continued later on in the afternoon with some confusion."

Table 3

Cognitive Linguistic Quick Test–Plus (CLQT+) Cognitive Domains

Cognitive domain	Score	Severity	Within normal limits (Age 18–69 years)
Attention	143	Mild	180–215
Memory	125	Moderate	155–185
Executive functions	23	Mild	24–40
Language	17	Severe	29–37
Visuospatial skills	75	Mild	82–105
Composite severity rating	2.4	Moderate	3.5–4.0

Table 4

Cognitive Linguistic Quick Test–Plus (CLQT+) Indices

Index	Score	Severity	Within normal limits (Age 18–69 years)
Non-linguistic cognition	34	Mild	39–49
Linguistic/Aphasia	34	Moderate	N/A
Clock drawing	8	Moderate	12–13

Discussion

JDM's case represents a relatively uncommon presentation of visual input-specific language impairments following a left PCA territory infarct. Such infarcts commonly result in right-sided homonymous hemianopsia, alexia without agraphia, colour agnosia or colour anomia and, less frequently, visual agnosia (De Renzi et al., 1987; Kim, 2016). However, JDM's case differed with his additional specific impairments in tasks of expressive language involving visual input compared to those involving other modalities or based in spontaneous expression.

Visual agnosia typically occurs with extensive occipital lobe damage and involves impairment in visual recognition of objects (Kim, 2016). On a cognitive processing level, information transmission for visual object/picture naming involves (a) access to a structural description (knowledge about the visual form of the object/picture), (b) access to a semantic representation (functional and associative characteristics of the object/picture), and (c) access to the phonological representation (object/picture name). This processing occurs in a cascade rather than serial stages and with top-down as well as bottom-up processes. Visual aperceptive agnosia involves impaired access to a structural description, while visual associative agnosia involves inability to access a semantic representation, as evidenced by lack of recognition. JDM's demonstrated recognition of the visually presented objects that he was unable to name indicates that a visual aperceptive or associative agnosia was unlikely at play. Rather, his performance was more consistent with partially impaired access to semantics from an intact structural description, which has been proposed to underlie optic aphasia (Hillis, 2007; Humphreys et al., 1997). Indeed, JDM's combination of right-sided homonymous hemianopsia, alexia without agraphia, and impaired colour naming, alongside impairment in tasks of expressive language involving visual input in the absence of agnosia is in keeping with previous descriptions of optic aphasia (Campbell & Manning, 1996; Coslett & Saffran, 1992; Ferreira et al., 1997; Hillis & Caramazza, 1995; Kwon & Lee, 2006; Marsh & Hillis, 2005; Rodrigues et al., 2008).

As described earlier, different models of semantics have been used to explain the selective deficit in naming from a visual input modality (Beauvois, 1982; Coslett & Saffran, 1989, 1992; Hillis & Caramazza, 1995). Hillis and Caramazza (1995) suggested a unitary semantics system located in the left hemisphere, with transmission of visual information being processed in the right hemisphere to the language centers in the left hemisphere becoming impaired due to the damaged splenium. While sufficient for producing pantomime and a description of the object, the level of activation of the semantic representation may not be precise enough to generate a specific name.

JDM's performance on the semantic comprehension task of the CLQT+ (8/10 correct responses) may offer evidence of incomplete access to semantic representations from visual input. While cut scores have not been established for this task, the test authors indicate that they would be at or near perfect performance (Helm-Estabrooks, 2017). This task required him to identify the correct picture from a page of 10 options in response to a simple verbal description of function or category. The observed errors, such as pointing to the cow for "lays eggs," would not be expected with complete access to semantic information. It is also possible that his errors resulted from impaired access to the structural description/visual form of the pictures, but his performance on other assessments did not suggest this degree of visuoperceptual impairment. Adding to the theory of incomplete access to semantics was JDM's very strong but imperfect score for describing an object or pantomiming its use. He also expressed confusion at times when a target name was supplied even in the context of his own accurate description and/or pantomime but would typically eventually express agreement.

Another proposal has been made that optic aphasia and visual associative agnosia exist along a continuum of deficits in access to semantic information, with some cases suggesting evolution of visual associative agnosia into optic aphasia with recovery (De Renzi & Saetti, 1997; De Renzi et al., 1987; Rubens, 1979). Although an isolated incident, JDM did demonstrate an apparent lack of recognition for one object during screening on Day 3 post-stroke, as evidenced by a completely incorrect description of its function.

Hillis and Caramazza's (1995) model provides a plausible, unified explanation for JDM's overall impairment profile, as it is also commonly proposed for the other impairments typical of left PCA territory infarcts: pure alexia and compensatory use of letter-by-letter reading (De Renzi et al., 1987; Hillis, 2007; Kim, 2016) and colour anomia (De Renzi et al., 1987). Consistent with this model, while detailed structural magnetic resonance imaging was not performed, JDM's impairment profile would support involvement of the splenium.

As part of JDM's pure alexia, he employed a letter-by-letter approach to reading but demonstrated impaired letter naming. Similar findings have been reported in cases of optic aphasia (Ferreira et al., 1997; Hillis & Caramazza, 1995). A kinesthetic strategy was noted to be beneficial and has been shown, alone or in combination with tactile input, to be an effective intervention for such patients (Nitzberg Lott et al., 2010). However, JDM was still noted to make some pronunciation errors after using this technique to correctly spell the word aloud. Despite these initial difficulties, a kinesthetic strategy was deemed a promising future therapy approach for JDM.

Cognitive impairment is also common following a left PCA territory infarct, and varies depending on the location and extent of the lesion (Kim, 2016; Ng et al., 2005). Ng et al. (2005) showed that cognitive functional independence measure scores were lower in patients with PCA infarction who presented with confusion or agitation, similar to this case. Exact anatomic localization of cognitive impairment in PCA stroke is difficult, with potential involvement of the medial temporal lobe, thalamus, hippocampal or parahippocampal region, and splenium all having been described (De Renzi et al., 1987; Kim, 2016; Ng et al., 2005; Park et al., 2009). JDM suffered a large area of infarction involving multiple structures, and thus the cognitive impairments noted are not surprising. Specific to the CLQT+, he performed below the cut score for a verbal working memory task (story retelling score: 4, cut score: 6), but met the cut score for a visual memory task (design memory task, as mentioned earlier). JDM's noted difficulty with generative naming was in contrast to his strong performance on other tasks of expressive language not involving visual input (e.g., sentence completion, conversation). These results likely reflect JDM's cognitive impairments, given the greater dependence on executive function (i.e., productive, flexible thinking and a disciplined linguistic search strategy) and memory in generative naming tasks (Helm-Estabrooks, 2017). More detailed discussion of the findings of cognitive impairment are beyond the scope of this paper.

Visuospatial deficits are commonly due to lesions of the non-dominant hemisphere; most commonly the right parietal lobe (Mesulam, 1981), which was not involved on imaging in this case. JDM performed well on bedside visuospatial tasks including line orientation/visual judgement and clock drawing, as well as subtests of the CLQT+, with no evidence of spatial disorganization or hemispace neglect. Formal scoring of his clock draw on the CLQT+ was discrepant with bedside testing, however, with moderate impairment noted. The cause of this discrepancy is not clear, though his errors did not indicate visuospatial impairment and may have been impacted by fatigue.

Plaut (2002) provided a summary table of 14 case reports involving patients with similar impairment profiles as presented herein. A wide range of severity is apparent across cases within a given area of assessment (e.g., 0–73% accuracy for visual object naming, 35–100% for tactile naming, 30–100% for gesturing to visual input). However, the same pattern of inferior performance in visual object naming compared to tactile naming, gesturing to visual input, and naming to spoken definition is preserved within each case. Our results fall on the more impaired end of the spectrum for visual object naming (15–20%) and on the less impaired end of the spectrum for gesturing to visual input (89%). As with most of these previous reports, JDM's tactile naming (53%) was not without impairment, and he demonstrated other features of language impairment in tasks not involving the visual input modality. That is, he demonstrated a mild overall language impairment in the background of his specific visual input modality impairments.

More in-depth assessment would have been valuable in testing theories regarding JDM's performance and more conclusively ruling out a visual aperceptive or associative agnosia. Specifically, further assessment of his visuoperceptual abilities and of each of the processing components involved in object/picture naming using a test such as the Birmingham Object Recognition Battery (Riddoch & Humphreys, 1993) would have been informative. JDM's ability to answer probing questions about an object/picture and to sort them by function or category would have further assessed for incomplete access to semantic representations from visual input. Unfortunately, further assessment was not possible within the short time frame that the patient was admitted to acute care. Follow-up testing would have been valuable to document the trajectory of JDM's impairment profile and his responsiveness to intervention, but this rehabilitation took place following transfer to another facility.

This case report is unique in its description of the overall language impairment profile as well as cognitive assessment results and in its presentation of a patient in the acute post-stroke period. We have proposed how a particular model of semantics may provide a unified explanation for this patient's presentation. We suggest that future case reports with similar patients expand on the current approach by including assessments such as written naming to tactile input, as well as those mentioned earlier. Together, these case reports may aid clinicians in recognizing similar atypical impairment profiles, thereby avoiding misidentification as a classic aphasia syndrome. Accurate diagnosis is essential for determining appropriate rehabilitation interventions.

References

Beauvois, M.-F. (1982). Optic aphasia: A process of interaction between vision and language. *Philosophical Transactions of the Royal Society B, 289*(1089), 35–47. https://doi.org/10.1098/rstb.1982.0070

Campbell, R., & Manning, L. (1996). Optic aphasia: A case with spared action naming and associated disorders. *Brain & Language, 53*(2), 183–221. http://doi.org/10.1006/brln.1996.0044

Coslett, H. B., & Saffran, E. M. (1989). Preserved object recognition and reading comprehension in optic aphasia. *Brain, 112*(4), 1091–1110. https://doi.org/10.1093/brain/112.4.1091

Coslett, H. B., & Saffran, E. M. (1992). Optic aphasia and the right hemisphere: A replication and extension. *Brain & Language, 43*(1), 148–161. https://doi.org/10.1016/0093-934X(92)90026-B

De Renzi, E., & Saetti, M. C. (1997). Associative agnosia and optic aphasia: Qualitative or quantitative difference? *Cortex, 33*(1), 115–130. https://doi.org/10.1016/S0010-9452(97)80008-1

De Renzi, E., Zambolin, A., & Crisi, G. (1987). The pattern of neuropsychological impairment associated with left posterior cerebral artery infarcts. *Brain, 110*(5), 1099–1116. https://doi.org/10.1093/brain/110.5.1099

Ferreira, C. T., Giusiano, B., Ceccaldi, M., & Poncet, M. (1997). Optic aphasia: Evidence of the contribution of different neural systems to object and action naming. *Cortex, 33*(3), 499–513. http://doi.org/10.1016/s0010-9452(08)70232-6

Freund, C. S. (1889). Ueber optische aphasie und seelenblindheit. *Archiv für Psychiatrie und Nervenkrankheiten, 20*, 371–416. https://doi.org/10.1007/BF02086691

Goodglass, H., Kaplan, E., & Barresi, B. (2001). *Boston Diagnostic Aphasia Examination* (3rd ed.). Lippincott Williams & Wilkins.

Helm-Estabrooks, N. (2017). *Cognitive Linguistic Quick Test–Plus (CLQT+)*. Psychological Corporation.

Hillis, A. E. (2007). Aphasia: Progress in the last quarter of a century. *Neurology, 69*(2), 200–213. https://doi.org/10.1212/01.wnl.0000265600.69385.6f

Hillis, A. E., & Caramazza, A. (1995). Cognitive and neural mechanisms underlying visual and semantic processing: Implications from "optic aphasia." *Journal of Cognitive Neuroscience, 7*(4), 457–478. https://doi.org/10.1162/jocn.1995.7.4.457

Humphreys, G. W., Riddoch, M. J., & Price, C. J. (1997). Top-down processes in object identification: Evidence from experimental psychology, neuropsychology and functional anatomy. *Philosophical Transactions: Biological Sciences, 352*(1358), 1275–1282. https://doi.org/10.1098/rstb.1997.0110

Kertesz, A. (2007). *Western Aphasia Battery–Revised*. Psychological Corporation.

Kim, J. S. (2016). Posterior cerebral artery disease. In J. C. Grotta, G. W. Albers, J. P. Broderick, S. E. Kasner, E. H. Lo, A. D. Mendelow, R. L. Sacco, & L. K. S. Wong (Eds.), *Stroke: Pathophysiology, diagnosis, and management* (6th ed., pp. 393–412. e5). Elsevier Health Sciences. https://doi.org/10.1016/C2012-0-02756-1

Kwon, M., & Lee, J.-H. (2006). Optic aphasia: A case study. *Journal of Clinical Neurology, 2*(4), 258–261. https://doi.org/10.3988/jcn.2006.2.4.258

Lhermitte, F., & Beauvois, M. F. (1973). A visual-speech disconnexion syndrome: Report of a case with optic aphasia, agnosic alexia and colour agnosia. *Brain, 96*(4), 695–714. https://doi.org/10.1093/brain/96.4.695

Marsh, E. B., & Hillis, A. E. (2005). Cognitive and neural mechanisms underlying reading and naming: Evidence from letter-by-letter reading and optic aphasia. *Neurocase, 11*(5), 325–337. https://doi.org/10.1080/13554790591006320

Mesulam, M.-M. (1981). A cortical network for directed attention and unilateral neglect. *Annals of Neurology, 10*(4), 309–325. https://doi.org/10.1002/ana.410100402

Ng, Y. S., Stein, J., Salles, S. S., & Black-Schaffer, R. M. (2005). Clinical characteristics and rehabilitation outcomes of patients with posterior cerebral artery stroke. *Archives of Physical Medicine and Rehabilitation, 86*(11), 2138–2143. https://doi.org/10.1016/j.apmr.2005.07.289

Nitzberg Lott, S., Syms Carney, A., Glezer, L. S., & Friedman, R. B. (2010). Overt use of a tactile/kinaesthetic strategy shifts to covert processing in rehabilitation of letter-by-letter reading. *Aphasiology, 24*(11), 1424–1442. https://doi.org/10.1080/02687030903580333

Park, K.-C., Yoon, S.-S., & Seo, K.-H. (2009). Splenium or parahippocampus involvement and its relationship to cognitive decline in posterior cerebral artery infarction. *Journal of Clinical Neuroscience, 16*(7), 914–917. https://doi.org/10.1016/j.jocn.2008.09.010

Plaut, D. C. (2002). Graded modality-specific specialisation in semantics: A computational account of optic aphasia. *Cognitive Neuropsychology, 19*(7), 603–639. https://doi.org/10.1080/02643290244000112

Riddoch, J. M., & Humphreys, G. W. (1993). *Birmingham Object Recognition Battery* (BORB). Psychology Press.

Rodrigues, M. A., Adda, C. C., de Souza Lucia, M. C., Scaff, M., & Miotto, E. C. (2008). Cognitive deficits associated with optic aphasia: *Neuropsychological contribution to a differential diagnosis. Dementia & Neuropsychologia, 2*(2), 151–154. https://doi.org/10.1590/S1980-57642009DN20200013

Rubens, A. B. (1979). Agnosia. In K. M. Heilman & E. Valenstein (Eds.), *Clinical neuropsychology* (1st ed., pp. 233–267). Oxford University Press.

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