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# Journal of the Neurological Sciences

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## Short communication

# Visual text hallucinations of thoughts in an alexic woman

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### ARTICLE INFO

Article history: Received 12 August 2013 Received in revised form 15 January 2014 Accepted 27 January 2014 Available online 1 February 2014

Keywords:
Dementia with Lewy bodies
Visual hallucination
Alzheimer's disease
Posterior cortical atrophy
Alexia
Agraphia

#### ABSTRACT

In this report we describe a patient with a clinical diagnosis of dementia with Lewy bodies, who had hallucinations of reading her thoughts in the air although she was alexic and unable to read. She also had severe visuoperceptual deficits and closing-in phenomenon. SPECT imaging demonstrated hypoperfusion of the left parieto-occipital cortices together with hyperperfusion of the left orbitofronto-temporal areas. Her visual text hallucinations may represent another type of textual hallucinations related to syntacto-semantic network hyperactivation, which is implicated in auditory hallucinations in schizophrenia.

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# 1. Introduction

Although auditory hallucinations are most common in schizophrenia [1], visual hallucinations still have a high lifetime prevalence in this disorder [2], and are also observed in patients with other disorders such as Parkinson's disease [3]. Moreover, the visual hallucinations of patients with organic neuropsychiatric conditions, including dementia with Lewy bodies (DLB), are often characterized by vivid and colorful images of persons or animals [4]. Here we report a patient with a clinical diagnosis of DLB who had hallucinations of reading her thoughts in the air although she was alexic and unable to read. Interestingly, ffytche et al. [5] have already reported similar textual visual hallucinations in a patient with pure alexia following bilateral occipito-temporal infarction; however, to our knowledge, this is the first report demonstrating visual text hallucinations in degenerative dementias, including Alzheimer's disease and DLB.

### 2. Case report

A 72-year-old housewife began to experience forgetfulness at age 71. Six months later, her husband noticed that she had difficultly reading and writing. Alzheimer's disease was diagnosed by a local neurosurgeon and treatment with donepezil 5 mg per day was initiated. Ten months after onset of symptoms, she began to experience visual

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hallucinations as well as delusions of jealousy, and as a result, became increasingly aggressive towards her husband. Therefore, she was referred to our memory disorder clinic at 12 months from the onset of symptoms.

At the initial visit, she described characteristic visual text hallucinations. She said, "I can see my thoughts as sentences in front of me. For example, when I thought about a conversation with my friend, I could see what I had said to her as sentences. I can read those sentences just as I read movie subtitles...." Her husband witnessed her tracing with her finger in the air, on the wall, or on a desk in silence. Furthermore, she occasionally did the same thing while talking as if she was reading aloud the text hallucinations in front of her. Therefore, when she hallucinates, she first experiences the text hallucinations of her thoughts and then reads aloud these text hallucinations in front of her. Her visual hallucinations consisted of both hiragana (phonograms) and kanji (Chinese characters). In contrast, she has never experienced any vivid and colorful hallucinations of persons/animals. Her vision was normal, and visual fields were intact. Parkinsonism was not detected. Electroencephalography showed diffuse slow waves without epileptiform abnormalities. Her past medical history was noncontributory except for hypertension, and her family history was negative.

On examination, her speech and verbal comprehension were normal, but recent memory was impaired. She scored 17/30 on the Mini-Mental State Examination (MMSE). Day-to-day fluctuations of cognitive function were noted. General visual perception was tested using the Visual Perception Test for Agnosia (VPTA) [6], which is a test standardized in Japan, designed to assess higher order visual perceptual abilities. She was impaired in simple visuoperceptual tasks including length and size discrimination, form discrimination, and visual counting tasks. There was also impairment in higher-order complex visuoperceptual

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tasks such as overlapping figure identification. The closing-in phenomenon, which is a tendency to draw near or on the target while copying figures, was frequently observed. In addition, she showed deficits in visual space perception tasks including line bisection, line cancelation, clock drawing, and drawing a man's face. Achromatopsia, prosopagnosia, and associative visual agnosia were absent.

Moreover, she had severe impairments in reading aloud and reading comprehension. Language was evaluated by the Standardized Language Test for Aphasia (SLTA) [7], which is most frequently used for individuals with aphasia in Japan. She was unable to read aloud in either hiragana or kanji. In the reading aloud task, she scored 0/10 points (0.0%) on both hiragana and kanji letters. Writing was relatively preserved for hiragana, but was impaired for kanji.

Brain magnetic resonance imaging (MRI) demonstrated mild atrophy of the bilateral dorsolateral prefrontal cortices and the left parieto-occipital lobes. Hippocampal atrophy was minimal. MRI also showed small white matter lesions in bilateral frontal and temporal lobes, which were not considered to be a cause of dementia. In addition, she underwent a single photon emission tomography (SPECT) scan

with 99mTc-ECD and did not experience any visual text hallucinations during the scan. SPECT and the easy z-score system (eZIS) [8] demonstrated extensive reduction of regional cerebral blood flow (rCBF) in the bilateral parietal and occipital lobes, being more prominent on the left (Fig. 1). In contrast, there was increased perfusion of the orbitofrontal regions and anterior temporal lobes. Quetiapine 25 mg per day was added to donepezil. After the dose of quetiapine was gradually increased up to 75 mg per day, her visual hallucinations and aggressive behavior improved significantly without extrapyramidal symptoms.

#### 3. Discussion

The age of onset and subsequent clinical course of this patient excluded the possibility of alternative diagnoses such as late-onset schizophrenia. Similarly, visual hallucinations often occur in delirium, however, the fact that her hallucinations occurred during clear conscious states and were remembered in detail, argue against this diagnosis. The patient also presented with some clinical features consistent with posterior cortical atrophy, a clinical syndrome characterized

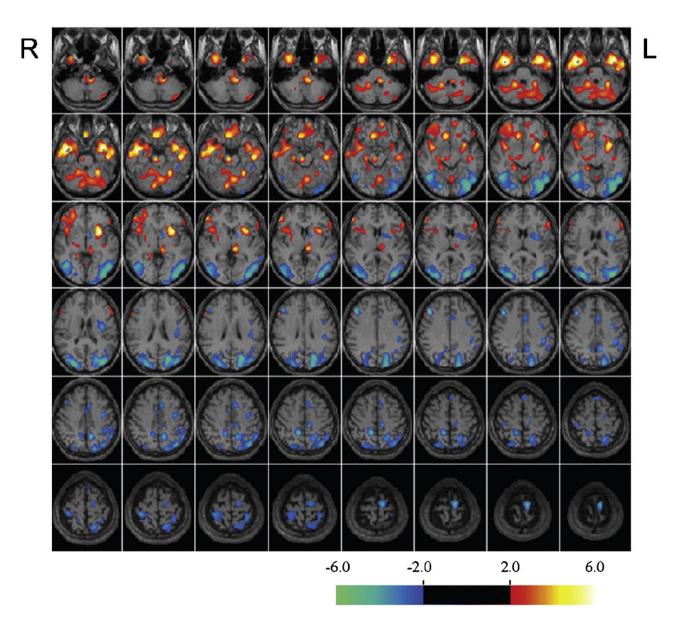


Fig. 1. Single photon emission tomography images analyzed with the easy z-score imaging system (eZIS) [8]. Extensive reduction of rCBF is seen in the parietal and occipital lobes bilaterally, being more prominent on the left side. In contrast, there is increased perfusion of the orbitofrontal cortices and the anterior temporal lobes. The z-score maps are overlaid on the surface projection of a spatially normalized MRI template. Colors represent the statistical significance (z-score) of the decrease or increase in rCBF.

by progressive complex visual dysfunction associated with atrophy of the occipital and occipito-parietal cortex and hypoperfusion in the same regions. However, since her memory impairment was clearly apparent prior to the onset of difficulties with reading and writing, and overall decline in her activities of daily living, we felt that degenerative dementias including DLB and Alzheimer's disease were more suitable for clinical diagnosis. Findings of severe visuoperceptual deficits [9], closing-in phenomenon [10], and cognitive fluctuations suggested a diagnosis of DLB rather than Alzheimer's disease. Furthermore, a previous study reported that patients with DLB have a much greater deficit in the reading of words included in the National Adult Reading Test (NART) compared to those with Alzheimer's disease, and suggested that severe visuoperceptual deficits in DLB may be related to their findings [11]. These findings were also consistent with the diagnosis of DLB in our patient. Significantly reduced rCBF in the occipital and parietal lobes further supported DLB. Although Parkinsonism was absent, she met at least two core clinical criteria which meet consensus criteria for the clinical diagnosis of 'probable DLB' [12].

Hallucinations are a key symptom of DLB, but the patient's visual text hallucinations were unusual. These symptoms occasionally occur in schizophrenia, but this is the first report to our knowledge of such hallucinations in DLB. Interestingly, the patient also had severe alexia and was unable to read. Accordingly, her "reading" of thoughts may have compensated for her alexia, ffytche et al. [5] reported similar textual visual hallucinations in a patient with pure alexia following bilateral occipito-temporal infarction. From a linguistic point of view, visual text hallucinations resemble auditory hallucinations of schizophrenia, the only difference being that they are read and not heard. Therefore, ffytche et al. posited that visual text hallucinations arise from the same cortical network dysfunction implicated in the auditory hallucinations of schizophrenia. The critical regions for auditory hallucinations are thought to include the lateral temporal cortex, inferolateral frontal lobe, and temporo-parietal cortex [13]. Moreover, there is some evidence that parts of the same network are activated in sentence reading and semantic processing tasks [14], implying that syntactic and semantic linguistic processes play a role in the generation of the auditory hallucinations. Because these brain regions were spared in their patient, ffytche et al. [5] speculated that the visual text hallucinations arose within visual subcompartments of the above-mentioned syntactosemantic network. Similar cognitive mechanisms could be postulated for visual text hallucinations of our patient. She presented with hallucinatory visual sentences because the cortical network subserving syntacto-semantics remained intact thereby activating similar brain regions responsible for auditory hallucinations of schizophrenia.

SPECT findings in our patient further support the notion that the syntacto-semantic network plays a crucial role in visual text hallucinations. Left parieto-occipital hypoperfusion has been well documented in previous cases of alexia [15]. Furthermore, our patient also showed increased rCBF in the left orbitofrontal and inferior frontal cortices and anterior temporal regions. Therefore, visual text hallucinations may represent an abnormal, compensatory cortical release phenomenon that arises from hypofunction of the left parieto-occipital cortices together with hyperfunction of the left orbitofronto-temporal areas.

Additionally, the notion of auditory-visual synesthesia, a form of synesthesia in which synesthetic perceptions (colors, textures, shapes and movements) are induced by the sounds of people's voices, may support the neural mechanism of our patient's experiences. A previous neuroimaging study performing a functional connectivity analysis found stronger connectivity of the left inferior parietal cortex (IPC) with both the primary auditory and primary visual cortices in auditory-visual synesthetes relative to controls, while there were no differences between synesthetes and controls in connectivity between the auditory cortex and visual areas [16]. These findings provide evidence for a disinhibited feedback mechanism in auditory-visual synesthesia, mediated by the

IPC as a sensory nexus area, rather than a direct linkage between the auditory and visual areas. In our case, the patient reads a presentation of words that she was thinking (inner speech) rather than what she was actually speaking (external speech). Therefore, we speculate that her text hallucinations may be induced by her inner rather than external speech, due to a disinhibited feedback mechanism similar to auditory-visual synesthesia. Furthermore, our SPECT findings of reduced rCBF in the left IPC may be consistent with this hypothesis.

In summary, we reported a patient with DLB who had visual text hallucinations although she was alexic. This case report provides additional evidence to support the hypothesis that the syntacto-semantic network plays a crucial role in visual text hallucinations.

### Consents

The patient gave informed consent for publication of this case report.

#### **Conflict of interests**

The authors have no conflicts of interest.

## Acknowledgments

The authors thank David Hong for reviewing the final version of the manuscript. This study was supported in part by a Grant-in-Aid from the Ministry of Education, Culture, Sports, Science and Technology to M.M. The patients described in this report gave their informed consent for their details to be published.

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