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# Asymmetric oculomotor apraxia, optic ataxia, and simultanagnosia with right hemispatial neglect from a predominantly left-sided lesion of the parieto-occipital area

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## ABSTRACT

**Introduction:** Bálint's syndrome involves bilateral damage to the parieto-occipital area. The extent of the effect of unilateral damage on the Bálint's triad (oculomotor apraxia, optic ataxia, and simultanagnosia) remains unknown.

**Methods:** We examined a 63-year-old, right-handed woman who developed right hemianopia, oculomotor apraxia, optic ataxia, simultanagnosia, and hemispatial neglect (HSN) for the right after a cerebral infarction, with detailed neuropsychological tests, magnetic resonance imaging, and single photon emission computed tomography (SPECT).

**Results:** Neuropsychological examination showed that oculomotor apraxia, optic ataxia, and simultanagnosia were more pronounced in the right hemi-space, probably due to the limited eye movement in the right visual field, whereas HSN was restricted to the right hemi-space. Diffusion-weighted MR images revealed hyperintensity in the left parieto-temporo-occipital region, and several spotty areas of the bilateral frontal and parietal subcortical regions. SPECT revealed hypoperfusion in the left parieto-occipital region and frontal operculum and small areas of the right superior parietal lobule.

**Conclusions:** The case suggests that asymmetric (more pronounced in the right hemi-space) oculomotor apraxia, optic ataxia, and simultanagnosia occur in an extensive lesion of the left parieto-occipital cortices. Although HSN is not a prerequisite for simultanagnosia, the coexistence of HSN aggravates simultanagnosia in the hemi-space opposite the lesion.

## ARTICLE HISTORY

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Trousseau's syndrome; dorsal  
simultanagnosia; optic ataxia

## Introduction

Bálint's syndrome is the triad of a spatial disorder of attention or simultanagnosia (inability to direct attention to more than one object due to constriction of the attentional visual field), psychic paralysis of gaze (difficulty shifting gaze voluntarily to an object; oculomotor apraxia) (Cogan & Adams, 1953), and optic ataxia (reaching disturbance under visual guidance), resulting from damage to the bilateral parieto-occipital region (Bálint, 1909).

Simultanagnosia of Bálint's syndrome is specifically called dorsal simultanagnosia, which can be differentiated from ventral simultanagnosia due to a left temporo-occipital lesion (Farah, 1990). Recently, we reported a patient with ventral simultanagnosia from a "right" temporal sulcus and angular gyrus lesion (Sakurai, Hamada, Tsugawa, & Sugimoto, 2016).

As described earlier, Bálint's syndrome involves bilateral damage to the parieto-occipital area. The extent of the effect of unilateral damage on the Bálint's triad remains unknown. Furthermore, Bálint's original case had a sign of left hemispatial neglect (HSN) (Rizzo & Vecera, 2002). Whether HSN affects simultanagnosia in Bálint's syndrome is also unknown. Some authors claim that HSN and Bálint's syndrome reflect different underlying deficits (Rizzo & Vecera, 2002; Robertson, 2003), whereas others argue that attention deficit in HSN is bilaterally represented in Bálint's syndrome (Farah, 1990).

Here, we report a patient with oculomotor apraxia, optic ataxia, simultanagnosia, and right HSN due to a predominantly left-sided lesion of the parieto-occipital area, and discuss whether or not a unilateral lesion can cause Bálint's triad, how HSN affects simultanagnosia, and also how dorsal simultanagnosia differs from ventral simultanagnosia.

## Materials and methods

### *Patient profile*

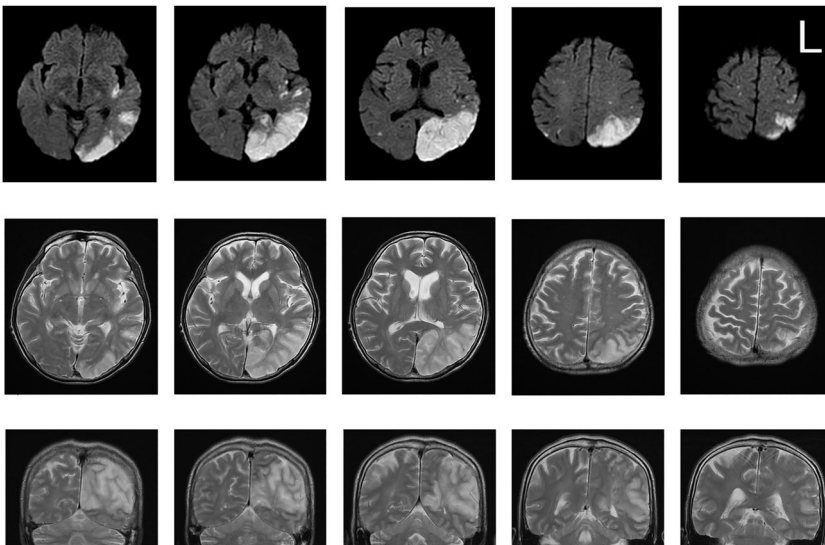
A naturally right-handed, 63-year-old woman who had graduated from senior high school felt general fatigue in January 2014 and was found to have difficulty communicating with her sister the next day. She had undergone total gastrectomy for adenocarcinoma eight years before, and was administered oral TS-1® (Tegafur 20 mg, Gimeracil 5.8 mg, and Oteracil potassium at 19.6 mg per capsule) for intrapelvic metastasis since October 2009. Metastatic pleural effusion and ascites were noted in July 2013. On admission, abdominal distention was noted. She was alert and spoke fluently, but could not understand what was said to her. Neurological examination three days after the disease onset showed right homonymous hemianopia on confrontation testing. When the patient was asked to fixate the examiner's index finger with both eyes in the centre of the visual field, she was aware of another moving finger or a penlight brought from the left periphery toward the centre, but not from the right periphery: when asked whether she could see anything moving or bright, she said, "I don't see it." In Goldmann perimetry, she did not detect a light because of her oculomotor apraxia and simultanagnosia, described later. Also, noted were fluent aphasia with pronounced alexia with agraphia (described later), Gerstmann's tetrad, verbal short-term memory impairment, and HSN for the right (she wrote characters on dictation on the left side of the paper). Auditory comprehension recovered (Yes–No question, e.g., "Will paper burn in fire?" and "Does a raccoon dog lay an egg?," scores: 9/10) so that she could follow the examiner's verbal commands. The sensorimotor function and coordination were intact. During hospitalisation, she left food uneaten on the right side of the tray. Laboratory tests showed Platelets  $13.6 \times 10^4/\mu\text{L}$ , PT-INR 1.40, D-dimer 52.0  $\mu\text{g}/\text{dL}$  (normal: 0.0–0.5), FDP 92  $\mu\text{g}/\text{mL}$  (normal: 0–4), and TAT 33.9  $\text{ng}/\text{mL}$  (normal: 0.0–3.0). Magnetic resonance imaging (MRI) performed three days after onset revealed hyperintensity in the left parieto-temporo-occipital region (cuneus, precuneus, lateral occipital, angular, posterior supramarginal and superior

and middle temporal gyri, and superior parietal lobule), insula, and several spotty areas of the bilateral frontal and parietal subcortical regions (Figure 1). Based on the history of gastric carcinoma, elevated plasma coagulation markers, and multiple cortical and subcortical infarction, we diagnosed her with Trousseau's syndrome.

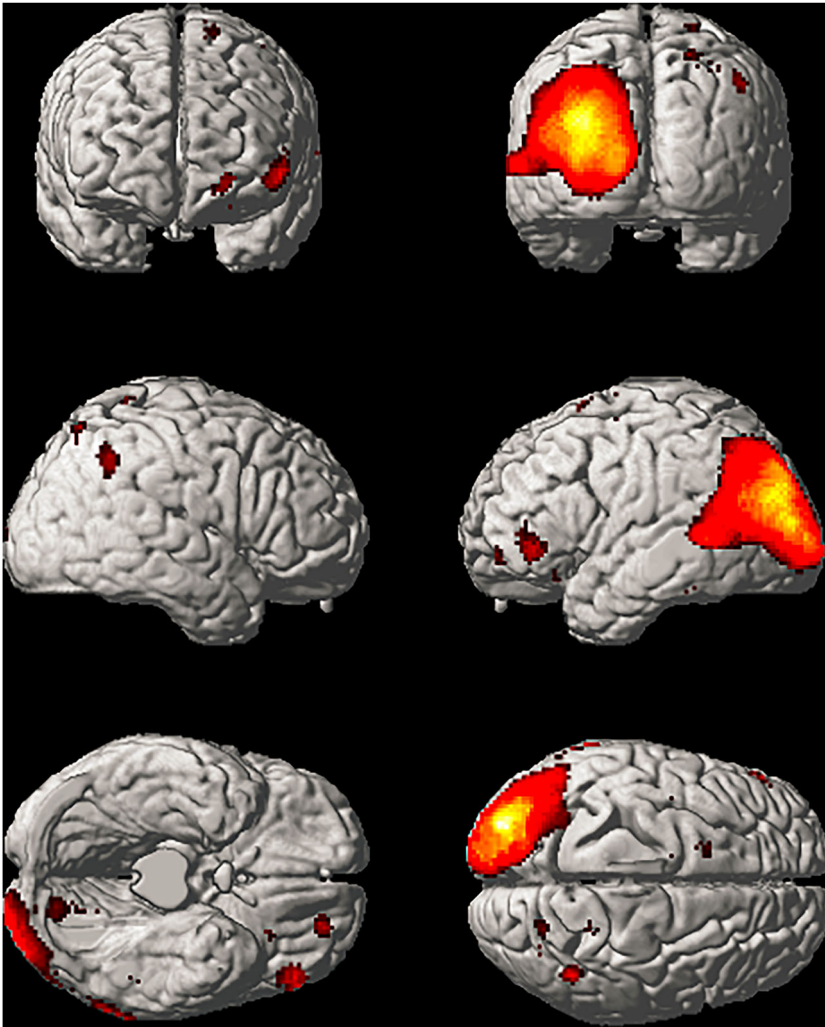
### Neuroimaging study

Single photon emission computed tomography with a  $^{99m}\text{Tc}$ -ethylcysteinate dimer ( $^{99m}\text{Tc}$ -ECD-SPECT) was performed two weeks after onset. Comparison with the normal subject database with the same generation and sex by the two sample *t*-test ( $n = 20$ , women, ages between 60 and 69 years old, uncorrected  $p < .001$ ) using an easy Z-score imaging system (eZIS) (Matsuda, Mizumura, Soma, & Takemura, 2004) and Statistical Parametric Mapping (Friston et al., 1994) version 2 revealed hypoperfusion predominantly in the left parieto-occipital region and frontal operculum, whereas hypoperfusion of the right parietal area was quite limited (Figure 2). Peak reduction was noted in the left middle occipital (Brodmann's Area [BA] 19), precuneus (BA 31), inferior and middle frontal (BA 45 and 46) and superior frontal gyri (BA 10), and in the right supramarginal gyrus (BA 40) and precuneus (BA 7).

To quantitatively determine the regional cerebral blood flow, we used a three-dimensional stereotaxic region-of-interest template (Takeuchi, Yonekura, Matsuda, & Konishi, 2002). In this system, the patient's data were first transformed for quantitative evaluation with the Patlak plot method (Matsuda et al., 2004), and then normalised to the Montreal Neurological Institute coordinate space with Statistical Parametric Mapping version 2. We



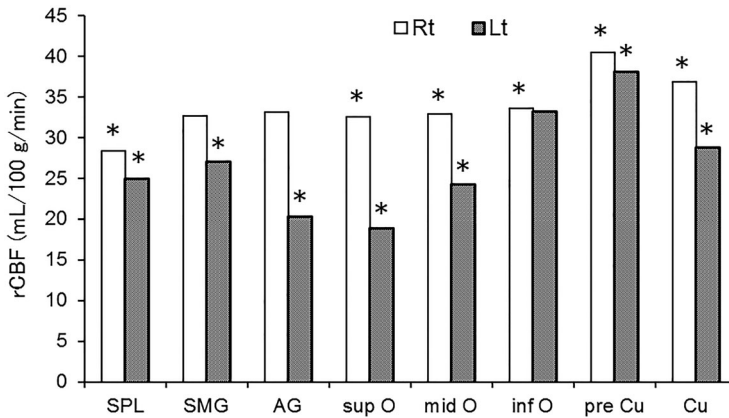
**Figure 1.** MRI scan of the patient. Diffusion-weighted (upper), and T2-weighted axial (middle) and coronal (lower) images obtained three days after onset revealed hyperintensity in the left parieto-temporo-occipital region (cuneus, precuneus, lateral occipital, angular, posterior supramarginal and superior and middle temporal gyri, and superior parietal lobule), insula, and several spotty areas of the bilateral frontal and parietal subcortical regions.



**Figure 2.**  $^{99m}\text{Tc}$ -ECD-SPECT images at two weeks after onset. Hypoperfusion was noted in the left parieto-occipital region and frontal operculum and small areas of the right superior parietal lobule. The patient's data were compared with the normal subject database with the same generation and sex by the two sample  $t$ -test ( $n = 20$ , uncorrected  $p < .001$ ) using statistical Parametric Mapping version 2.

compared regional cerebral blood flow (early picture method) (Matsuda et al., 1992) of some of the anatomically determined region of interests in the parietal and occipital lobes between the left and right hemispheres (Figure 3). As shown, hypoperfusion was most pronounced in the left angular and superior and middle occipital gyri. It is also noteworthy that blood flow in the right parietal and occipital areas was lower compared with the normal control (mean, above 40 mL/100 g/min in each region;  $n = 22$ , ages between 60 and 87 years old) (Matsuda, 2004).

The patient underwent the following tests during 4–20 days after onset. She said, “I’ve become demented,” and was cooperative to the examinations. Written informed consent



**Figure 3.** Regional cerebral blood flow of the parietal and occipital lobes. Hypoperfusion was the most pronounced in the left angular and superior and middle occipital gyri. The patient's data were first transformed for quantitative evaluation with the Patlak plot method, and then normalised to the Montreal Neurological Institute coordinate space with Statistical Parametric Mapping version 2. \* More than 2 SD below the normal mean.

Notes: AG: angular gyrus, Cu: cuneus; inf O: inferior occipital gyrus, mid O: middle occipital gyrus, pre Cu: precuneus, rCBF: regional cerebral blood flow, SMG: supramarginal gyrus, SPL: superior parietal lobule, sup O: superior occipital gyrus.

was obtained from the patient's family. The study protocol was approved by the research ethics committee of our hospital.

### **Neuropsychological assessment**

On smooth pursuit, she had difficulty following the examiner's index finger. Her eye movement was limited to the left horizontal half space. She could pursue the target moving to the left, with saccadic (stepwise) eye movement, and then could no longer gaze up or down; her eyes immediately returned to the midline. She also could not pursue the target moving to the right; her eyes stopped moving at the midline. However, when verbally urged repeatedly, she could move her eyes in all directions promptly and without saccadic eye movement. On the other hand, she could not point to a mirror, a sink, or an infusion bag stand that was placed within two metres in front of her in the left half of the room. She complained that she could not find objects that had been placed in the room.

Next, we asked her to grasp a hammer head that was positioned in the four quadrants visual space. She could hardly grasp the head in the left half space with either hand, first grasping the helve, and then approaching the head, with the performance with the right hand being poorer. When the hammer was positioned in the right half space near the midline, she searched for the hammer with her eyes fixed, staring into empty space like a blind person, with either hand.

### **Standard cognitive tests**

In the following tests, all the stimulus cards or sheets of paper were placed in the front left half space of the patient because she did not notice a stimulus in her right visual field



**Table 1.** Standard neuropsychological test scores.

Test	Score
MMSE	10.1/30
WAIS-III	
Block design	0
WAB	
Spontaneous speech	
Information content	3/10
Fluency	9/10
Naming total	4.1/10
Object naming	36/60
Repetition	3.2/10
Comprehension total	3.15/10
Yes/no questions	44/60
Auditory word recognition	11/60
Sequential commands	8/80
Reading total	0.38/10
Comprehension of sentences	0/40
Kanji written word-object matching	1/3
Kana written word-object matching	1/3
Recognition of orally spelled kanji	0/6
Oral spelling of kanji characters	0/6
Writing total	1.7/10
Copying of sentences	1/10
Kanji writing from dictation	1.5/6
Kana writing from dictation	2/6
Drawing	4/30
Calculation	6/24
Praxis	lt. 9.17/10, rt. 9.17/10
BIT	
Line crossing	lt. 4/18, rt. 2/18
Letter cancellation	lt. 0/20, rt. 0/20
Star cancellation	lt. 4/27, rt. 0/27
Figure and shape copying	0/4
Line bisection	lt. 0/3, rt. 0/3
Representational drawing	0/3
Article reading	0/9
Address copying	0/4
Sentence copying	0/5
VPTA	
Counting of scattered dots	0/3
Discrimination <sup>a</sup> and matching <sup>b</sup> of configuration	6/6 (delayed)
Overlapping figures	0/3
Copying of figures	1/3
Categorization of line-drawings	0/5
Picture comprehension	partial
Naming of famous faces	5/8
Pointing of famous faces <sup>b</sup>	2/8
Discrimination of unfamiliar faces <sup>a</sup>	2/4 (delayed)
Matching of unfamiliar faces <sup>b</sup>	1/3 (delayed)
Emotional expression	3/3 (delayed)
Discrimination of sex <sup>a</sup>	0/4
Discrimination of age <sup>a</sup>	4/4

Notes: MMSE: Mini-Mental State Examination; BIT: Behavioural Inattention Test; VPTA: Visual Performance Test for Agnosia (Japan Society for Higher Brain Dysfunction).

<sup>a</sup>Alternative choice task.

<sup>b</sup>Sixfold choice task.

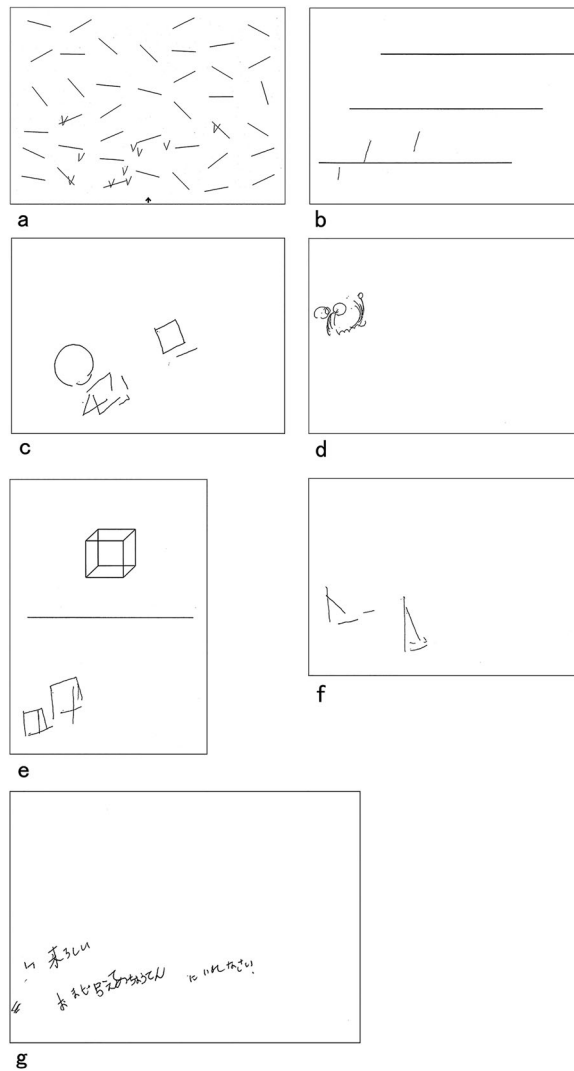
(Table 1). Due to visuospatial cognitive disorder and aphasia, the patient did not perform nearly all subtests of the Wechsler Adult Intelligence Scale-III (WAIS-III). The digit span forward score was two. She did not even perform the first two-block assignment in the Block Design test.

The Western Aphasia Battery (WAB, Japanese edition) (Sugishita, 1986) revealed fluent speech: poor information with perseveration and phonemic paraphasia that lead to *conduite d'approche* (self-correction). On describing a picture card that was placed in her left half space, she referred only to a car and a girl pouring a drink into a glass, which were on the left side of the card. In auditory word recognition, she did not point to a target object out of six items on a desk (0/6), in a drawing card (1/6), or in a room (1/6) (simultanagnosia), nor did she point to a body part (2/6), a finger (2/5), or a left or right body part (1/7) (Gerstmann's syndrome). She did not even point to a target object among three objects on a desk, saying, for example, "Pencil? There is no pencil." In written word-object matching, the patient first looked at a stimulus card on which a word was printed in kanji (Japanese morphograms) or kana (Japanese phonetic writing), then she was asked to point to the corresponding object from six items on a desk or a card (each item arranged in 2 rows by 3 columns, that is, six choices). She had difficulty with this task because of aphasic reading impairment, an inability to turn her eyes from the stimulus card (oculomotor apraxia), and a deficit of searching for multiple objects (simultanagnosia). Reading of sentences was very difficult, and was limited to words. Writing was also markedly impaired; she could not spell out her own name. Impaired kanji recall (lexical agraphia) and phonemic paraphasia of kana (phonological agraphia) were noted. Written characters were illegible and located in the lower left half of the paper, and the character lines slanted upward, suggesting spatial agraphia due to the right HSN (Figure 4(g)). Poor repetition and abundant phonemic paraphasia and paraphasia in reading and writing led to the diagnosis of conduction aphasia. Poor comprehension was inconsistent with conduction aphasia. However, this was mainly caused by simultanagnosia. In drawing, constructional disorder was noted. She could not draw anything but a circle or a square (Figure 4(c)). Specifically, impairment of mental imagery was evident: she drew bizarre shapes on the spontaneous drawing of a person (Figure 4(d)). Calculation was also impaired: alexia of digits as well as anarithmetia (impaired mental arithmetic) accounted for the low score. In praxis, the patient used a finger comb (body part as object) and gestured to fold a paper incorrectly, suggesting slight ideomotor apraxia.

The patient's line crossing, letter and star cancellation, line bisection, and drawing of figures in the Behavioural Inattention Test (BIT, Japanese edition) (Ishiai, 1999) revealed the right HSN. However, the fact that lines, letters, and stars were rarely cancelled in nearly the entire space of the paper suggested the involvement of oculomotor apraxia and simultanagnosia (Figure 4(a,b)). Furthermore, the copying of figures was poor due to HSN and visuoconstructive disorder (Figure 4(e,f)).

The Visual Performance Test for Agnosia (VPTA) (Japan Society for Higher Brain Dysfunction, 1997) revealed the following: (1) Visual processing was generally slow. (2) Multiple choice (sixfold) tasks (correct response total: 6/14) were less successful than alternative choice tasks (correct response total: 9/15). (3) Fundamental visual perception, such as line length, shape, and line slope, was preserved. (4) Counting of scattered dots was unsuccessful because of HSN for the right and simultanagnosia: she did not notice one to three dots on the right side of the sheet. (5) Counting of overlapping figures was incomplete. (6) Understanding a picture portraying a complex situation in which a boy has secretly eaten a girl's donut was impossible, although she could recognise a boy and





**Figure 4.** Drawing samples. (a) Line cross (BIT): Lines on the lower left were predominantly checked. A checking mark was displaced in some lines (optic ataxia). (b). Line bisection (BIT): The patient first said “There is no line.” Thus, the examiner pointed to the lines. All the marks were deviated to the left and not on the lines (optic ataxia). (c) Spontaneous drawing (WAB). Circle, square, and cube were drawn from the left. Figures were rotated in a counterclockwise direction. (d) Spontaneous drawing of a person (WAB). The patient stated that she had drawn eyes and legs but had not done this well. (e) Copying a cube (BIT): The patient said, “This is a square pyramid?” The left figure was drawn in the second trial. (f) Copying a large triangle, a parallelogram with a diagonal line, and a small triangle (BIT): The patient recognised the left triangle. The right half of the parallelogram and the right small triangle were neglected. (g) Writing on dictation (WAB): A sentence “新しい 甘酒を 5本のひょうたんに 入れなさい (Pour new sweet sake into five cups of gourd)” was dictated. Both linguistic agraphia (lexical agraphia of kanji and phonological agraphia of kana) and motor agraphia (spatial agraphia) were noted. Written characters were illegible, overlapped, and located on the left lower half of the paper, and the character lines slanted upward, suggesting spatial agraphia. Notes: BIT: Behavioural Inattention Test, WAB: Western Aphasia Battery.

two girls eating donuts (simultanagnosia). (7) Alternative discrimination of unfamiliar faces or sex was difficult.

Characteristically, in some of the alternative or multiple choice tasks, the patient saw an object, but she said that in the next moment the object looked different from what she had seen. On other occasions, she said, "I see it, but it looks vague."

At 19 days after onset, the patient pursued the target moving to the right visual field. Optic ataxia recovered so that she could grasp a hammer head correctly in the left hemifield with either hand. In the right hemifield, she grasped the target when asked to direct her attention to the right with either hand. She saw a pencil and comb among three objects on the left side of the desk, but did not find a book that was placed nearest to the centre of the desk. When these items were placed further to the left, she said she saw a book this time. However, when asked about where the book was a few minutes later, she could not find it again. In the left hemi-space of a room, she could find a mirror and sink that she could not find 11 days before, but pointed to an empty area in the left hemi-space when asked about the position of an infusion bag stand in the right hemi-space.

The patient suffered a second infarction at 22 days after onset, this time in the right periventricular white matter and diffuse spotty areas in the right frontal and parietal sub-cortical region. She became comatose and died at 34 days after the first stroke. Autopsy was not conducted.

## Discussion

The patient presented with right homonymous hemianopia, conduction aphasia with Gerstmann's syndrome, some features of Bálint's syndrome (oculomotor apraxia, optic ataxia, and simultanagnosia), HSN for the right, slight ideomotor apraxia, constructional disorder, and impaired mental imagery of letters and objects. Oculomotor apraxia, optic ataxia, and simultanagnosia were more pronounced in the right hemi-space.

The lesion was bilaterally distributed. However, the right hemisphere lesion was diffuse multiple lacunar and too small to cause symptoms, although the MRI lesion demarcation does not itself rule out the possibility of neural damage. In contrast, the left hemisphere lesion involved a large area in the parieto-temporo-occipital region, including the superior parietal, posterior supramarginal, angular, superior, and middle occipital gyri, and precuneus. In fact, all the symptoms and signs, except for some features of Bálint's syndrome, could be explained by the left hemisphere lesion.

### *Bálint's syndrome?*

First, we will discuss briefly whether or not our patient showed Bálint's syndrome. We considered that she had simultanagnosia on the basis of the following findings: (i) she could hardly point to objects in a room on verbal commands; (ii) she identified only a few objects in a picture card and did not grasp the situation of the picture (WAB and VPTA); (iii) she could not correctly count scattered dots; and (iv) she showed difficulty with multiple choice (three or more) tasks. However, the fact that she noticed a moving finger at least in the left visual field on confrontation testing, with her eyes fixed on the examiner's index finger in the centre of the visual field, contradicts the diagnosis of

simultanagnosia (inability to perceive more than one object at a time). On the other hand, the fact that she also showed difficulty with alternative tasks (between-object distance: 8–9 cm away from each other), such as the discrimination of configurations or faces (VPTA), suggested a subtle or milder form of simultanagnosia (Coslett & Chatterjee, 2003; De Renzi, 1985), in which case a patient cannot describe a complex scene of a picture. We thought, from a strict point of view, that our patient did not show Bálint's syndrome as described by Bálint (1909), although she had mild simultanagnosia or simultanagnosia in a broader sense (inability to grasp the meaning of a picture) that shared some features with Bálint's simultanagnosia.

It is possible to think that the patient had unilateral (right-sided) simultanagnosia in a strict sense (inability to perceive more than one object). In fact, she did not notice a target in the right visual field, with her eyes fixed on the centre of the visual field on confrontation testing. However, this was caused by right hemianopia: if she had simultanagnosia in a strict sense, she could have found the target in the end. There still remains the possibility that right-sided simultanagnosia in a strict sense was masked by concomitant right hemianopia.

An interesting phenomenon that occurred during examinations was that the patient sometimes did not find an object directly in front of her. She saw the object, but saw it differently in the next moment, saying "It looks different." or "It looks vague" (Neuropsychological assessment). This was probably because visual images of objects were so unstable that she did not match them with the real objects. Another explanation is that this "seeing, but seeing differently" phenomenon occurred in the centre of the visual field, that is, a boundary between the left (attentional) visual field and the right, more impaired (attentional), visual field. It is possible that different degrees of attentional processes in the left or right hemisphere interacted with each other in the centre of the visual field.

### ***Unilateral lesion or not?***

The present case does not contradict the conventional view that Bálint's syndrome is caused by damage to the bilateral parieto-occipital region. However, it is possible that the present oculomotor apraxia, optic ataxia, and simultanagnosia resulted from a unilateral lesion of the left parieto-occipital area. We will discuss this issue in the following few paragraphs.

On smooth pursuit in the test of oculomotor apraxia, her eyes moved horizontally to the left, but did not move to the right from the midline. Saccade was prompt and without apparent stepwise movement, although she had difficulty shifting her gaze. It is known that contralateral pursuit gaze is impaired in unilateral parieto-occipital lesions (Sharpe, 2008). Specifically, in the case of HSN, the absence of saccade (failure to shift gaze) or delayed and staircase-pattern saccade in the contralateral hemifield was observed (Girotti, Casazza, Musicco, & Avanzini, 1983). On the other hand, on smooth pursuit in a case of Bálint's syndrome, similar saccadic eye movement was observed in the right hemifield and horizontal eye movement was completely abolished in the left hemifield (Girotti et al., 1982). It is likely that the patient, in this case, had left hemianopia and left HSN. Taken together, deficit of smooth pursuit in the right hemifield in our patient resulted from the left parietal lesion, whereas her restricted gaze in the left hemifield cannot rule out the involvement of the right parietal lobe lesion.

On the test of optic ataxia, the patient could hardly grasp the head of the hammer in the left half space, especially with the right hand (hand effect) (Perenin & Vighetto, 1988). When the hammer was positioned in the right space near the midline, she searched for the hammer, with her eyes fixed, like a blind person, with either hand (field effect) (Perenin & Vighetto, 1988). These findings could be explained by a single lesion of the left hemisphere.

In some tests for dorsal simultanagnosia, there was also prominent left–right asymmetry, despite a stimulus card or a sheet being placed in the left hemi-space of the desk, where the effect of right hemianopia was prevented. In describing the scene on a drawing card of the WAB test, she referred to only two objects on the left side of the card. On counting the scattered dots, she did not notice a few dots on the right side of the card. In line crossing and star cancellation, only the items in the lower left half of the paper were crossed or cancelled. These findings suggested that simultanagnosia covered the entire visual field, but when right HSN accompanied it, the right side was completely unattended. Previous studies suggested the involvement of the bilateral parieto-occipital area in dorsal simultanagnosia (Chechacz et al., 2012), with the only exception of the unilateral (right) cuneus and precuneus subcortical haemorrhage that resulted in dorsal simultanagnosia and optic ataxia (Clavagnier, Fruhmann Berger, Klockgether, Moskau, & Karnath, 2006). Therefore, it is possible that our patient's simultanagnosia was caused solely by the left parietal lobe lesion.

Overall, it remains unknown whether or not all of oculomotor apraxia, optic ataxia, and simultanagnosia occur due to a unilateral (left hemisphere) lesion, although optic ataxia and simultanagnosia can be caused by a unilateral lesion. An important point here is that the patient showed predominantly right-sided oculomotor apraxia, optic ataxia, and simultanagnosia. This symptomatic asymmetry could not be explained unless we consider that the left lesion was more responsible for the symptoms. If this was the case, how could a unilateral lesion cause Bálint's (-like) syndrome? With respect to this point,  $^{99m}\text{Tc}$ -ECD-SPECT showed that blood flow was diffusely lowered not only in the left but also in the right hemisphere. This hypoperfusion of the right hemisphere was probably caused by interhemispheric diaschisis (reduction of metabolism and blood flow in the cortex contralateral to a lesion by interruption of transhemispheric pathways) (Carrera & Tononi, 2014). It is possible to consider that an extensive lesion in the left parieto-occipital area transcallosally suppressed the function of the opposite area, which resulted in oculomotor apraxia, optic ataxia, and simultanagnosia.

In fact, the three symptoms recovered to various degrees not only in the left but also in the right hemi-space 19 days after onset (Neuropsychological assessment). Simultanagnosia in the left hemifield recovered, whereas simultanagnosia in the right hemifield persisted. This suggested the quicker recovery of the less damaged right hemisphere lesion and supported the hypothesis that a unilateral lesion can cause asymmetric (predominantly contralateral) simultanagnosia.

### ***Simultanagnosia with or without HSN***

In the present patient, oculomotor apraxia, optic ataxia, and simultanagnosia were observed in the entire visual space, but the eye movement impairment was severer in the right visual space, contralateral to the lesion, in oculomotor apraxia. On the other

hand, limited eye movement prevented the patient from directing attention to the target in the right hemi-space, which contributed to the poorer performance in reaching (optic ataxia) and searching (simultanagnosia) in the right than in the left hemi-space. As described in the previous session, an extensive lesion of the left parieto-occipital cortices is sufficient to cause simultanagnosia in a broader sense, and HSN is not a prerequisite for the symptom. However, almost complete unawareness of the right hemi-space with sparing of the left lower hemi-space in line crossing, star cancellation (BIT), and picture description (WAB) suggests that accompanying HSN served to aggravate simultanagnosia in the right hemi-space.

It has been reported that concomitant hemianopia does not exacerbate HSN (Halligan, Marshall, & Wade, 1990). However, it is not known whether accompanying hemianopia affects simultanagnosia. Given that the eye movement was more limited in the right visual field in our patient, it is also possible that the hemianopia aggravated simultanagnosia in the right hemi-space. This is why we set the stimulus cards or sheets in the left hemi-space of the patient. However, it is uncertain whether this condition avoided the influence of right hemianopia completely.

### ***Dorsal simultanagnosia vs. Ventral simultanagnosia***

The differences between dorsal (DS) and ventral (VS) simultanagnosia are summarised in Table 2. Both have a common feature of an inability to understand the meaning or situation of an entire picture or scene, despite the fact that the individual parts are accurately perceived (Wolpert, 1924). A cardinal difference is that simultanagnosia is evident in activities of daily living in DS (Coslett & Chatterjee, 2003), whereas the deficit is limited to the test situation in VS (Sakurai et al., 2016). That is, a patient with DS cannot find an object around him. In contrast, a patient with VS has no difficulty with daily life. Farah (1990) suggested counting scattered dots for the differential diagnosis; a patient with DS fails to count some dots in this test, whereas a patient with VS can count the number without difficulty. This test is simply applicable. However, pointing to objects in a room is more feasible.

**Table 2.** Dorsal compared with ventral simultanagnosia.

	Dorsal type	Ventral type
Pathogenesis	spatial inattention	parallel processing deficit
Visual perception	Normal	Impaired
Scattered dots	Impaired	Normal
Overlapping figure	Impaired	Normal
Visual searching	Impaired	Normal
Multiple choice	Impaired	Normal
Lesion	bil. P-O	lt. inf O-T rt. sup T-P
Complications	Hemispatial neglect	Prosopagnosia (rt.) <sup>a</sup> Pure alexia (lt.)
	Constructional disorder Spatial agraphia <sup>b</sup>	Constructional disorder Spatial agraphia

Notes: bil.: bilateral; P: parietal; O: occipital; T: temporal.

<sup>a</sup>Sakurai et al. (2016).

<sup>b</sup>Agraphia from a left parietal lesion consists not only of motor (spatial) but also of linguistic (lexical or phonological) level agraphia.

The second principal difference is that fundamental visual perception, e.g., line length, figure size, slope, and shape, is preserved in DS, but is more or less impaired in VS. This is conceivable given that the occipitotemporal area is the lesion site for VS (Sakurai et al., 2016).

The third principal difference is that a patient with DS cannot deal with multiple (three-fold or more) choice tasks, whereas a patient with VS can easily perform these tasks (Sakurai et al., 2016). It should be noted that our patient with DS did not point to a target object among three objects on the left side of the desk, although she could discriminate two stimuli in some tasks.

The above differences are consistent with the view that DS is essentially an attention deficit (Rizzo & Hurtig, 1987) whereas VS is an impairment of visual parallel processing (Sakurai et al., 2016).

### Concluding remarks

The present study suggests that a large lesion of the left parieto-occipital cortices is sufficient to cause oculomotor apraxia, optic ataxia, and simultanagnosia, although the symptoms are more marked in the opposite (right) hemi-space. How these asymmetric (predominantly contralateral) symptoms differ from Bálint's syndrome due to the bilateral lesion of the parieto-occipital area should be elucidated further. It is clinically important to determine whether a lesion producing Bálint's (-like) syndrome is bi- or unilateral because a unilateral lesion is expected to be extensive and sometimes life-threatening. Furthermore, our study draws attention to the importance of determining whether there is any left-right symptomatic asymmetry in Bálint's syndrome due to a bilateral lesion because it serves to predict which hemisphere is more affected. That is, in a bilaterally damaged case, a more disturbed side suggests the more involvement of the opposite hemisphere, and the possible coexistence of USN for that side.

The limitations of the study are that the patient showed aphasia and short-term memory impairment, which reduced the performance test scores. Some problems remain unresolved. For example, it is not clear why our patient with DS could not specifically discriminate the faces or sex of unfamiliar persons. Further study is needed to clarify such issues.

### Disclosure statement

No potential conflict of interest was reported by the authors.

### References

- Bálint, R. (1909). Seelenlähmung des Schauens, optische Ataxie, räumliche Störung, der Aufmerksamkeit. *European Neurology*, 25, 51–66.
- Carrera, E., & Tononi, G. (2014). Diaschisis: Past, present, future. *Brain*, 137(Pt 9), 2408–2422.
- Chechlacz, M., Rotshtein, P., Hansen, P. C., Riddoch, J. M., Deb, S., & Humphreys, G. W. (2012). The neural underpinnings of simultanagnosia: Disconnecting the visuospatial attention network. *Journal of Cognitive Neuroscience*, 24(3), 718–735.
- Clavagnier, S., Fruhmann Berger, M., Klockgether, T., Moskau, S., & Karnath, H. O. (2006). Restricted ocular exploration does not seem to explain simultanagnosia. *Neuropsychologia*, 44(12), 2330–2336.



- Cogan, D. G., & Adams, R. D. (1953). A type of paralysis of conjugate gaze (ocular motor apraxia). *Archives of Ophthalmology*, 50(4), 434–442.
- Coslett, H. B., & Chatterjee, A. (2003). Bálint's syndrome and related disorders. In T. E. Feinberg & M. J. Farah (Eds.), *Behavioral neurology and neuropsychology* (2nd ed., pp. 325–335). New York, NY: McGraw-Hill.
- De Renzi, E. (1985). Disorders of spatial orientation. In J. A. M. Frederiks (Ed.), *Handbook of clinical neurology* (Vol. 45, pp. 405–422). Amsterdam: Elsevier.
- Farah, M. J. (1990). *Visual agnosia: Disorders of object recognition and what they tell us about normal vision*. Cambridge, MA: The MIT Press.
- Friston, K. J., Holmes, A. P., Worsley, K. J., Poline, J.-P., Frith, C. D., & Frackowiak, R. S. J. (1994). Statistical parametric maps in functional imaging: A general linear approach. *Human Brain Mapping*, 2, 189–210.
- Girotti, F., Casazza, M., Musicco, M., & Avanzini, G. (1983). Oculomotor disorders in cortical lesions in man: The role of unilateral neglect. *Neuropsychologia*, 21(5), 543–553.
- Girotti, F., Milanese, C., Casazza, M., Allegranza, A., Corridori, F., & Avanzini, G. (1982). Oculomotor disturbances in Balint's syndrome: Anatomoclinical findings and electrooculographic analysis in a case. *Cortex*, 18(4), 603–614.
- Halligan, P. W., Marshall, J. C., & Wade, D. T. (1990). Do visual field deficits exacerbate visuospatial neglect? *Journal of Neurology, Neurosurgery & Psychiatry*, 53(6), 487–491.
- Ishiai, S. (1999). *Behavioural inattention test* (Japanese ed.). Tokyo: Shinkoh-Igaku Shuppansha.
- Japan Society for Higher Brain Dysfunction. (1997). *Visual perception test for agnosia: VPTA*. Tokyo: Shinkoh-Igaku Shuppansha.
- Matsuda, H. (2004). SPECT. *Rinsho Seishin Igaku*, 33(Suppl), 556–552.
- Matsuda, H., Mizumura, S., Soma, T., & Takemura, N. (2004). Conversion of brain SPECT images between different collimators and reconstruction processes for analysis using statistical parametric mapping. *Nuclear Medicine Communications*, 25(1), 67–74.
- Matsuda, H., Tsuji, S., Shuke, N., Sumiya, H., Tonami, N., & Hisada, K. (1992). A quantitative approach to technetium-99m hexamethylpropylene amine oxime. *European Journal of Nuclear Medicine*, 19(3), 195–200.
- Perenin, M. T., & Vighetto, A. (1988). Optic ataxia: A specific disruption in visuomotor mechanisms. I. Different aspects of the deficit in reaching for objects. *Brain*, 111(Pt 3), 643–674.
- Rizzo, M., & Hurtig, R. (1987). Looking but not seeing: Attention, perception, and eye movements in simultanagnosia. *Neurology*, 37(10), 1642–1642.
- Rizzo, M., & Vecera, S. P. (2002). Psychoanatomical substrates of Bálint's syndrome. *Journal of Neurology, Neurosurgery & Psychiatry*, 72(2), 162–178.
- Robertson, L. C. (2003). Binding, spatial attention and perceptual awareness. *Nature Reviews Neuroscience*, 4(2), 93–102.
- Sakurai, Y., Hamada, K., Tsugawa, N., & Sugimoto, I. (2016). Ventral simultanagnosia and prosopagnosia for unfamiliar faces due to a right posterior superior temporal sulcus and angular gyrus lesion. *Neurocase*, 22(1), 122–129.
- Sharpe, J. A. (2008). Neurophysiology and neuroanatomy of smooth pursuit: Lesion studies. *Brain and Cognition*, 68(3), 241–254.
- Sugishita, M. (1986). *The Western aphasia battery, Japanese edition*. Tokyo: Igaku-Shoin.
- Takeuchi, R., Yonekura, Y., Matsuda, H., & Konishi, J. (2002). Usefulness of a three-dimensional stereotaxic ROI template on anatomically standardised 99mTc-ECD SPET. *European Journal of Nuclear Medicine and Molecular Imaging*, 29(3), 331–341.
- Wolpert, I. (1924). Die simultanagnosie—störung der gesamtauffassung. *Zeitschrift für die Gesamte Neurologie und Psychiatrie*, 93, 397–415.