

[ CASE REPORT ]

## Conscious Hemiasomatognosia with No Somatosensory Disturbance Other Than a Unique Problem in Tactile Localization

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### Abstract:

Conscious hemiasomatognosia is a disorder of the bodily self, involving subjective symptom where patients feel as if their whole body or part of one side has disappeared. Somatosensory disturbance is considered an essential component of conscious hemiasomatognosia. We herein report a 64-year-old man with conscious hemiasomatognosia of the right arm that developed after a left parietotemporal infarction, without any somatosensory disturbance except for a unique tactile localization problem. His response to the tactile localization test suggested impaired recognition of the positional relationship of his right arm relative to the entire body but normal recognition of positional relationships within the arm.

**Key words:** conscious hemiasomatognosia, infarction, somatosensory disturbance, superior parietal lobule

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### Introduction

Conscious hemiasomatognosia refers to a state of the bodily self, wherein a patient is evidently aware and complains that the entire side of the body contralateral to the lesion, or a part thereof, has disappeared, and they are unable to locate it (1). The incidence of this symptom shows no difference according to the damaged side of the brain (2). The condition differs from nonconscious hemiasomatognosia, which almost exclusively occurs in patients with right-brain damage; in these cases, patients pay no attention to the side of the body contralateral to the lesion and behave as if it is nonexistent (1).

In previously reported cases of conscious hemiasomatognosia somatosensory disturbances attributable to the affected side of the body were noted (2-4). Most cases had severe disturbances in all somatosensory modalities (2, 3); thus, somatosensory disturbance was considered an essential compo-

nent of conscious hemiasomatognosia (1-3). Wolpert et al. (4) described a patient in whom conscious hemiasomatognosia was associated with a superior parietal lobule cyst but whose primary somatosensory modalities (tactile, vibratory, and positional) (5) were functioning normally. However, in that patient, the cortical somatosensory modalities (5) dealing with size, texture, shape, and objects were impaired.

We herein report a case of conscious hemiasomatognosia with a unique tactile localization issue but without any other disturbances in either the primary or cortical somatosensory modalities.

### Case Report

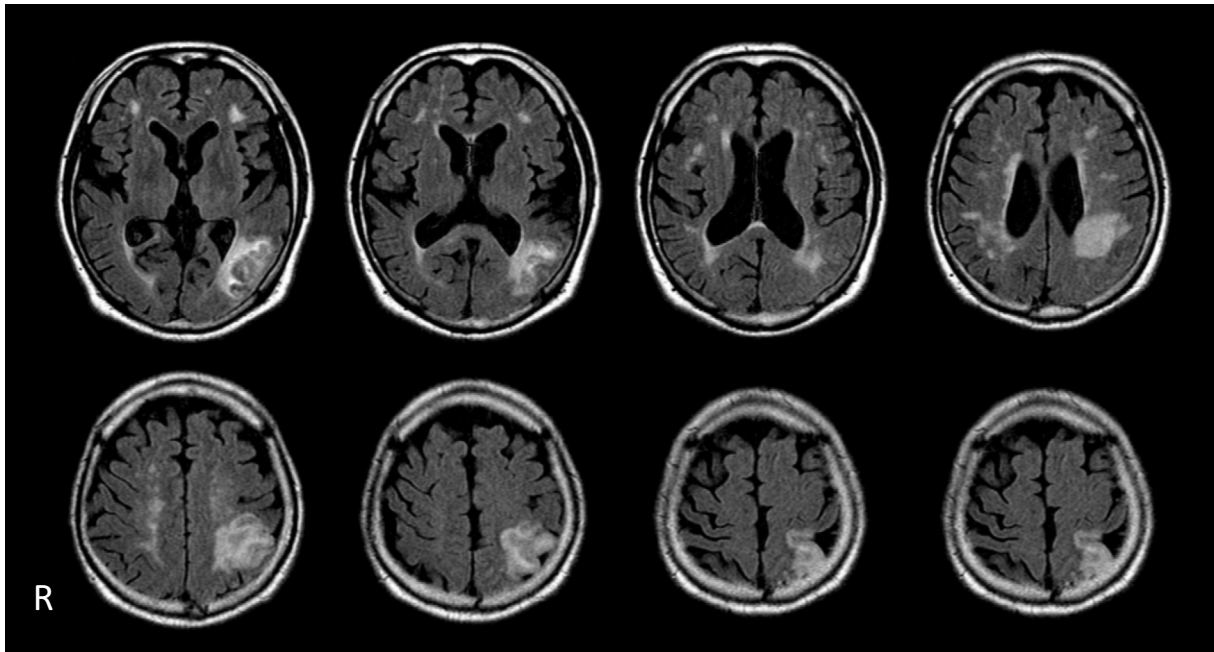
Written consent for the publication of this report was obtained from the patient.

A 64-year-old right-handed man with 12 years of education was admitted to our hospital because he reported diffi-

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**Figure 1.** Magnetic resonance images. Fluid-attenuated inversion recovery images indicating infarction in the posterior left postcentral gyrus, anterolateral superior parietal lobule, anterior intraparietal sulcus, superior supramarginal gyrus, anterosuperior angular gyrus, and middle temporal gyrus, as well as in the subcortical white matter of these regions.

culty in seeing his right side. He had a history of atrial fibrillation. He exhibited right homonymous upper quadrantanopia, right optic ataxia, agraphia, acalculia, ideational apraxia, and color anomia. No common patterns of somatosensory disturbances were detected by standard testing methods when the patient was hospitalized. Magnetic resonance imaging (MRI) of the brain revealed an infarction that involved the deep part of the anterior wall in the left postcentral sulcus, anterolateral superior parietal lobule, anterior intraparietal sulcus, superior supramarginal gyrus, anterosuperior angular gyrus, and middle temporal gyrus, as well as the subcortical white matter of these regions (Fig. 1). As MR angiography excluded stenosis of major cerebral arteries, cardiogenic embolism was considered the likely cause. No epileptiform discharge was observed in the electroencephalogram, although brain activity was reduced in the left hemisphere during sleeping, suggesting mild dysfunction in the left hemisphere.

He was discharged three months after the stroke onset. Ideational apraxia and color anomia resolved within a few months after discharge. During an outpatient visit 1.5 years after the stroke onset, he reported the persistence of the following symptoms since the stroke: “My right arm disappears when I am not looking at it... I do not know where it is, but I think ‘not there’ is the right phrase to describe what I feel. My right arm appears when I see it or touch it with my left hand, and no part other than my right arm from the shoulder to the fingers seems to be missing... I have been suffering from this condition since the onset of the infarction.” Because of this symptom, the patient failed certain tasks if he could not actually see his right upper limb, such as putting

his right arm into a sleeve to wear a shirt or grabbing something out of his sight with his right hand.

To further investigate this case, we conducted neuropsychological tests and somatosensory examinations. The results of neuropsychological tests indicated complete righthandedness. There were no disturbances in his general attention, intelligence, or spoken language, but mild agraphia and severe acalculia were observed. There was no hemispatial neglect (Table 1). The results of the somatosensory examinations of the left and right upper extremities are presented in Table 2.

The patient’s thermal nociception, tactile sense, vibratory sense, position sense, two-point discrimination, graphesthesia, size discrimination, roughness discrimination, material identification, two- and three-dimensional shape identification, and object identification abilities were normal, with no marked differences between the left and right sides. The patient did not show tactile extinction. Therefore, the primary sensory modalities were deemed to be functioning normally, as well as his cortical sensory modalities, except for the issue with tactile localization described below.

Tactile localization was examined by the following method (6): The patient was blindfolded throughout the examination. The examiner touched various points on his right or left arm with a pencil tip for approximately 0.5 seconds. The patient was asked to indicate the points by touching the same locations using a finger of the other arm immediately. Compared with the results of healthy volunteers, noteworthy differences were observed between the actual and patient-indicated locations on many occasions (Table 2). However, the patient promptly corrected the discrepancies exhibited in

**Table 1. Results of Neuropsychological Tests.**

Test	Performance
<b>Handedness</b>	
Edinburgh Handedness Inventory (max: 100)	100
<b>General attention</b>	
Digit span	7
Spatial span	5
<b>Intelligence</b>	
Mini-Mental State Examination (max: 30)	26
Addenbrooke's Cognitive Examination Revised (max: 100)	94
Raven's Colored Progressive Matrices (max: 36)	26
<b>Language and calculation</b>	
Standard Language Test of Aphasia	
Listening (max: 100)	100
Speech (max: 100)	98.0
Reading (max: 100)	95.0
Writing (max: 100)	77.5
Calculation (max: 100)	45.0
<b>Hemispatial neglect</b>	
Catherine Bergego Scale (max: 30)	0

max: maximum

the first attempt by moving his finger to the correct location (Fig. 2). The patient stated that, "I do not know where my arm is, so I touch it by guessing. Then, I become aware of the correct location immediately, so I touch that location." When the same points on the left arm were touched with a finger of the right hand, there were no substantial differences between the actual and patient-indicated locations (Table 2). If the patient could see the upper right limb when the examiner was touching or when he was indicating the points by touching the same locations, he could correctly touch the same location where the examiner touched.

Somatosensory evoked potentials were elicited and recorded by electrical stimulation of the median nerves at the wrist. N20 was evoked at 21.0 mseconds with 3.95  $\mu$ V (N20-N25) in the right hemisphere and at 21.2 mseconds with 2.25  $\mu$ V (N20-N25) in the left hemisphere, suggesting there was no marked difference in the latency or amplitude between the hemispheres (Fig. 3).

The right homonymous upper quadrantanopia, right optic ataxia, agraphia, acalculia and conscious hemiasomatognosia persisted for three years after the stroke.

## Discussion

Severe somatosensory disturbance have been considered as an essential manifestation of conscious hemiasomatognosia (1-3). Wolpert et al. (4) described a patient with this symptom whose primary somatosensory modalities were functioning normally. Even in that patient, the cortical somatosensory modalities dealing with size, texture, shape, and objects were impaired. However, in the present patient, these cortical somatosensory modalities had no disturbances. Furthermore, the primary somatosensory modalities were intact,

and the somatosensory evoked potential was also normal, indicating that conscious hemiasomatognosia can occur in the absence of somatosensory disturbances, with the exception of the above-mentioned unique tactile localization problem.

Local brain injuries that cause conscious hemiasomatognosia include lesions in the pons (1), thalamus (1, 2), and parietal lobe (3, 4). In the present case, conscious hemiasomatognosia was caused by a lesion in the left parietotemporal region of the brain. Previous studies have reported conscious hemiasomatognosia of the upper extremities occurring as a manifestation of epilepsy (7, 8) and in response to electrical stimulation of the cortex (8). In one case, the right parietal operculum and superior parietal lobule behind the post-central gyrus were resected to treat epilepsy (7), and in another case, the focus involved was located in the left superior parietal lobule immediately posterior to the postcentral gyrus (8). However, in these cases, the symptoms lasted briefly, and detailed somatosensory examinations were not performed. In a human functional MRI study (9), activation of the left superior parietal lobule was observed during the performance of a task in which the subjects were asked to point to their own body parts, such as the shoulders, elbows, and knees, according to written instructions. These findings as well as those obtained by Wolpert et al. (4) suggest that the lesions responsible for conscious hemiasomatognosia of the upper extremities are located in the anterior part of the superior parietal lobule. In the present case, the lesion included the same region.

In the current case, when visual information was unavailable, the patient failed the task of using his left hand to touch his right arm at the locations originally touched by the examiner. However, the differences between the actual and patient-indicated locations were not as substantial as ex-

**Table 2. Results of Somatosensory Examinations.**

Examination	Performance	
	Left arm	Right arm
Thermal nociception <sup>†</sup>	No left-right differences	
Pain threshold (thumb pulp, g)	12	12
Light touch <sup>†</sup>	No left-right differences	
Vibratory sense	No left-right differences	
Position sense <sup>‡</sup> (max: 40)	40	40
Two-point discrimination (mm)		
Thumb pulp	5.0	4.5
Tip of the middle finger	2.0	2.0
Palm	9.0	8.0
Back of the hand	20.0	20.0
Distal end of the anterior surface of the forearm	23.0	24.0
Proximal end of the anterior surface of the forearm	55.0	59.0
Distal end of the anterior surface of the upper arm	115.0	110.0
Lateral surface of the shoulder	75.0	80.0
Graphesthesia <sup>§</sup> (numbers from 1 to 9, max: 63)	63	63
Size discrimination <sup>¶</sup> (max: 5)	5	5
Roughness discrimination <sup>a</sup> (max: 5)	5	5
Material identification <sup>b</sup> (max: 7)	7	7
Identification of two-dimensional shapes <sup>c</sup> (max: 6)	6	6
Identification of three-dimensional shapes <sup>d</sup> (max: 5)	5	5
Object identification <sup>e</sup> (max: 6)	6	6
Tactile extinction <sup>f</sup>	-	
Tactile localization (touch the examiner-touched location) (distance, mm)		
Thumb pulp	0 (15±6)	18 (4±6)
Tip of the middle finger	10 (14±16)	51 (5±8)
Proximal end of the thenar	11 (14±6)	27 (16±8)
Proximal end of the anterior surface of the forearm	10 (23±10)	92 (16±10)
1/3rd from the proximal end of the anterior surface of the forearm	15 (13±10)	22 (15±12)
1/3rd from the distal end of the lateral surface of the forearm	10 (18±7)	52 (16±15)
1/4th from the distal end of the anterior surface of the upper arm	10 (18±7)	52 (16±15)

<sup>†</sup>The thumb pulp, palm, anterior surface of the forearm, and anterior surface of the upper arm were stimulated.

<sup>‡</sup>The distal interphalangeal joint of the index finger, proximal interphalangeal joint of the middle finger, wrist joint, and elbow joint were moved by 1/10th of the normal range of motion, ten times for each joint.

<sup>§</sup>The palm, back of the hand, distal end of the anterior surface of the forearm, proximal end of the anterior surface of the forearm, distal end of the anterior surface of the upper arm, proximal end of the anterior surface of the upper arm, and lateral surface of the shoulder were stimulated.

<sup>¶</sup>Requested to rank the order of ball sizes for balls with diameters of 50, 40, 35, 30, 25, and 20 mm and having approximately equal weights.

<sup>a</sup>Requested to rank the order of roughness of sandpaper pieces containing 40, 80, 100, 180, and 320 particles per cm<sup>2</sup>.

<sup>b</sup>Velvet, sponge, vinyl, wood, sandpaper, smooth cloth, and rough cloth were used.

<sup>c</sup>Circles, triangles, hexagons, rhombuses, squares, and rectangles were used.

<sup>d</sup>Spheres, cones, cylinders, cubes, and rectangular parallel pipes were used.

<sup>e</sup>Coins, keys, scissors, wristwatches, sharpened pencils, and unsharpened pencils were used.

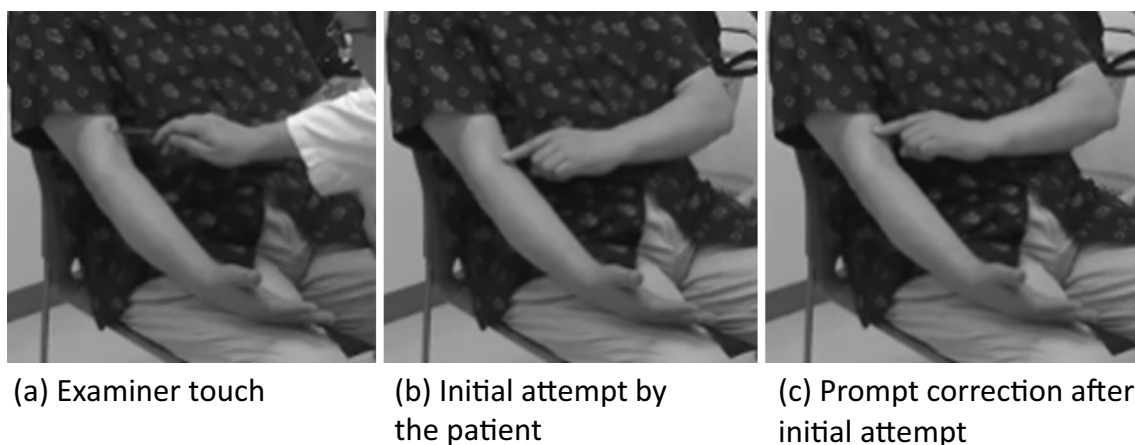
<sup>f</sup>The thumb pulp, tip of the middle finger, proximal end of the thenar, proximal end of the anterior surface of the forearm, 1/3rd from the proximal end of the anterior surface of the forearm, 1/3rd from the distal end of the lateral surface of the forearm, and 1/4th from the distal end of the anterior surface of the upper arm were stimulated.

Numbers in parentheses in the last two columns indicate values derived from 10 healthy volunteers (5 men and 5 women; mean age, 64.7±4.4 years).

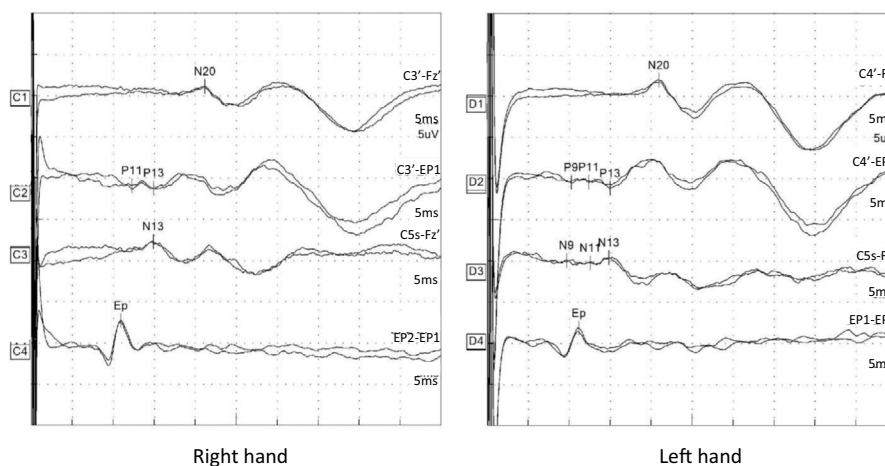
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pected based on his introspective statement of, “I do not know where my arm is.” Therefore, some neural mechanism that allowed limited processing of the approximate positions of body parts was functioning, despite his inability to con-

sciously perceive such information. However, to process detailed positional information of body parts, a neural mechanism responsible for recognizing the existence of body parts is necessary; this mechanism may be impaired in patients



**Figure 2.** Patient reactions during the examination of point localization. The examiner touched a point on the patient's right arms with a pencil tip for approximately 0.5 s (a). The patient was asked to indicate the point by touching the same location using a finger of the left arm immediately. Noteworthy differences were observed between the actual and patient-indicated locations (b). However, the differences apparent in the initial attempt were promptly corrected by the patient by moving his finger from the first location to the correct location (c).



**Figure 3.** Somatosensory evoked potentials (SSEP) of the patient. N20 was evoked at 21.0 ms with 3.95  $\mu\text{V}$  (N20-N25) in the right hemisphere and at 21.2 ms with 2.25  $\mu\text{V}$  (N20-N25) in the left hemisphere.

with conscious hemiasomatognosia.

When the location indicated in the initial attempt was incorrect, the patient was able to promptly correct it by moving the finger from its initial location. Furthermore, he reported that once he touched his arm, he immediately realized the correct location of the touched point. These facts indicate that the positional relationship in the arm between the point touched by the examiner and the point touched by the patient was correctly understood, despite the lack of understanding of the positional relationship between the rest of his body and his arm. This finding suggests that different neural mechanisms are involved in the recognition of the positional relationship of the entire body relative to its major body parts and of the positional relationships within that major part, and that only the mechanisms involved in the

former are impaired in conscious hemiasomatognosia.

**The authors state that they have no Conflict of Interest (COI).**

## References

1. Frederiks JAM. Disorders of the body schema. In: Handbook of Clinical Neurology. Vol 4. Vinken PJ, Bruyn GW, Eds. North-Holland, Amsterdam, 1969: 207-240.
2. Suzuki N, Amano T, Gotoh F. Responsible lesions for conscious hemiasomatognosia. Rinsho Shinkeigaku (Clin Neurol) **22**: 543-551, 1982 (in Japanese).
3. Sasaki K, Hamada Y, Daimaru O, Iwata K. Conscious hemiasomatognosia of the right upper extremity an autopsy case. Nou to Shinkei (Brain Nerve) **27**: 757-765, 1975 (in Japanese).
4. Wolpert DM, Goodbody SJ, Husain M. Maintaining internal repre-

- sentations: the role of the human superior parietal lobe. *Nat Neurosci* **1**: 529-533, 1998.
5. Klingner CM, Witte OW. Somatosensory deficits. *Handb Clin Neurol* **151**: 185-206, 2018.
  6. Halligan PW, Hunt M, Marshall JC, Wade DT. Sensory detection without localization. *Neurocase* **1**: 259-266, 1995.
  7. Salanova V, Andermann F, Rasmussen T, Oliver A, Quesney LF. Parietal lobe epilepsy. Clinical manifestations and outcome in 82 patients treated surgically between 1929-1988. *Brain* **118**: 607-627, 1995.
  8. Nishibayashi H, Nakai Y, Tamura M, Ogura M, Uematsu Y, Itakura T. Ictal asomatognosia due to dominant superior parietal cortical dysplasia. *J Clin Neurosci* **18**: 141-142, 2011.
  9. Felician O, Romaguère P, Anton JL, et al. The role of human left superior parietal lobule in body part localization. *Ann Neurol* **55**: 749-751, 2004.

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