

# Visual form agnosia in multiple sclerosis

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We report a case of multiple sclerosis with visual form agnosia and callosal syndromes. Initially, the patient's visual recognition of object form was severely disturbed at the perceptual stage, in association with left-sided ideomotor apraxia and agraphia. Magnetic resonance imaging showed large white matter lesions in the bilateral frontal and occipital lobes, the latter extending to the occipitotemporal junction, and widespread corpus callosum lesions. Over the course of one year follow-up, neuropsychological examinations indicated that the patient's visual recognition defects occurred not only at the early substage of form perception, but also at the stage of reproducing the shape of objects from visual memory store. The present case suggests that neural connections between the striate cortex and occipitotemporal visual areas are crucial for both the perceptual and associative stages of visual object recognition.

Key words: visual form agnosia; visual perception; occipitotemporal visual area; parallel visual information processing; diaschisis; multiple sclerosis

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Visual object agnosia has been usually classified by Lissauer's distinction between apperceptive and associative agnosia (1). Apperceptive visual agnosia is attributed to a basic defect in visual perception, while associative visual agnosia is attributed to the rupture of connection linking perceptual centers with representational centers (2, 3). Although Lissauer's division has been widely accepted by clinicians, the entity of visual agnosia still remains controversial, mainly due to the difficulty in indexing recognition (4, 5). In the literature, the quality of visual agnosia varies from case to case, particularly in apperceptive agnosics. Warrington defined traditionally labelled apperceptive agnosics with inability to perceive bidimensional shapes as "pseudoagnosia" (6). According to her criterion, convincing cases of apperceptive agnosia seem very rare. Although the point of view of early and later substages of perceptual processing appears justifiable, the qualitative differences between apperceptive agnosics in the literature do not necessarily indicate the validity of her classification (7). Above all, there is no definable point of demarcation between perception and recognition (8). Several cases have been reported to be lying between apperceptive and associative visual agnosia (7, 9–11). These facts raise a question as to

whether visual information processing is carried out in the two hierarchical stages proposed by Lissauer. In fact, experimental and psychological studies have revealed independent parallel substages of visual recognition process (10–12). In this regard, the label of visual form agnosia, termed by Benson (13), would be more appropriate to describe cases with difficulty of form perception, including apperceptive visual agnosia (14).

In the majority of cases with visual form agnosia, causative lesions are centered over the bilateral striate cortex. Visual form agnosia followed pathological processes such as carbon monoxide poisoning, mercury intoxication, cardiac arrest, and basilar artery occlusion (3, 13). In this paper, we present a patient of multiple sclerosis (MS) with visual form agnosia and callosal syndromes. Relatively high frequency rates of cognitive dysfunction have been reported in MS (15, 16), which are mainly related to callosal involvement. Neuropsychological studies have demonstrated left-ear dichotic listening suppression and prolonged latency in the left visual field on tachistoscopic naming tasks (17, 18). Yet, a cortical type of focal cognitive deficits such as aphasia, agnosia or apraxia is very rare (19, 20). To our knowledge, this is the first report to demonstrate visual form agnosia in MS,

and is a challenge to the traditional view that visual form agnosia is invariably caused by lesions in the striate and prestriate cortices.

#### Case report

A 56-year-old, right-handed, woman (KK) was admitted to our Hospital because of transverse myelitis (the first admission between August 1992 and December 1992). She was left with spastic paraparesis, but with normal mental functions. Seven months after the discharge, she began to forget places in her house and go a wrong way. She used to get lost in familiar surroundings. She complained of visual difficulties and could not appreciate visual information such as television or magazines. She was readmitted to our Hospital in August 1993. On admission, physical examination showed no abnormal findings. Neurological examination showed alert consciousness but impaired recognition (Mini-Mental State Examination score 20/30). Precise measurement of visual acuity and visual field were impossible due to severe impairment of visual recognition. However, she could follow moving visual stimuli or bright light, and could respond to confrontation stimuli. The range of eye movements was full. The other cranial nerves were preserved. Muscular strength was normal in the upper limbs and mildly decreased in the lower limbs with spasticity. Deep tendon reflexes were generally increased, and Babinski reflex was bilaterally positive. All modalities of sensations were severely disturbed in the right upper limb. Superficial sensations were preserved in the other limbs but vibration sense was mildly disturbed bilaterally in the legs. Cerebellar ataxia was undetectable. The cerebrospinal fluid contained 2 mononuclear cells/mm<sup>3</sup>, protein 83 mg/dl, and glucose 70 mg/dl. IgG oligoclonal band was negative but myelin basic protein was increased (9.5 ng/ml; normal less than 4). Visual evoked potentials (VEPs) with pattern reversal checkerboard were poorly elicited, and P100 latencies were undetectable in response to each eye stimulation. Magnetic resonance imaging (MRI) showed white matter mass lesions in the bilateral frontal and occipital lobes and widespread involvement of the corpus callosum. The occipital lesions extended to the occipitotemporal junction (Fig. 1). Single-photon emission computed tomography (SPECT) demonstrated a decrease in cerebral blood flow (CBF) in the left occipital lobe, spreading to the left occipitotemporal region (Fig. 2A). A three-dimensional surface display generated from SPECT data (21, 22) revealed hypoperfusion in the left peristriate cortex (Fig. 2B). She underwent brain biopsy of the frontal lesion, which revealed demyelination and

gliosis. Since she was diagnosed as having clinically definite MS (23), a large dose of methylprednisolone (500 mg daily) was administered intravenously for 3 days, followed by oral prednisolone. In October 1993, her visual acuity was 0.05 (0.3 with glasses) in the right eye and 0.04 (0.2 with glasses) in the left eye. Right-sided homonymous hemianopia was found. In May 1994, follow-up VEPs revealed normal P100 latencies to each eye stimulation (104.4 ms and 107.7 ms to right and left eye stimulations, respectively).

#### Neuropsychological examination

Between August 1993 and September 1993, the following neuropsychological observations were made. KK's speech was fluent with normal comprehension. Visual recognition was severely impaired. She could not name visually presented objects or read letters. The other language abilities were preserved except for impaired ability to write. She was unable to point to objects named by the examiners and could not match materials to sample items by vision alone. However, her visual ability to judge dimension of lines and size of objects was relatively preserved. When she handled materials, they were more properly recognized, but not perfectly. By hearing characteristic sounds such as bells or clocks, she could easily identify the objects. Copying of line drawings was impaired with either the right or the left hand. Recognition of faces and colors was also defective. Voluntary movements of the right hand were clumsy due to proprioceptive sensory disturbance. She could manipulate objects with the left hand. Details of neuropsychological assessment were carried out between October 1993 and August 1994, the results of which follow.

#### Intelligence and language

In March 1994, the Wechsler Adult Intelligence Scale (WAIS) score was 92 for the verbal tests. No evidence of aphasia was found except for impairment of reading and writing.

#### Praxis

In October 1993, ideomotor apraxia was evident when using the left hand. Praxic disorders of the right hand could not be precisely assessed due to impairment of proprioception. KK could make fine finger movements or manipulate objects with the left hand, but not with the right hand. In response to verbal command, however, she could not correctly perform gestures such as waving goodbye with the left hand. The erroneous pantomimes

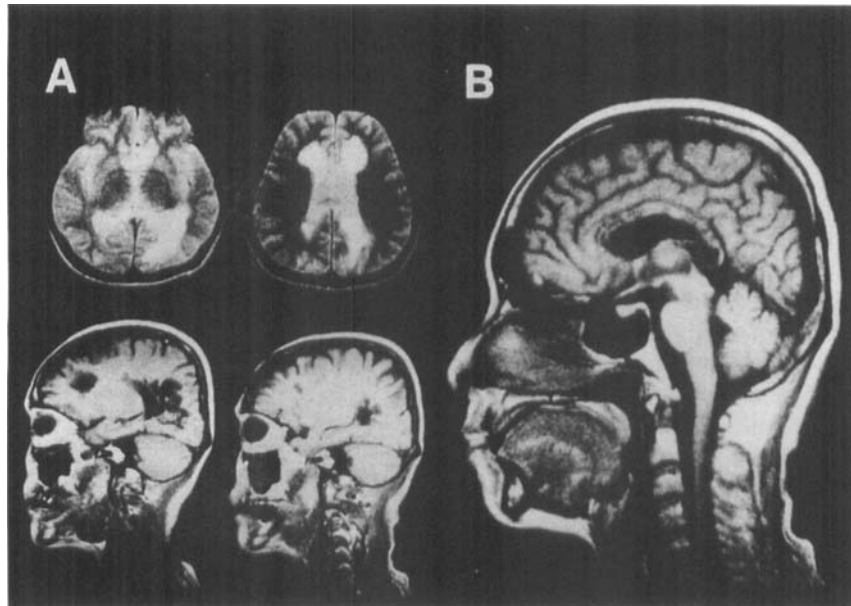


Fig. 1. Axial T2 weighted MRI showing white matter mass lesions in the bilateral frontal and occipital lobes (A top: left/right reversed). Sagittal T1 weighted MRI showing bilateral white matter lesions extending to the occipitotemporal junction (A bottom: left and right images showing the left and right hemispheres, respectively), and many lesions in the corpus callosum (B).

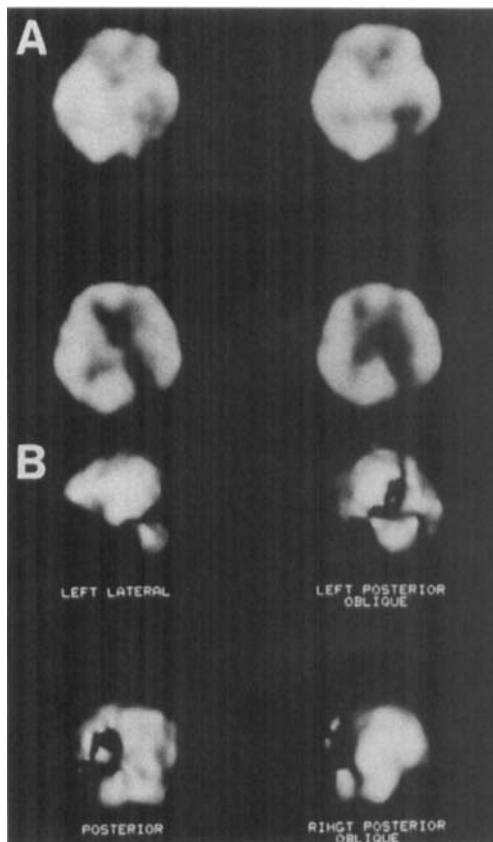


Fig. 2. SPECT early images showing a decrease in cerebral blood flow in the left occipital lobe and occipitotemporal region (A: left/right reversed). Three-dimensional surface display (55% threshold value of the global maximum counts) showing hypoperfusion in the left peristriate cortex (B).

were not improved by imitating. Despite the clumsiness of the right hand, she performed gestures more easily with the right hand than with the left hand. Her left-sided ideomotor apraxia was compatible with callosal apraxia (20, 24).

#### Visual recognition

A) *Real objects* – To assess abilities of object recognition, fifteen items were selected. Each object was placed on a table in front of KK. She was asked to identify them by vision alone on three separate occasions between October 1993 and July 1994. The number of correct responses ranged from 60 to 80% during that period (Tables 1, 2). In March 1994, she could, in part, verbally describe the features of materials that were not named (e.g. a description of a spoon as “slender – having a head”), and she could identify objects by touch that were not visually identified.

B) *Line drawings* – Eight line drawings were presented after assessment of real object identification. KK’s failure of perception of line drawings was more severe than that of perception of real objects. The rate of correct responses was still only 38% in July 1994 (Tables 1, 2). Frequently, she could not identify line drawings of real objects that were correctly named. There was a tendency of perseveration. Meaningful overlapping tests consisting of four overlapping drawings (Poppelreuter) demonstrated her severe deficits.

Table 1. Object recognition

	Oct 1993	Mar 1994	July 1994
Naming of real objects	9/15	11/15	12/15
Naming of line drawings	2/8	3/8	3/8
Naming of overlapping figures	1/4	1/4	2/4
Object-object matching	4/10	9/10	10/10

C) *Object-object matching* – KK was requested to match a real object to sample items consisting of three objects including the target object. The three items were of similar size. For example, the alternatives of a pencil were a fountainpen and a tooth brush. On this test, she showed remarkable improvement and performed it completely in July 1994 (Table 1).

D) *Photographs* – KK's ability to recognize photographs of objects and animals was most severely impaired in visual recognition tasks. The photographs contained 12 familiar objects and animals. She could not identify any of the photographic images even as late as in July 1994. She named correctly some of real objects such as a match or a pencil, but was unable to identify photographic images of these objects.

E) *Faces* – In October 1994, KK hardly recognized the faces of familiar persons, although she could identify them by voice. Her difficulty in face recognition gradually improved. In July 1994, she recognized faces of the hospital staff and relatives almost correctly, and matched 75 percent of photographs of famous people correctly.

F) *Colors* – From October 1993 to August 1994, KK's ability to name colors remained defective. She could not correctly point to colors named by the examiner. The rate of correct response was 2/8 in October 1993 and 5/8 in August 1994. Yet, she performed comfortably verbal-verbal tasks (e.g. What color is a banana?) or color matching.

Drawing

In August 1993, KK's difficulty in visual perception was so severe that she could not copy real objects. She copied only simple figures such as a cross, a circle, and a triangle in inaccurate manners. Her deficits of drawing improved to some extent. She drew objects more accurately with the left hand than with the right hand, due to clumsiness of the right hand. In August 1994, her drawing from memory was more defective, compared with copying real objects or line drawings (Fig. 3).

Table 2. Object recognition and responses

	Nov 1993	Mar 1994	July 1994
<b>Real objects</b>			
ball	○	○	○
gun	match	○	○
screwdriver	X	X	key
match	○	○	○
hammer	○	○	○
tooth brush	○	○	cap of bottle
cap opener	○	○	○
scissors	○	○	○
spoon	tooth brush	X	○
clock	○	bell	○
pencil	○	○	○
harmonica	comb	○	○
comb	○	○	○
cup	X	eraser	○
lighter	X	○	X
<b>Line drawings</b>			
hammer	X	○	○
key	○	X	○
comb	chimney	X	match
spoon	smoke	tooth brush	lighter
pencil	pipe	○	match
cup	smoke	clock	pan
candle	chimney	lighter	fire
ball	○	○	○

A circle indicates KK's correct response, and a cross indicates her inability to name.

Description from memory

KK was able to generate accurate verbal descriptions of object's characteristics and define differences between pairs of objects or animals (e.g. an elephant and a rabbit) fairly well. She described an electric fan as a round bowl and circling long arms, and an elephant as having a long nose and four big legs even in October 1993, when she was unable to identify line drawings or photographs of either the electric fan or the elephant. In addition, she answered correctly the functions of materials and living objects.

Reading and writing

A) *Reading* – In comparison with KK's disabilities of visual object recognition, her ability to read letters and words was comparatively preserved. In October 1993, she read appropriately 80% of Kanji

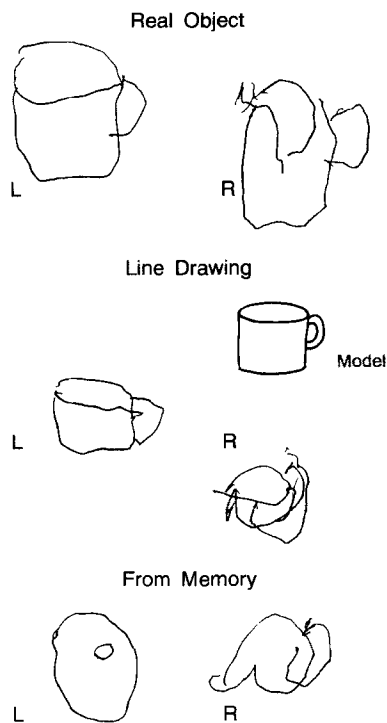


Fig. 3. Drawing of a real cup (top), its line drawing (middle), and from memory (bottom). L: left hand, R: right hand.

letters and 65% of Kana letters without a letter-by-letter manner.

B) *Writing* – In November 1993, KK could purposively write her name with the right hand, but not with the left hand. In July 1994, she could spell several letters and words to dictation with the right hand, while her writing with the left hand still remained quite defective. Her deficits of writing was attributable to callosal agraphia (20, 25).

### Discussion

The case of KK demonstrated unique clinical features and recovery course of visual agnosia during one year follow-up. Initially, her deficits of visual identification were quite severe, resembling a case reported by Benson (13). She presented with a profound failure of visual recognition when performing tasks of naming, matching, and copying. Even if her elementary visual functions such as visual acuity and visual field were impaired, the deficits could not completely account for her visual recognition failure, since she could respond to visual stimuli and reach visually presented objects. Above all, she had primary visual competence enough to look at objects with glasses when precise neuropsychological examinations were done. One year later, her abilities of visual recognition improved to some extent, but still remained agnos-

tic, particularly at the stage of visual form perception (visual form agnosia). She could identify real objects more easily than line drawings or photographs. This indicates that surface properties and depth can assist KK's visual perceptual failure (26). Her visual recognition deficits appear to have occurred at the early substage of integrating perceived components of a stimulus. According to Marr's 3-D model (27), KK had difficulty in deriving a 3-D sketch in which a volumetric representation of a stimulus is constructed.

The most striking finding was improved ability of matching, while her copying of real objects or line drawings still remained defective. Both matching and copying are traditionally used to assess the integrity of visual perception (3). In the recovery course of apperceptive visual agnosia, as is the case for associative visual agnosia, patients perform comparatively on matching and copying tasks (7, 9). The dissociation between KK's abilities of matching and copying raises a question as to whether both of these functions share entirely the same perceptual processes. Intact matching requires a correct discrimination of shape, and intact copying requires a correct perception of its global configuration and the spatial arrangement of its part (7). Namely, copying requires greater abilities for visuospatial orientation than does matching. KK's disability to copy may, at least in part, be attributable to impairment of visuomotor processing of orientation information (14). Object recognition appears to be influenced by spatial attention (10). It seems likely that in visual form agnosics disorders of perceptual process can affect the abilities to copy and match independently. In the strict sense, even associative agnosics show abnormal abilities to copy on occasions (10, 11). These facts are consistent with the idea that both the apperception and the association stages may themselves be divided into various substages (9–11).

The other interesting finding was the dissociation between KK's describing and drawing from memory. She performed fairly well on the former task but poorly on the latter task. Some authors have reported poor long-term memory or a disturbance of drawing objects from memory in visual form agnosics (7, 9, 28, 29). These deficits were attributable to impoverishment of stored visual images or a loss of access to visual representations. Yet, KK's preserved ability to describe the characteristics of objects and animals indicates intact stored knowledge about their appearance. She was able to access semantic knowledge about objects via a verbal route, but could barely access it via a visual route. Conversely, a case with visual form agnosia has been reported to retain greater ability to draw objects from memory than real objects (26). Thus,

damage to the image generating process is not uniform among visual form agnostics. The inconsistent deficits may arise from complex patterns of damage to visual information processing that range from the stage of inspecting the visual buffer to the stage of reproducing the shape of objects (29). KK exhibited more difficulty in transferring visual images to drawings than in copying real objects. It appears that her visual recognition defects occurred not only at the early substage of form perception but also at the stage of reproducing the shape of objects from visual memory store.

In the past, visual form agnostics, classified as apperceptive type, were associated with lesions in the bilateral striate and prestriate cortices, so these cortical regions were considered to be concerned with perceptual process of visual form recognition (13). Within the last decade, however, the contributions of the other cortical regions to visual perception have been clarified (12, 30). Some authors have indicated that damage to the other cortical areas such as the parietal and temporal cortices, even on the unilateral side, can induce visual form agnosia (31). Experimental studies have revealed three parallel pathways, i.e. magnocellular, parvocellular-interblob, and parvocellular-blob systems, that process distinct visual information (12). Clinical reports of the patient, DF, suggested that visual form agnosia was attributable to a selective damage to the magnocellular visual system (14, 32). We could not confine responsible lesions for KK's deficits to a single visual pathway. Rather, we would like to explain her deficits by the theory of two separate visual information processings proposed by Goodale & Milner (33). According to their hypothesis, the ventral stream, projecting from the striate cortex (V1) to the inferior temporal cortex (TE), plays a major role in identifying objects, and the dorsal stream, projecting from V1 to the posterior parietal cortex (PG), is specialized for spatial perception. Each stream receives inputs from both the magno and parvo pathways. KK appears to have suffered mainly from damage to the ventral stream. Her white matter lesion involved the left peristriate areas, encompassing the striate cortex. The white matter lesion in the right occipital lobe was smaller, but the splenium of the corpus callosum was also involved. Both occipital lesions extended to the occipitotemporal junction. These white matter lesions can disrupt the segregated connectivity patterns of hierarchical visual processing that exist in the occipitotemporal cortex as well as in the striate cortex (34, 35). Recovery of P100 response in pattern-reversal VEPs indicates sparing of V1 but damage to the neural circuits originating from V1 (14, 36). In fact, her white matter lesions on

MRI extended to the occipitotemporal junction without involvement of the striate cortex, and CBF was decreased in both the left peristriate cortex and occipitotemporal region. Associated prosopagnosia and achromatopsia could also occur following lesions in the occipitotemporal white matters (29, 37). In KK, therefore, interruption of the occipitotemporal connections may have involved both the magnocellular and parvocellular systems, resulting in impaired recognition of objects, faces, and colors.

White matter lesions can affect higher cortical functions, presumably due to remote effect or diaschisis (22). In addition to visual form agnosia, KK presented with callosal syndromes including apraxia and agraphia in the left hand. These signs of callosal disconnection are very rare in MS, although the corpus callosum is frequently involved in MS (20, 38). KK's neighboring white matter lesions in connection with the corpus callosum lesions may have contributed to the development of ideomotor apraxia and agraphia in the left hand (20). The clinical features of this case are unique in that white matter lesions induced multi-modal disorders of higher cortical functions including recognition, praxis, and language. Although a possibility of some damage to the cortex could not be strictly excluded, we believe that the white matter lesions contribute mostly to the higher cortical dysfunctions.

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