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A case of prosopometamorphopsia caused by infarction of the splenium of the corpus callosum and major forceps

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ABSTRACT

An adult female complained of enlargement of right eyes in other people. Diffusion-weighted imaging detected an abnormal high-intensity area in the region from the splenium of the corpus callosum to the major forceps on the right side. The patient reported that right eyes appeared larger in size, which suggested prosopometamorphopsia. Adichotic listening test identified left-ear deficit. Acombination of prosopometamorphopsia and left-ear deficit was not identified in the reported patients. Prosopometamorphopsia in most of the reported patients included the eye as did that in our patient. This result suggested the importance of information on the eye in recognizing faces.

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KEYWORDS

Prosopometamorphopsia; the splenium of the corpus callosum; the major forceps; infarction; left-ear deficit

Introduction

Metamorphopsia is a phenomenon where visual objects appear to be modified to the perceiver (Hishizawa et al., 2015; Lee, 2015; Saito et al., 2014; Tokita & Tagawa, 2014; Uchiyama et al., 2012). Metamorphopsia limited to features of faces is called prosopometamorphopsia (Barghouthi & El Husseini, 2018; Lee, 2015; McCarty et al., 2017; Saito et al., 2014). Information from the faces of other people includes expressions and the arrangement of facial components such as eyes, nose, and mouth, and are highly important for perceivers (Nagaishi et al., 2015). In the cerebrum, the ventral occipitotemporal cortex plays a crucial role in processing information from faces (Barghouthi & El Husseini, 2018; Hishizawa et al., 2015; Saito et al., 2014; Schroeder et al., 2017). The right cerebrum is considered to be dominant in processing information from faces; however, conveyance between each cerebrum through the corpus callosum is necessary for the integration of information from faces in each cerebrum to recognize faces precisely (Hishizawa et al., 2015; Nagaishi et al., 2015; Saito et al., 2014). Prosopometamorphopsia has been reported in patients with lesions of the corpus callosum and nearby regions (Amano et al., 2014; Barghouthi & El Husseini, 2018; Cho et al., 2011; Ebata et al., 1991; Funatsu et al., 2017; Ganssauge et al., 2012; Hishizawa et al., 2015; Imai et al., 1995; Katsura et al., 2010; Kobayashi et al., 2013; Lee, 2015; McCarty et al., 2017; Nagaishi et al., 2015; Saito et al., 2014; Schroeder et al., 2017; Tokita & Tagawa, 2014; Uchiyama et al., 2012). We report here a female patient who showed prosopometamorphopsia in addition to left-ear deficit, which is a symptom of disconnection syndrome (Kasahata et al., 1999; Landry & Fuente, 2017; Otsuki., 2009; Van der Knaap & van der

Ham, 2011), caused by infarction of a region from the splenium of the corpus callosum to the major forceps.

Case description

A 73-year-old right-handed woman noted an acute onset of the right eyes of other people appearing larger than left eyes and was admitted to our hospital the next day. Hypertension and ventricular arrhythmia had been recorded in her past history. On admission, she was hypertensive (178/93 mmHg) and afebrile (36.5°C). Heart rate was regular at 73 beats per minute. General internal findings were normal. On neurological examination, she was alert and conscious and showed good orientation with regard to names, places, and dates. There were no abnormalities in the cranial nerves including the visual field and eye movements, muscle strength or coordination of the four extremities, deep tendon reflexes of the four extremities, or standing and gait. Babinski's sign was negative, as was a sensory disturbance. For high order functions, a visual perception test for agnosia noted no abnormality of fundamental functions of visual sensations, topographical orientation, or cognition of objects, faces, colors, symbols, visual fields. In sketches of the faces of other people with both eyes, the right eye was sketched at an enlarged scale compared with the normal left eye (Figure 1). The right eye was also seen similarly with one eye. The enlargement of the right eye disappeared with the presentation of the lateral side of the face. The degree of enlargement remained unchanged with the presentation of various proportions of the white of the eye and the iris. On the analysis of disconnection syndrome, alexia of the left visual field was not detected. A dichotic listening test was performed

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Figure 1. Sketch of faces of other persons. The right eyes of other persons were visualized with an enlarged size compared with the normal left eye. (Rt = right).



Figure 2. Dichotic listening test. A set of three successive numbers was presented through headphones as unilateral stimuli for 30 trials, then bilateral simultaneous stimuli were supplied the same time for 30 trials. With the unilateral stimuli, the rate of the correct answers was perfect (100%) in each ear. With the bilateral simultaneous stimuli, the correct answer was supplied at a rate of 93% for the right ear; however, for the left ear the rate was as low as 10%. This result suggested the presence of left-ear deficit.

as well (Figure 2). One stimulation consisted of the sounds of three successive numbers. Unilateral stimuli were presented in each ear (contralateral side; noise) for 30 trials each. Bilateral simultaneous stimuli were then applied for 30 trials. The answer rate of each ear for the unilateral stimuli was 100%. With the bilateral simultaneous stimuli, the answer rate for the left ear was decreased to 10%, whereas that of the right ear remained at a high rate of 93%. This result suggested the presence of leftear deficit (Figure 2). Routine blood hematology and biochemistry were normal except for a mild increase of cholinesterase. The cardiac ultrasonography and electrocardiogram were normal. The carotid artery ultrasonography showed mild atherosclerosis of the bilateral internal carotid arteries, not accompanying stenosis. Diffusion-weighted magnetic resonance imaging (MRI) on admission showed a small highintensity area in the region from the splenium of the corpus callosum to the major forceps on the right side (Figure 3). The head magnetic resonance angiography was normal. Based on the MRI finding, the patient was diagnosed as having cerebral infarction. Intravenous administration of the ozagrel sodium



Figure 3. Brain diffusion-weighted magnetic resonance imaging. An abnormal high-intensity area was shown in the region from the splenium of the corpus callosum to the major forceps on the right side.

(160 mg/day) and the edaravone (60 mg/day) was performed in 14 days. Prosopmetamophosia gradually subsided and disappeared at the tenth hospitalized day after admission. After the therapy of the two drugs, oral administration of aspirin (100 mg/day) was performed.

Discussion

The corpus callosum is divided into the genu, truncus, and splenium from the anatomical perspective (Otsuki., 2009). Function-anatomically, the corpus callosum is divided into the anterior third, anterior midbody, posterior midbody, isthmus, and splenium (Otsuki., 2009). The splenium is mainly supplied with the posterior pericallosal artery originating from the posterior cerebral artery (Goto, 1986). The small infarct was detected in the distribution of the posterior pericallosal artery in the patient; however, the results of its location and the neurological symptoms were inconsistent with the characteristics of small-artery occlusion in the "Trial of Org 10172 in Acute Stroke Treatment (TOAST)" classification (Adams et al., 1993). Thus, the etiology of infarction in the patient was grouped into the stroke of undetermined etiology in the TOAST classification (Adams et al., 1993) because the cause of infarction was indefinite.

Metamorphopsia is a condition in which the size, contour, shape, color, depth, direction, or number in objects are altered for perceivers (Hishizawa et al., 2015; Lee, 2015; Saito et al., 2014; Tokita & Tagawa, 2014; Uchiyama et al., 2012). Prosopometamorphopsia is a metamorphopsia that is limited to faces (Barghouthi & El Husseini, 2018; Lee, 2015; McCarty et al., 2017; Saito et al., 2014). The ventral occipitotemporal cortex, which is called the core system (Lee, 2015), is crucial to facial recognition (Barghouthi & El Husseini, 2018; Hishizawa et al., 2015; Saito et al.,

2014; Schroeder et al., 2017). The core system consists of three regions; i.e., the occipital face area, the fusiform face area, and faceselective regions in the posterior superior temporal sulcus (Lee, 2015), and plays a central role in the processing facial recognition (Hishizawa et al., 2015; Lee, 2015; Saito et al., 2014; Schroeder et al., 2017). Recent studies suggested that information on faces in each cerebrum is necessitated in the process of facial recognition with no association with the dominance of the cerebrum and handedness (Nagaishi et al., 2015; Tokita & Tagawa, 2014). Based on this explanation, information on the right part of the face is predominantly projected to the right cerebrum (Funatsu et al., 2017; Nagaishi et al., 2015; Saito et al., 2014). On the other hand, information on the left part of the face is predominantly projected to the left cerebrum (Funatsu et al., 2017; Nagaishi et al., 2015; Saito et al., 2014). Information on faces in the core system is projected to the other cerebrum through the corpus callosum and then integrated to recognize faces (Lee, 2015; Steinmann et al., 2014). In prosopometamorphopsia caused by lesions in the corpus callosum and nearby regions (Amano et al., 2014; Barghouthi & El Husseini, 2018; Cho et al., 2011; Ebata et al., 1991; Funatsu et al., 2017; Ganssauge et al., 2012; Hishizawa et al., 2015; Imai et al., 1995; Katsura et al., 2010; Kobayashi et al., 2013; Lee, 2015; McCarty et al., 2017; Nagaishi et al., 2015; Saito et al., 2014; Schroeder et al., 2017; Tokita & Tagawa, 2014; Uchiyama et al., 2012), disruption of facial information in each cerebrum is considered as the cause of prosopometamorphopsia (Funatsu et al., 2017; Hishizawa et al., 2015; Lee, 2015; Nagaishi et al., 2015; Saito et al., 2014; Tokita & Tagawa, 2014). Thus, prosopometamorphopsia of the right-sided face is caused by a lesion in the right cerebrum (Saito et al., 2014; Uchiyama et al., 2012), and that of the left-sided face is caused by a lesion in the left cerebrum (Funatsu et al., 2017; Saito et al., 2014; Uchiyama et al., 2012). Information on the face in each cerebrum was considered to be impaired by a lesion in the splenium of the corpus callosum, which leads to the incidence of prosopometamorphopsia in our patient. Prosopometamorphopsia caused by lesions in the corpus callosum showed simple deformations compared with that caused by lesions in the cerebrum in general (Nagaishi et al., 2015; Uchiyama et al., 2012) and included deformation of the eyes in a high rate (Funatsu et al., 2017). In particular, information on the region of eyes is considered to be highly important in recognizing faces (Funatsu et al., 2017; Nagaishi et al., 2015). Prosopometamorphopsia in our patient was a simple deformation and was limited to the region of the eye. The laterality of the lesion was consistent with that of prosopometamorphopsia. These results were consistent with those of prosopometamorphopsia caused by lesions of the corpus callosum (Funatsu et al., 2017; Nagaishi et al., 2015; Saito et al., 2014; Uchiyama et al., 2012).

Eighteen patients have been reported who showed prosopometamorphopsia caused by lesions in the corpus callosum and its nearby regions (Amano et al., 2014; Barghouthi & El Husseini, 2018; Cho et al., 2011; Ebata et al., 1991; Funatsu et al., 2017; Ganssauge et al., 2012; Hishizawa et al., 2015; Imai et al., 1995; Katsura et al., 2010; Kobayashi et al., 2013; Lee, 2015; McCarty et al., 2017; Nagaishi et al., 2015; Saito et al., 2014; Schroeder et al., 2017; Tokita & Tagawa, 2014; Uchiyama et al., 2012). These 18 patients and our patient are listed in Table 1. Overall, 13 patients were right-handed including our patient (Barghouthi & El Husseini, 2018; Cho et al., 2011; Ebata et al., 1991; Funatsu et al., 2017; Hishizawa et al., 2015; Katsura et al., 2010; Kobayashi et al., 2013; Lee, 2015; McCarty et al., 2017; Nagaishi et al., 2015; Tokita & Tagawa, 2014), and 1 patient was left handed (handedness not reported for the other 5 patients) (Saito et al., 2014). Diagnosis of lesions was infarction in 16 patients including our patient (Amano et al., 2014; Barghouthi & El Husseini, 2018; Cho et al., 2011; Funatsu et al., 2017; Hishizawa et al., 2015; Imai et al., 1995; Katsura et al., 2010; Lee, 2015; McCarty et al., 2017; Nagaishi et al., 2015; Saito et al., 2014; Schroeder et al., 2017; Tokita & Tagawa, 2014; Uchiyama et al., 2012), resection of arteriovenous malformation in one patient (Ganssauge et al., 2012), hemorrhage in one patient (Ebata et al., 1991), and hypoglycemia in one patient (Kobayashi et al., 2013). Lesions were located on the right side in nine patients including our patient (Cho et al., 2011; Funatsu et al., 2017; Hishizawa et al., 2015; Nagaishi et al., 2015; Saito et al., 2014; Schroeder et al., 2017; Tokita & Tagawa, 2014), and the left side in nine patients (Amano et al., 2014; Barghouthi & El Husseini, 2018; Cho et al., 2011; Ganssauge et al., 2012; Imai et al., 1995; Katsura et al., 2010; Lee, 2015; McCarty et al., 2017; Uchiyama et al., 2012). The lesion in one patient with hypoglycemia involved the bilateral splenium (Kobayashi et al., 2013). The lesions involved in the splenium in 13 patients including our patient (Amano et al., 2014; Barghouthi & El Husseini, 2018; Cho et al., 2011; Funatsu et al., 2017; Hishizawa et al., 2015; Katsura et al., 2010; Kobayashi et al., 2013; Lee, 2015; McCarty et al., 2017; Nagaishi et al., 2015; Saito et al., 2014) and the retrosplenium in four patients (Ganssauge et al., 2012; Imai et al., 1995; Tokita & Tagawa, 2014; Uchiyama et al., 2012). The lesion in one patient involved either (Ebata et al., 1991), and that in one patient was located in the major forceps and did not involve the splenium or the retrosplenium (Schroeder et al., 2017). No patients had noted prosopagnosia or visual field defects. Prosopometamorphopsia was present in the lateral side in 18 patients (Amano et al., 2014; Barghouthi & El Husseini, 2018; Cho et al., 2011; Ebata et al., 1991; Funatsu et al., 2017; Ganssauge et al., 2012; Hishizawa et al., 2015; Imai et al., 1995; Katsura et al., 2010; Kobayashi et al., 2013; Lee, 2015; Nagaishi et al., 2015; Saito et al., 2014; Schroeder et al., 2017; Tokita & Tagawa, 2014; Uchiyama et al., 2012); otherwise, laterality of prosopometamorphopsia that was limited to the mouth was not shown in one patient (McCarty et al., 2017). In comparison with each laterality of prosopometamorphopsia and the lesion, the laterality was the same in 12 patients including our patient (Lee, 2015; Uchiyama et al., 2012; Saito et al., 2014; Barghouthi & El Husseini, 2018; Nagaishi et al., 2015; Schroeder et al., 2017; Ebata et al., 1991; Imai et al., 1995; Cho et al., 2011; Ganssauge et al.; Amano et al., 2014) and opposite in 5 patients (Cho et al., 2011; Funatsu et al., 2017; Hishizawa et al., 2015; Katsura et al., 2010; Tokita & Tagawa, 2014). For the form of prosopometamorphopsia, various types of deformation were exhibited in all 19 patients (Table 1); however, other types were infrequent and included brightness in 2 patients (Ganssauge et al., 2012; Imai et al., 1995) and doubling in 1 patient (Nagaishi et al., 2015). These results on the laterality and contents of prosopometamorphopsia were consistent with the previously reported characteristics (Funatsu et al., 2017; Lee, 2015; Nagaishi et al., 2015; Uchiyama et al., 2012). For the extent of prosopometamorphopsia, the region of the eye was

authors	/ane	-babded-	prosopometamorphopsia	other metamorphopsia _{ni}	ocono- visua	field	ther neurological	disconnection	locations of	diagnosis of
(years)	sex	ness	region (contents)	objects (contents) pi	ignosia def	ect	findings	syndrome	lesions	lesions
Ebata et al. (1991)	60/f	ť	rt entire face (micropsia), facial	I	I		oss of superficial	I	rt retrosplenium	hemorrhage
lmai et al. (1995)	51/f	n.d.	outline (distortion) It entire face (dropped and darkened)	lt side of objects and	I		ensation in it face. -	I	to spienium boundary of It CG and retrosplenium	infarction
				numbers (blurred)					-	
Katsura et al. (2010)	67/f	t	rt entire face (distorted)	I	I		I	Ι	lt splenium	infarction
Cho et al. (2011)	58/f	t	It eye (elongated outward), nose and mouth (bent downward)	I	I		I	I	lt splenium	infarction
Cho et al. (2011)	53/m	ť	It eyelid (swollen), nose (bent downward), facial outline (bulged and writhod)	I	I		I	Ι	rt area and midline of splenium	infarction
Ganssauge et al. (2012)	68/f	n.d.	tt eye (distortion, micropsia, brightened)	I	I		I	Ι	lt retrosplenium to o/t iunction	resection of AVM
Uchiyama et al. (2012)	57/m	n.d.	It entire face (stretched outward)	It side of objects (stretched outward)			I	I	lt retrosplenium	infarction
Kobayashi et al. (2013)	29/f	ť	It upper face including eye		I	- overs	ight (It letters, rt lines and ficures	alexia of It visual field	bil splenium	hypo- dlvremia
Saito et al. (2014)	61/f	Ħ	rt entre face (distorted, drooped,	I	I				rt splenium to maior forcens	infarction
Tokita and Tagawa (2014)	75/m	Ľ	anotened) It eye and eyebrow (dropped, micropsia)	I	I		I	Ι	rt retrosplenium	infarction
Amano et al. (2014)	67/m	n.d.	It jaw and outer canthus (distorted)	I			I	I	lt splenium	infarction
Nagaishi et al. (2015)	78/f	ť	rt eye (micropsia, macropsia, doubled), eyebrow (cut off, blink), nostril (micropsia), corner of mouth (raised)	rt side of objects (doubled, deepened)			I	1	rt splenium	infarction
Hishizawa et al. (2015)	70/f	ť	It eye and corner of mouth (hung	I	I		I	Ι	rt splenium	infarction
Lee (2015)	52/f	ť	It eye (micropsia), lip and cheek (swollen)	I	I		I	I	lt splenium	infarction
McCarty et al. (2017)	62/m	t	mouth (distorted, enlarged)	I	- slig dis Ge	htly corted ntral	TGA (3 years before)	alexia (letter /number)	lt splenium	infarction
Schroeder et al. (2017)	58/f	n.d.	rt entire face (back-shifted, deformed)	I	1		I	I	rt posterior CG, major forceps	infarction
Funatsu et al. (2017) Barghouthi and El	66/f 67/f	דד	It eye (adducted) It eye (moved to the outside and	1 1	1 1		1 1	1 1	rt splénium It splenium	infarction infarction
Husseini (2018) Our case	73/f	Ľ	upward) rt eye (enlarged)	I	I		I	left-ear deficit	rt splenium to major forceps	infarction
The impaired side includiin not described, o/t: occip	ng metai vitotemp	norphop oral, rt: ri	sia describing the laterality of the object or person oth. TGA: transient clobal amnesia. * This sympto	. Abbreviations; AVM: ar m was caused by an old	teriovenous ma	lformation, bil	: bilateral, CG: cingulate he richt side. ** This svr	gyrus, CC: corpus ca notom was associat	allosum, f: female, lt: lef red with tortuosity of th	:, m: male, n.d.: e optic nerves.

Table 1. Nineteen patients with prosopometamorphopsia caused by lesions in the corpus callosum and its nearby regions.

included in 17 patients including our patient (Barghouthi & El Husseini, 2018; Cho et al., 2011; Ebata et al., 1991; Funatsu et al., 2017; Ganssauge et al., 2012; Hishizawa et al., 2015; Imai et al., 1995; Katsura et al., 2010; Kobayashi et al., 2013; Lee, 2015; Nagaishi et al., 2015; Saito et al., 2014; Schroeder et al., 2017; Tokita & Tagawa, 2014; Uchiyama et al., 2012). In particular, sole involvement of the eye was shown in five patients including our patient (Barghouthi & El Husseini, 2018; Funatsu et al., 2017; Ganssauge et al., 2012; Tokita & Tagawa, 2014). This result suggested the importance of information from the region of eyes in recognizing faces (Funatsu et al., 2017; Hishizawa et al., 2015; Nagaishi et al., 2015).

Disconnection syndrome is caused by lesions in the corpus callosum (Kasahata et al., 1999; Otsuki., 2009). Lesions in the splenium of the corpus callosum often cause alexia and anomia of the left visual field and left-ear deficit (Kasahata et al., 1999; Landry & Fuente, 2017; Otsuki., 2009; Van der Knaap & van der Ham, 2011). Left-ear deficit indicates the presence of verbal extinction of the left ear and is also called the right-ear advantage (Van der Knaap & van der Ham, 2011). This phenomenon is confirmed by a dichotic listening test (Landry & Fuente, 2017; Otsuki., 2009). In the 19 patients (Table 1), 2 patients noted alexia (Kobayashi et al., 2013; McCarty et al., 2017). Alexia in one patient was noted in the left visual field (Kobayashi et al., 2013), although the visual field of alexia in another patient was not identified (McCarty et al., 2017). A combination of prosopometamorphopsia and left-ear deficit was identified in our patient alone among the 19 patients (Table 1). In the auditory pathway (Figure 4) (England & Wakely., 1991), the hair cells in the cochlea are innervated by bipolar neurons whose cell bodies are located in the spiral ganglion (England & Wakely., 1991). Neurofibers of bipolar neurons run in the cochlear division of the vestibulocochlear nerve (VIII) in the pontomedullary junction. On entering the brainstem, the neurofibers separate into the dorsal cochlear nucleus and the ventral cochlear nucleus. Some neurofibers from the two nuclei ascend in the lateral lemniscus through the superior olivary nucleus on the ipsilateral side and then approach the inferior colliculus. On the other hand, other neurofibers from the ventral cochlear nucleus decussate through the trapezoid body and join the decussated neurofibers from the dorsal cochlear nucleus. These joined neurofibers ascend in the lateral lemniscus, and then approach the inferior colliculus. From the nucleus of the inferior colliculus, neurofibers approach the medial geniculate body through the brachium of the inferior colliculus, and then the next neurofibers in the auditory radiation finally approach the primary auditory cortex. Some neurofibers decussate at the level of the nuclei of the lateral lemniscus and the inferior colliculus (England & Wakely., 1991). Accordingly, each auditory cortex receives auditory information from the bilateral ears (England & Wakely., 1991). Furthermore, each primary auditory cortex is connected to one another through the posterior third, isthmus, and splenium in the corpus callosum (Figure 4) (England & Wakely., 1991; Otsuki., 2009). With unilateral sound stimuli, auditory information is processed bilaterally; however, with



Figure 4. The auditory pathway from the cochlea (modified from the original figure by England MA and Wakely J (England & Wakely., 1991)). The first neurofibers arising from the cochlea approach the ventral and dorsal cochlear nuclei in the pontomedullary junction. Part of the neurofibers from the two nuclei ascend in the ipsilateral lateral lemniscus. The other neurofibers from the two nuclei decussate in the pontomedullary junction and ascend in the contralateral lateral lemniscus. The other neurofibers from the two nuclei decussate in the pontomedullary junction and ascend in the contralateral lateral lemniscus. The neurofibers on both sides approach the primary auditory cortex through the inferior colliculus and the medial geniculate body. Each of the primary auditory cortex receives neurofibers from the opposite cortex through the corpus callosum. Some neurofibers decussate at the nuclei of the lateral lemniscus and the inferior colliculus.

the bilateral simultaneous stimuli, the contralateral (decussated) auditory pathway becomes dominant because the ipsilateral auditory pathway is suppressed (Otsuki., 2009; Van der Knaap & van der Ham, 2011). Furthermore, with the bilateral simultaneous stimuli of different verbal sounds, the left cerebrum becomes dominant because the left cerebrum possesses the function to discriminate verbal sounds (Otsuki., 2009; Van der Knaap & van der Ham, 2011). In this process, verbal sounds from the right ear are easy to discriminate because they enter the left primary auditory cortex directly (Otsuki., 2009; Van der Knaap & van der Ham, 2011). On the other hand, verbal sounds from the left ear enter the right primary auditory cortex, then they next approach the left primary auditory cortex through the corpus callosum. However, in cases with dysfunction of the corpus callosum, it is hard for verbal sounds from the left ear to approach the left cerebrum because there is little conveyance in the corpus callosum (Otsuki., 2009; Van der Knaap & van der Ham, 2011). As a result, left-ear deficit can occur. In our patient, the conveyance of verbal sounds from the left ear was considered to be inhibited in the right part of the splenium of the corpus callosum, which caused left-ear deficit.

In conclusion, the highly importance of information from eyes was indicated in recognizing faces. Prosopometamorphopsia limited to the eye like our patient also supported this consideration. Left-ear deficit was identified, based on a dichotic listening test. These two symptoms suggested dysfunction of conveyance between each cerebrum through the corpus callosum.

Disclosure statement

The authors state they have no Conflicts of Interest (COI) to disclosure.

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