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Original

Parietal Ataxia : 13 Cases Plus a Review of Relevant Literature

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Abstract : Parietal ataxia is a rare condition of hemi- or mono-ataxia caused by contralateral lesions in the parietal lobe. Parietal ataxia is categorized into sensory ataxia and pseudocerebellar ataxia, and is usually reported in single-case studies. Here we report 13 cases of hemi- or mono-ataxia caused by acute brain infarction in the parietal lobe, and discuss these cases with respect to past case studies and the MRI lesion visualizations. Patients with motor paresis were excluded from this study. Patients with sensory disturbance were categorized into a sensory ataxia group and pseudocerebellar ataxia group. Lesions on cranial MRI were classified as white matter inferior to the posterior central gyrus, posterior central gyrus, or superior or inferior parietal lobules. Six patients in the sensory ataxia group showed lesions in the gray or white matter of the posterior central gyrus. We found that parietal ataxia patients showed a high rate of decomposition and slowness in the finger-nose test and decomposition in diadochokinesis. Oscillation in the finger-nose test and slowness in diadochokinesis were more frequently observed in the patients with sensory ataxia than in those with pseudocerebellar ataxia, although the difference was not lesion dependent. This study thus found clinical differences between the sensory ataxia and pseudocerebellar ataxia groups of patients with parietal ataxia.

Key words : parietal ataxia, sensory ataxia, pseudocerebellar ataxia, parietal lobe

Introduction

Here we discuss disconnection in patients with hemi- or mono-ataxia with lesions in the parietal lobe with a view to identifying the relevant clinical features and an underlying causative mechanism. In the book, "The Parietal Lobes", Macdonald Critchley[1] provides a clear description of ataxia linked to parietal lesion, describing two clear types. The first is proprioceptive or sensory ataxia caused by a loss of postural control, which is worse when the eyes are closed. The second is parietal ataxia without motor or sensory disturbance ; this type is difficult to distinguish from cerebellar ataxia, and it is thus known as pseudocerebellar ataxia. Studies of parietal ataxia usually involve only a few patients. Furthermore, none have reported the differences between sensory and pseudocerebellar ataxia. Magnetic resonance imaging (MRI) now enables us to compare lesions in patients with parietal ataxia, thus providing clearer diagnostic guidelines. In this paper we report 13 patients with parietal ataxia and analyze their clinical symptoms in relation to MRI findings.

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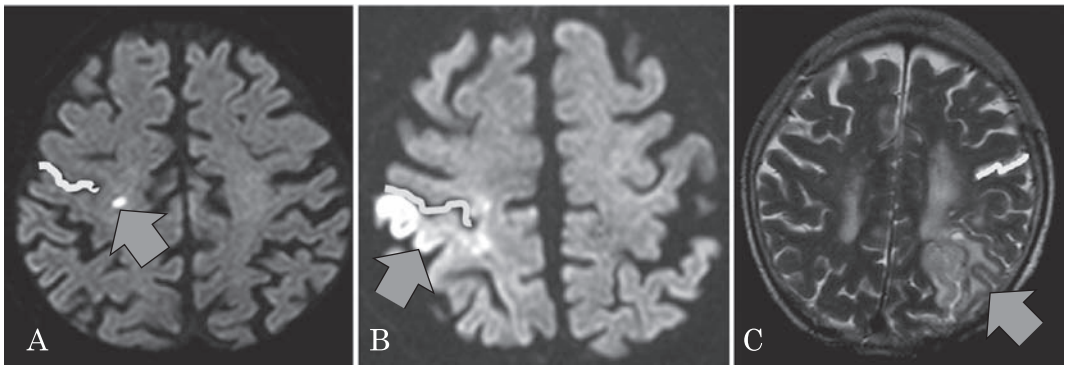

Fig. 1. MRI lesions

A: Pseudocerebellar ataxia linked to acute brain infarction was found in the white matter inferior to the right posterior central gyri. A lesion is shown on diffusion MRI (indicated with arrow).

B: Sensory ataxia linked to acute brain infarction in the right posterior central gyrus. A lesion is shown on diffusion MRI (indicated with arrow).

C: Pseudocerebellar ataxia, linked to acute brain infarction in the left angular gyrus. A lesion is shown on T2 MRI (indicated with arrow).

Yellow line: central sulcus.

Materials and methods

Thirteen patients were admitted to our hospital from 2002 to 2013 with hemi- or mono-ataxia caused by acute brain infarction in the parietal lobe. These cases showed no motor paresis, after-effects of previous strokes, or cognitive decline, enabling us to assess sensory disturbance.

We looked for motor paresis, muscle tonus, sensory disturbance, and ataxia on the day of admission or one day later, with at least two neurologists performing the neurological examinations. Ataxia was assessed using the finger-nose test, diadochokinesis, and heel-knee test. Symptoms of ataxia were categorized as decomposition, hypermetria, oscillation, and slow movement. We also examined whether ataxia was worse when the eyes were closed. To evaluate sensory disturbance, we examined pain, position sensation, graphesthesia, two-point discrimination sense, and stereognosis. Position sensation was examined by joint sensation and using the thumb localizing test, which provides one of the most sensitive measures of deep sensory disturbance[2]. MRI was performed for all patients at Showa University Hospital using a 1.5 T MRI.

Patients with sensory disturbances were placed into the sensory ataxia group, with the remainder placed in the pseudocerebellar ataxia group. We then compared performance. MRI lesions were categorized as: A) white matter inferior to the posterior central gyrus, B) posterior central gyrus, or C) superior or inferior parietal lobules (Fig. 1).

Results

Sensory disturbance

Six patients showed some sensory disturbance and were thus placed into the sensory ataxia

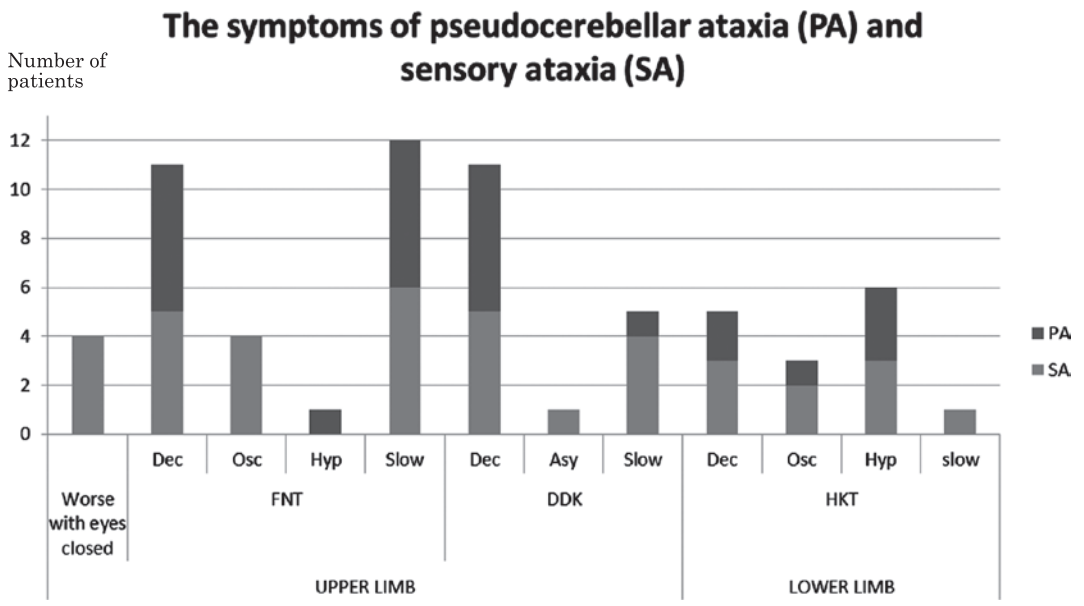

Fig. 2.

Dec : decomposition, Osc : oscillation, Hyp ; hypermetria, Slow : slowness, Asy : asynergy, FNT : finger-nose test, DDK : diadochokinesis, HKT : heel-knee test.

group. Of these, four had pain or two-point discriminative disturbance, three had position sensation disturbance, and two had graphesthesia or stereognosis.

Ataxia

Fig. 2 illustrates the ataxia, which was more frequently seen in the upper limb than the lower limb. Decomposition and slowness in the finger-nose test and decomposition by diadochokinesis were seen with a high frequency in all patients. The patients with sensory ataxia showed a greater variety of symptoms of ataxia than the patients with pseudocerebellar ataxia. Oscillation in the finger-nose test and slowness by diadochokinesis were seen more with sensory ataxia than with pseudocerebellar ataxia. Sensory ataxia was frequently worse with the eyes closed, unlike pseudocerebellar ataxia.

Correlation between parietal ataxia and lesions

The decomposition and slowness by finger-nose test and decomposition by diadochokinesis were not related to lesions in the parietal lobe. The patients with sensory ataxia had lesions in the gray or white matter of the posterior central gyrus (MRI lesion A or B). However, while oscillation by finger-nose test and slowness by diadochokinesis were seen more with sensory ataxia than pseudocerebellar ataxia, the symptoms were not lesion dependent.

Motor symptoms

Six patients showed the Wartenberg escape sign, but no clumsiness or limb-kinetic apraxia. The presence of the Wartenberg escape sign made no differences regarding the ataxia.

Table 1. Clinical features of parietal ataxia for 13 cases

	Cases		1	2	3	4	5	6	7	8	9	10	11	12	13
	Lesion		A				B						C		
Ataxia	Worse with eyes closed				+	+	+	+							
	Finger-nose test	Decomposition	+	+	+	+	+		+	+	+	+	+	+	
		Oscillation			+	+		+	+						
		Hypermetria													+
		Slowness	+	+	+	+	+	+	+	+	+	+	+	+	
	Disdiadochokinesis	Decomposition	+	+	+	+	+		+	+	+	+	+	+	
		Asynergy				+									
		Slowness				+	+	+	+				+		
	Heel-knee test	Decomposition			+	+	+			+	+				
		Oscillation			+	+					+				
		Hypermetria				+	+	+		+	+		+		
		Slowness					+								
Sensory disturbance	Pain					+	+	+	+						
	Position					+	+	+							
	Discrimnative	Graphesthesia					+	+							
		Two-point		+	+		+	+							
		Stereognostic					+	+							
Motor disturbance	Wartenberg escape sign		+				+		+		+	+			+
	Muscle hypotonus				+					+					

Discussion

Parietal ataxia is well described by Macdonald Critchley[1] as "decomposition of movement, hypo- and hypermetria, and intention tremor…swaying movements and falling away of the outstretched hand…on attempting to touch an object, the arm swayed widely back and forth… in finger to nose testing, there was marked cerebellar ataxia, with the limb rather hypotonic… rapidly alternating movements were performed badly…there was considerable ataxia on the heel-knee test"[2].

Based on the results presented herein, we suggest the following as additional clinical features of parietal ataxia : 1) ataxia is seen in the upper limbs more than the lower limbs, 2) frequent decomposition and slowness by finger-nose test, and decomposition by diadochokinesis, 3) oscillation on finger-nose test is seen more frequently with sensory ataxia than with pseudocerebellar ataxia, 4) ataxia is worse with the eyes closed in sensory ataxia than in pseudocerebellar ataxia.

The finger-nose test is decomposed into two steps, with the elbow bent first, followed by the shoulder when patients touched their own nose using the index finger. We also observed that the index finger movement was smooth, but that every movement of the index finger during

one examination in the same patient was different. Slowness in the finger-nose test was seen both at the beginning and during the movement, while in the diadochokinesis assessment, rapidly alternating movements were decomposed and the elbow swayed widely on the affected side. Yamanaka *et al*[3] believe that elbow fixation in diadochokinesis, which was shown by most of our patients, is one of the most sensitive indicators of mild ataxia. Disturbed elbow fixation results from dis-coordination of the upper arm, shoulder, and trunk, and thus parietal ataxia may indicate uncoordination of proximal muscles.

In their classical study of 1922, Foix and Thévenard[4] reported diminished muscle strength in both the upper and lower limbs on the left without full paralysis, while more recently, Attig[5] reported ataxia caused by a parietal lesion with hemiparesis. However, our study indicates that parietal lesions may cause ataxia without paralysis. Many studies also reported clumsy hand movements[4, 6–9], which along with limb-kinetic apraxia could indicate disturbance of finger dexterity. This is in contrast to parietal ataxia, which possibly indicates uncoordinated arm muscles. Parietal ataxia thus shows different symptoms from clumsy hand.

Foix *et al* (1927)[7] reported not only deep sensory disturbance, but also disturbance of two-point discrimination sense, weight sense, and material discrimination sense in a patient with parietal ataxia. Thus, position sense and other sensory disturbances should be assessed in such patients. Here, we found that two-point discrimination sense was frequent in the patients with sensory ataxia, and conclude that sensory disturbances could exacerbate ataxia.

There are some reports of lesions causing parietal ataxia. Claude & Lhermitte (1916)[10], Foix and Thévenard (1922)[4], Appenzeller and Hanson (1966)[6], and Ota and Tsuchiya (2005)[8] reported lesions in the posterior central gyrus, while Foix *et al* (1927)[7], Van Bogaert and Delbeke (1933)[11], and Ghika *et al* (1995)[9] reported lesions in the superior and inferior parietal lobules. Thus, identifying the clinical neurological features of parietal ataxia requires analysis of symptomatology with respect to MRI studies.

The current patients showed sensory ataxia mostly in the posterior central gyrus or white matter inferior to the posterior central gyrus, while pseudocerebellar ataxia was more commonly located in the white matter inferior to the posterior central gyri or the superior and inferior parietal lobules. We believe this study makes an important contribution to our understanding of sensorimotor integration in the parietal lobe by suggesting a strategic location for ataxia.

Fig. 3 proposes a mechanism of parietal ataxia, wherein the cerebellar-thalamo-cortical and cortico-pontine-cerebellar pathways are essential to movement. Brodmann 5 is the main parietal area projecting to the ventrolateral nucleus of the thalamus, in the same region that receives fibers from the cerebellum and motor cortex[12–14]. Thus, lesions in the superior and inferior parietal lobules, where Brodmann 5 is located, may cause parietal ataxia. Here, the lesions of sensory ataxia were in white or gray matter of the posterior central gyrus (Brodmann 3), and Prevosto *et al*[15] reported that Brodmann 3 projects proprioceptive information to Brodmann 5. We thus propose that disturbance of the u-fiber between Brodmann 3 and Brodmann 5 could be linked to pseudocerebellar ataxia lesions in white matter inferior to the posterior central gyri.

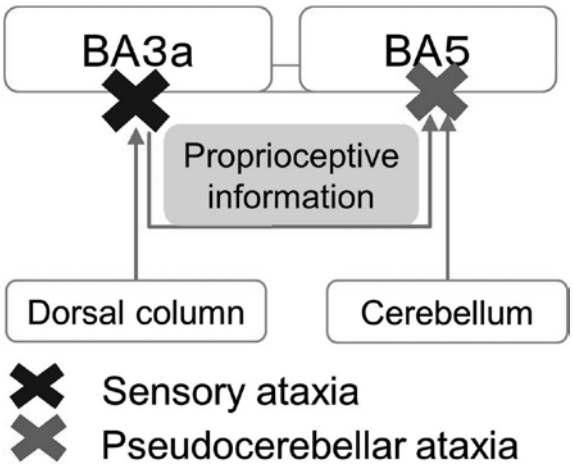

Fig. 3. Theoretical model of parietal ataxia.

Studies of parietal ataxia typically include few patients and therefore, further evidence is needed to correlate clinical symptoms with parietal lobe lesions. In this context, our report of 13 parietal ataxia cases enhances our understanding of sensory motor integration in the parietal lobe.

Acknowledgments

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