Received: 18 May 2016 | Accepted: 26 September 2016

DOI: 10.1002/jgf2.54

CASE REPORT

Journal of General and Family Medicine Open Access WILEY

Isolated astasia caused by a localized infarction in the suprathalamic white matter

Keiko Takahashi MD[1] | Akiyoshi Osaka MD[1] | Hiromasa Tsuda MD, PhD[1] | Hiroki Ogasawara MD[2]

[1]Department of Neurology, Tokyo Metropolitan Health and Medical Corporation Toshima Hospital, Itabashi-ku, Tokyo, Japan

[2]Department of Rehabilitation, Tokyo Metropolitan Health and Medical Corporation Toshima Hospital, Itabashi-ku, Tokyo, Japan

Correspondence
Hiromasa Tsuda, MD, PhD, Department of Neurology, Tokyo Metropolitan Health and Medical Corporation Toshima Hospital, Itabashi-ku, Tokyo, Japan.
Email: hiromasa_tsuda@tokyo-hmt.jp

Abstract

A 73-year-old woman with essential hypertension and diabetes mellitus abruptly developed astasia. There were no other neurological abnormalities. Cranial diffusion-weighted magnetic resonance imaging demonstrated a localized infarction in the right-sided suprathalamic white matter. Under antiplatelet agent and rehabilitation, the patient became asymptomatic within 4 days. This is a first reported case of isolated astasia due to a suprathalamic white matter infarction. We speculated that disruption of the connection from the ventrolateral nucleus of the thalamus to the posterior cingulate gyrus and/or supplementary motor area might cause astasia.

KEYWORDS

astasia, cingulate gyrus, corpus callosum, supplementary motor area, thalamus

1 | INTRODUCTION

Astasia designates motor incoordination with inability to stand, despite good strength.[1–12] Because astasia is a rare symptom, its pathogenesis remains uncertain. Regarding responsible region for astasia, the thalamus,[1–5] thalamo-mesencephalic junction,[6] mesencephalon,[7] pontomesencephalic locomotor region,[8] posterior cingulate gyrus,[9–11] and supplementary motor area[12] were reported (Table 1). We herein describe a first reported case of isolated astasia due to a suprathalamic white matter infarction.

2 | CASE REPORT

A 73-year-old Japanese woman abruptly developed inability to stand and was carried to our emergency room. Valsartan 80 mg/day and amlodipine 5 mg/day underwent for essential hypertension, and glimepiride 6 mg/day and metformin hydrochloride 500 mg/day for type 2 diabetes mellitus, respectively. There was no other contributory medical history. The patient was not smoker and had no habit of alcohol drinking. Consciousness was alert. The patient was found to have a blood pressure of 146/84 mm Hg and the heart rate of 60/minutes with sinus rhythm. General examination demonstrated no abnormalities. Cranial nerve impairment was not detected. The patient was right-handed. Hand grasping power was 22 kg in the right and 18 kg in the left. In the upper extremities, Barré sign was negative. In the lower extremities, neither Barré sign nor Mingazzini sign was positive. Manual muscle testing was all normal in the neck and extremities. Bend and extension were normal in both sides of the fingers. In the extremities, muscle tonus was all normal, and neither muscle atrophy nor fasciculation was observed. Deep tendon reflexes were all normal, and pathological reflexes were not detected in the extremities. Superficial sensation (touch sensation, pain sensation, temperature sensation, and topesthesia), deep sensation (joint sensation and vibratory sense), and combined sensation (two-point discrimination, graphaesthesia, stereognosis, and double simultaneous stimulation) were all normal. In finger-nose test, nose-finger-nose test, and arm stopping test, there were no abnormalities. Hyperpronation test, hand

TABLE 1 Previous reported cases of astasia in the English language literature

Cause	Patient's age/sex	Reference	Accompanying symptoms	Location of lesion	Period to stand without support
	65/M	1	Rt. hemi-sensory disturbance	Lt. suprathalamic white matter	1 day
	72/M	1	Rt. hemi-sensory disturbance	Lt. thalamus with spared its anteromedial part	died on 10 days after ictus
	73/F	1	not described	Rt. ventrolateral and deep parietal thalamus	6 days
	74/M	1	Lt. hemi-sensory disturbance	Rt. ventroposterior thalamus and suprathalamic white matter	4 days
	79/F	1	Lt. hemi-sensory disturbance	Rt. ventrolateral thalamus	1 day
	79/F	1	Lt. hemi-sensory disturbance	Rt. ventroposterolateral thalamus and internal capsule	6 days
	76/M	2	ataxia in the Lt. arm, Lt. cheiro-oral distribution of hypesthesia	Rt. posterolateral thalamus	7 days
Infarction	70/M	3	Lt. ptosis, Rt. facial palsy, weakness of the Lt. deltoid muscle	Lt. anterolateral thalamus	not described
	82/M	4	rightward body lateropulsion	Lt. centromedian thalamus	more than 6 weeks
	42/F	5	Rt. asterixis, Lt. ptosis, vertical one-and-a-half syndrome, Lt. internuclear ophthalmoplegia, skew deviation, convergence palsy	Lt. posterior thalamo-subthalamic paramedian thalamus	2 days
	70/F	7	Rt. asterixis, slurring speech	Lt. rostral midbrain	5 days
	67/M	9	none	Lt. posterior cingulate gyrus	7 days
	58/M	10	ataxia in the Rt. arm	Lt. posterior cingulate gyrus	3 weeks
	65/M	11	headache, visual hallucinations, leftward body lateropusion	Rt. cingulate gyrus and anterior corpus callosum	1 month
	61/M	12	leftward body lateropusion	Rt. supplementary-motor area	2 days
	73/F	present case	none	Rt. suprathalamic white matter	4 days
Hemorrhage	55/M	1	Lt. hemi-sensory disturbance	Rt. dorsal thalamus	21 days
	56/M	1	Rt. hemi-sensory disturbance	Lt. dorsoposterolateral thalamus	35 days
	63/F	1	impairment of leftward pursuit eye movement, ataxia and rigidity in the Lt. extremities	Rt. dorsal thalamus	20 days
	66/M	1	Lt. hemi-sensory disturbance	Rt. dorsal thalamus	32 days
	68/M	1	Rt. hemi-sensory disturbance	Lt. lateral thalamus	4 days
	74/M	1	Lt. hemi-sensory disturbance	Rt. dorsal thalamus and pulvinar	30 days
	76/M	1	disorientation, aphasia	Lt. anterior and dorsomedial thalamus	10 days
	58/F	6	asymmetric asterixis with Lt. dominant	Rt. thalamo-mesencephalic junction	2 months
	83/F	8	upward gaze palsy, skew deviation, convergence spasm	Rt. ponto-mesencephalic junction	more than 4 months
Tumor	57/F	1	not described	Rt. anterolateral thalamus	inability of stand was progressive
	64/M	1	Lt. hemi-sensory disturbance	Rt. ventrobasal thalamus	inability of stand was progressive

pronation supination test, finger wiggle, and foot pat were all normal. No abnormalities were detected in heel-knee test and shin-tapping test. Sit ability was almost normal. However, on attempt to stand, the patient showed postural instability due to a marked tendency to fall backward. Therefore, the patient could not stand or sit up unassisted. There were no other neurological abnormalities. As a result, the patient was diagnosed as having isolated astasia. Complete blood cell count was within normal ranges. Blood chemistry demonstrated elevated HbA1c level at 10.3%. Electrocardiogram and chest roentgenogram findings were normal. Cranial magnetic resonance imaging (MRI) demonstrated a localized infarction in the suprathalamic white matter on the right side (Figure 1). Cranial magnetic resonance

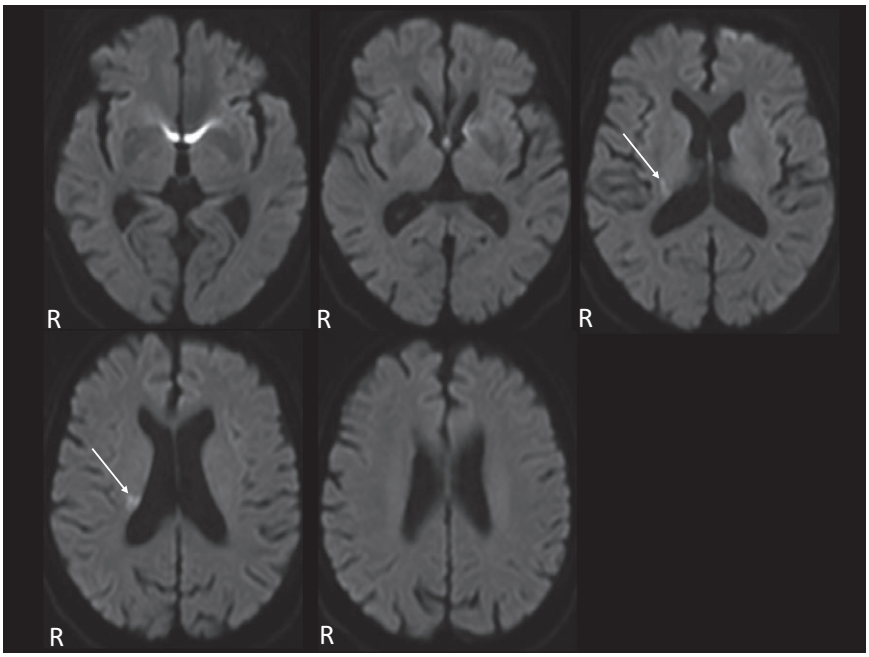

FIGURE 1 Cranial diffusion-weighted magnetic resonance imaging on axial image demonstrated a localized infarction in the suprathalamic white matter on the right side (arrow)

angiography finding was normal. The patient was admitted to our Neurologic Ward. Under antiplatelet agents and rehabilitation, the patient became asymptomatic within 4 days.

3 | DISCUSSION

Masdeu et al.[8] reported a case of astasia secondary to a localized hemorrhage in the pontomesencephalic junction. Convergence spasm, upward gaze palsy, and skew deviation were observed. The authors stated that astasia might be caused by impairment of the fastigial efferent fibers at the pedunculopontine area.[8] Song et al.[7] noted a case of astasia and unilateral asterixis secondary to midbrain infarction. de Pablo–Fernández et al.[6] reported a case of astasia with asymmetrical asterixis and pretectal syndrome due to a thalamo-mesencephalic hemorrhage.

Masdeu et al.[1] advocated that the term "thalamic astasia", based on 15 cases of astasia due to a unilateral thalamic lesion. Lee et al.[2] noted a case of astasia secondary to right-sided thalamic infarction. Ataxia in the upper extremity and cheiro-oral distribution of hyperesthesia on the left side was observed. The etiology of thalamic astasia might be a result from disruption of the fastigial fibers of the vestibulocerebellar pathway, which projected fibers from the superolateral part of the ventrolateral nucleus of the thalamus to the medial part of the primary motor area.[1,2] Kauser et al.[3] described a case of astasia with left-sided ptosis and facial weakness secondary to an infarction of the left-sided anteromedial part of the thalamus. Elwischger et al.[4] noted a case of astasia with body lateropulsion toward the right side secondary to centromedian thalamic infarction on the left side. The authors speculated that damage to the graviceptive pathway might cause astasia with body lateropulsion. Ramakrishnan et al.[5] reported a case of left-sided asterixis, astasia, and vertical one-and-a-half syndrome due to a posterior thalamo-subthalamic paramedian infarction on the left side.

Kataoka et al.[9] noted a first reported case of astasia secondary to a posterior cingulate gyrus infarction. Repetitive scrubbed movements of the fingers was observed as initial manifestation. Satow et al.[10] described a second reported case of astasia caused by a posterior cingulate gyrus infarction. In addition to astasia, dysarthria, and ataxia in the unilateral upper extremity were observed. The authors[9,10] speculated that the disruption of connection between the cingulate motor area and the vestibulocerebellar system through the thalamus might induce astasia. Zhang et al.[11] noted a case of astasia secondary to infarction of the anterior corpus callosum and cingulate gyrus. Headache and visual hallucinations were observed. The authors stated that astasia might be a result from damage to the cingulate gyrus as well as anterior corpus callosum.[11] Wada et al.[12] noted a case of astasia secondary to infarction of the supplementary motor area. The authors speculated that astasia might be caused by the disruption of the connection between the ventrolateral part of the thalamus and supplementary motor area.

In our present patient, we speculated that astasia might be elicited by the disruption of the connection between the ventrolateral nucleus of the thalamus and the posterior cingulate gyrus and/or supplementary motor area. In the English language literature, there was only one reported case of astasia due to a unilateral suprathalamic white matter infarction (Table 1).[1] Cranial MRI was not performed. Because hemisensory disturbance was observed in this case, the infarct lesion might be more extended than in our case.[1]

In cases of acute onset of astasia, cerebral infarction or hemorrhage in the vicinity of the thalamus, mesencephalon, cingulate gyrus, and supplementary motor area should be suspected. Moreover, we emphasize that isolated astasia may be caused by a suprathalamic white matter infarction.

CONFLICT OF INTEREST

The authors have stated explicitly that there are no conflicts of interest in connection with this article.

REFERENCES

1. Masdeu JC, Gorelick PB. Thalamic astasia: Inability to stand after unilateral thalamic lesions. Ann Neurol. 1988;23:596–603.
2. Lee PH, Lee JH, Joo US. Thalamic infarct presenting with thalamic astasia. Eur J Neurol. 2005;12:317–9.
3. Kauser H, Antonios N. Combined thalamic ptosis and astasia. J Clin Neurosci. 2013;20:1471–4.
4. Elwischger K, Rommer P, Prayer D, Mueller C, Auff E, Wiest G. Thalamic astasia from isolated centromedian thalamic infarction. Neurology. 2012;78:146–7.
5. Ramakrishnan S, Narayanaswamy VR. Unilateral asterixis, thalamic astasia and vertical one and a half syndrome in a unilateral posterior thalamo-subthalamic paramedian infarct: An interesting case report. J Neurosci Rural Pract. 2013;4:220–3.
6. de Pablo-Fernández E, Villarejo A, Domínguez-González C, Hidalgo FS, Callero EC. Astasia, asymmetrical asterixis and pretectal syndrome in thalamo-mesencephalic hemorrhage. Clin Neurol Neurosurg. 2009;111:766–7.
7. Song I, Kim J, An J, Kim Y, Lee K. Co-occurrence of astasia and unilateral asterixis caused by acute mesencephalic infarction. Eur Neurol. 2007;57:106–8.
8. Masdeu JC, Alampur U, Cavaliere R, Tavoulareas G. Astasia and gait failure with damage of the pontomesencephalic locomotor region. Ann Neurol. 1994;35:619–21.
9. Kataoka H, Sugie K, Kohara N, Ueno S. Novel representation of astasia associated with posterior cingulate infarction. Stroke. 2006;37:e3–5.
10. Satow T, Komuro T, Kobayashi A. Caudal cingulate infarction manifesting astasia. Case Rep Neurol. 2014;6:14–7.
11. Zhang J, Xing S, Li J, et al. Isolated astasia manifested by acute infarct of the anterior corpus callosum and cingulate gyrus. J Clin Neurosci. 2015;22:763–4.
12. Wada Y, Nishimura Y. Isolated astasia in acute infarction of the supplementary-motor area. BMJ Case Rep. doi:10.1136/bcr.01.2010.2618.

How to cite this article: Takahashi K, Osaka A, Tsuda H, Ogasawara H. Isolated astasia caused by a localized infarction in the suprathalamic white matter. *J Gen Fam Med*. 2017;18:275–278. https://doi.org/10.1002/jgf2.54