

## Thalamic infarct presenting with thalamic astasia

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Astasia, inability to stand unsupported despite good strength, resembles the marked balance impairment of patients with vestibulocerebellar disease. We describe a patient with unilateral thalamic infarct that presented with astasia. A 76-year-old hypertensive woman was admitted to our hospital because of marked unsteadiness. On neurological examination, she could not stand unsupported and the woman's body swayed back and forth markedly. The swaying was not compensated for by her taking a step forward or backward, and she frequently collapsed when support was withdrawn. Diffusion-weighted magnetic resonance image revealed a discrete infarct within the right posterolateral thalamus. Brain single photon emission computerized tomography revealed markedly decreased regional cerebral blood flow within in the right thalamus with concomitant left superior cerebellar region. We discuss the possible patho-mechanisms of thalamic astasia.

### Introduction

Astasia, inability to stand unsupported despite good strength, resembles the marked balance impairment of patients with vestibulocerebellar disease. Astasia has rarely been described in association with various thalamic lesions such as hemorrhage, infarct, and tumor, and such instances of astasia have been termed 'thalamic astasia' (Masdeu and Gorelick, 1988). Because astasia is often resolved rapidly within days and an uncommon manifestation of thalamic syndrome, thalamic astasia has not been reported in English literature since Masdeu and Gorelick (1988) first described this condition in detail in 1988. Here, we describe a patient with unilateral thalamic infarct that presented with thalamic astasia.

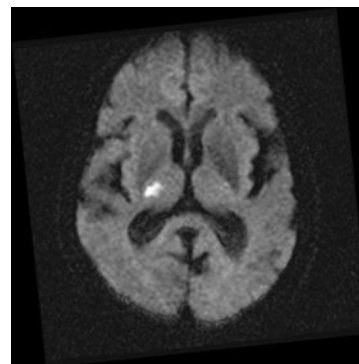
### Case report

A 76-year-old, right handed woman was admitted to our hospital because of postural instability; specifically, she swayed back and forth whilst standing. One day prior to admission, the woman felt a sudden onset of numbness in her left face and arm. Several hours later, she was no longer able to stand or sit unassisted, and was only able to move about by crawling. The woman had a 5-year-long history of poorly controlled hypertension.

On the day of admission, she could not stand unsupported, but her ability to sit was relatively preserved. On being helped to stand, the woman's body swayed back and forth markedly; the swaying was not compensated for by her taking a step forward or backward, and she

frequently collapsed when support was withdrawn. When asked to stand up from a sitting position on a chair or bed, a nearby means of support (e.g. the bedrail) with both hands to pull herself up without using her axial muscles. On neurological examination, she was alert, attentive and communicative. The cheiro-oral distribution of hypesthesia on the left side was noted, but position and vibration senses were normal. Her strength and the muscle tone of the extremities and the trunk were normal. When she was asked to hold her arms outstretched whilst keeping her eyes either open or closed, there was predominantly dystonic posture in the left hand with hyperextension of the fingers and flexion of the metacarpophalangeal joints. A finger-to-nose maneuver revealed terminal dysmetria in the left arm. Deep tendon reflexes were normoactive without pathologic reflexes.

On the after admission, brain magnetic resonance imaging (MR) and magnetic resonance angiography was undertaken. The angiogram was normal, but diffusion weighted imaging revealed a discrete infarct within the right posterolateral thalamus (Fig. 1). Three



**Figure 1** Diffusion weighted imaging demonstrates small and discrete infarct on the right posterolateral region of thalamus.

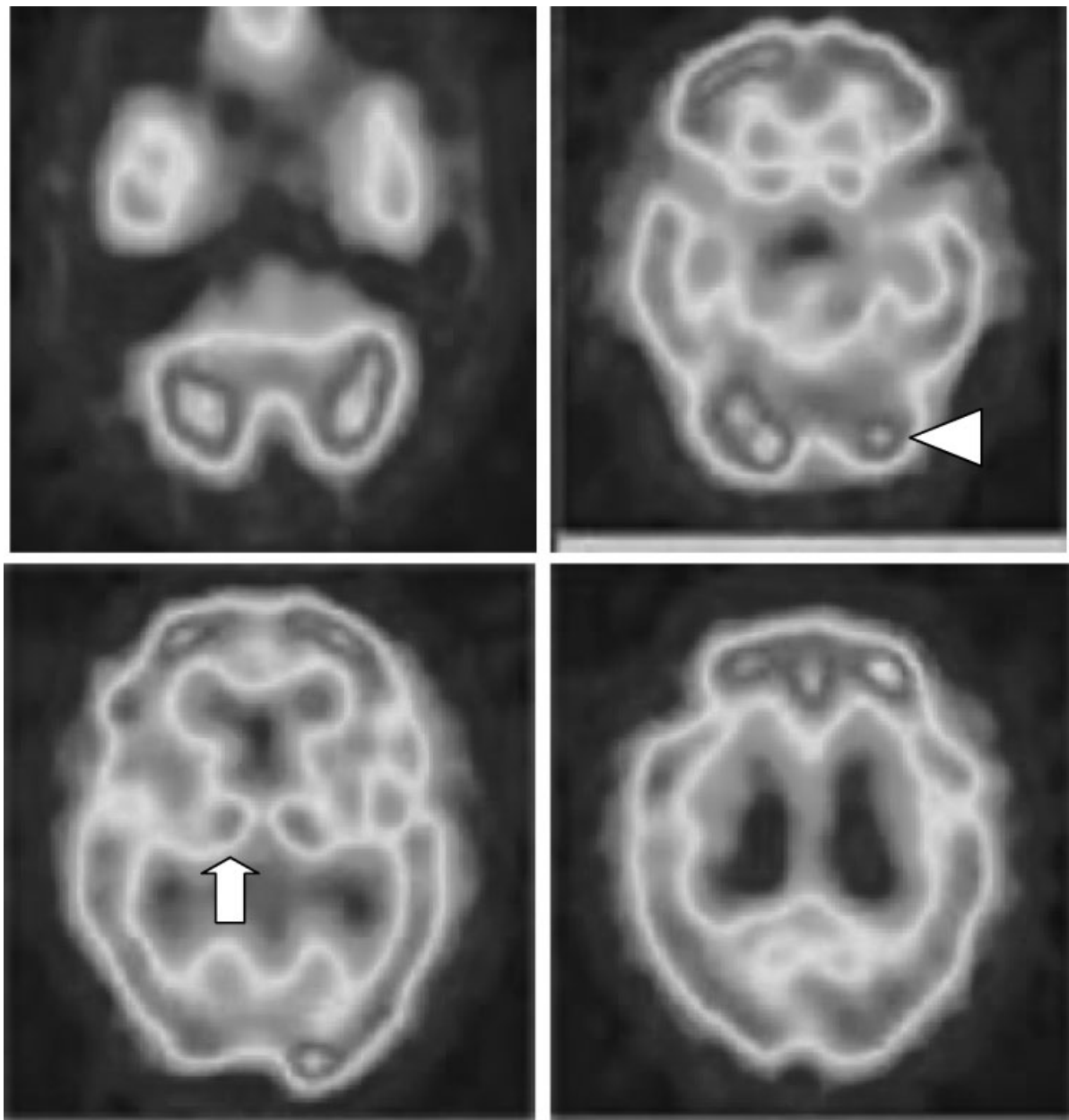
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days after admission, her inability to stand and severe postural tottering was markedly improved, and she was able to stand using axial muscles. Although she was able to take some steps in a forward direction, her gait was widened base, shortened stride and slow with slight stooped posture, which was similar to cautious gait (Nutt *et al.*, 1993), and postural instability still came out on turning. A single photon emission computerized tomography (SPECT) brain image was acquired 5 days after admission, which revealed markedly decreased

regional cerebral blood flow within the right thalamus and left superior cerebellar region (Fig. 2). At that time, she could walk far away though a support was still needed. Seven days after admission, she could walk independently.

## Discussion

Inability of stand unsupported, marked truncal instability, and the use of distal limb muscles rather than



**Figure 2** Brain SPECT undertaken on the second day of admission reveals perfusion defect in right thalamus (arrow) and superior cerebellar region (arrow head), however, cortical perfusion is relatively preserved.

axial muscles to stand up in our patients was compatible with typical clinical characteristics of thalamic astasia that was described by Masdeu and Gorelick (1988). In addition, the progression of astasia in our patient suggests that the clinical spectrum of thalamic astasia may be diverse and changeable at different time; initial inability to sit up was followed by astasia, and thereafter by abasia.

The responsible anatomical region for astasia or astasia-abasia has been known to be related with the thalamic lesion affecting mainly posterolateral thalamus. In addition, astasia associated with midbrain tegmental lesion was also described (Masdeu *et al.*, 1994). According to Masdeu and Gorelick (1988), the propensity of thalamic lesion to cause impairment of balance and gait might be because of a disconnection from vestibulocerebellar information to vestibular cortex. However, the exact anatomical origin of astasia is not known. Although it is not well understood how the brain controls the upright body posture, the posterolateral thalamus might be regarded as a structure basically involved in our control of upright body posture. Karnath *et al.* (2000a) demonstrated the second graviceptive system contributing to human's control of upright body posture might exist in human, and that this system may be different from subjective visual vertical. Additionally, analysis of MR lesion in patients with contraversive pushing indicated that posterolateral thalamus might be fundamentally involved in the graviceptive system (Karnath *et al.*, 2000b). In animal study using electrophysiological stimulation or tracing techniques (Buttner and Henn, 1976; Shiroyama *et al.*, 1999), posterolateral thalamus was known to be a relay structure of vestibular pathway on its way to the cortex.

Astasia in our patient lasted for approximately 2 days, which was in accordance with Masdeu and Gorelick's (1988) report; however, difficulty to walking had a tendency to last for about 7 days. Bilateral representation of the fastigial vestibulocerebellar projection may be one of the factors explaining the transient nature of astasia. As position and vibration senses were normal, we speculated that astasia in our patient may not be ascribed to sensory ataxia.

Brain SPECT revealed a perfusion defect in right thalamus with concomitant left superior cerebellar region, which was compatible with the crossed cerebellar diaschisis (Engelborghs *et al.*, 2000). By contrast, the cortical perfusion was well preserved. It is possible that

crossed cerebellar diaschisis in our patient may have contributed to the genesis of astasia. However, ataxia caused by cerebellar diaschisis usually presented with unilateral manifestation. Furthermore, crossed cerebellar diaschisis has been associated with dysfunctions of fine coordination rather than with severe disequilibrium, which is because of deactivation of the dentatorubrothalamic pathway (Tanaka *et al.*, 1992; Engelborghs *et al.*, 2000). In addition, normal cortical perfusion in our patient indicates that cortical dysfunction secondary to thalamic lesions may not have contributed to the genesis of thalamic astasia. Therefore, we speculate that, rather than cerebellar diaschisis or cortical dysfunction, astasia in our patient may be a direct result of the thalamic lesion that interrupted the second graviceptive pathway, although its afferents and neural network are not well-defined (Mittelstaedt, 1998).

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