

## Case report

## Isolated gait apraxia from an acute unilateral parasagittal lesion

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## 1. Introduction

Gait apraxia (GA) is defined by the inability to activate and maintain ambulation from deficits that are not attributable to motor, sensory, cerebellar, or psychiatric disease [1]. In GA, particular difficulty is seen with initiating locomotion and turning [1]. Lesional GA has mainly been reported in bilateral medial frontal lesions [1,2]. Unilateral lesions causing isolated GA, also predominantly in frontal cortex, have rarely been demonstrated in the magnetic resonance imaging (MRI) era, and usually present in a chronic and progressive fashion [1,2]. We report a patient experiencing sudden onset isolated GA, providing unique insights into its clinical and neuroanatomical correlates.

## 2. Case report

A 35-year-old right-handed pregnant woman at 37 weeks gestational age presented with sudden onset nausea and difficulty ambulating. She denied headache, limb weakness, or sensory symptoms. Her past medical history included gestational diabetes, successfully controlled by diet. Her medications only included a prenatal vitamin. She had no toxic habits or significant family history for any neurological disorder.

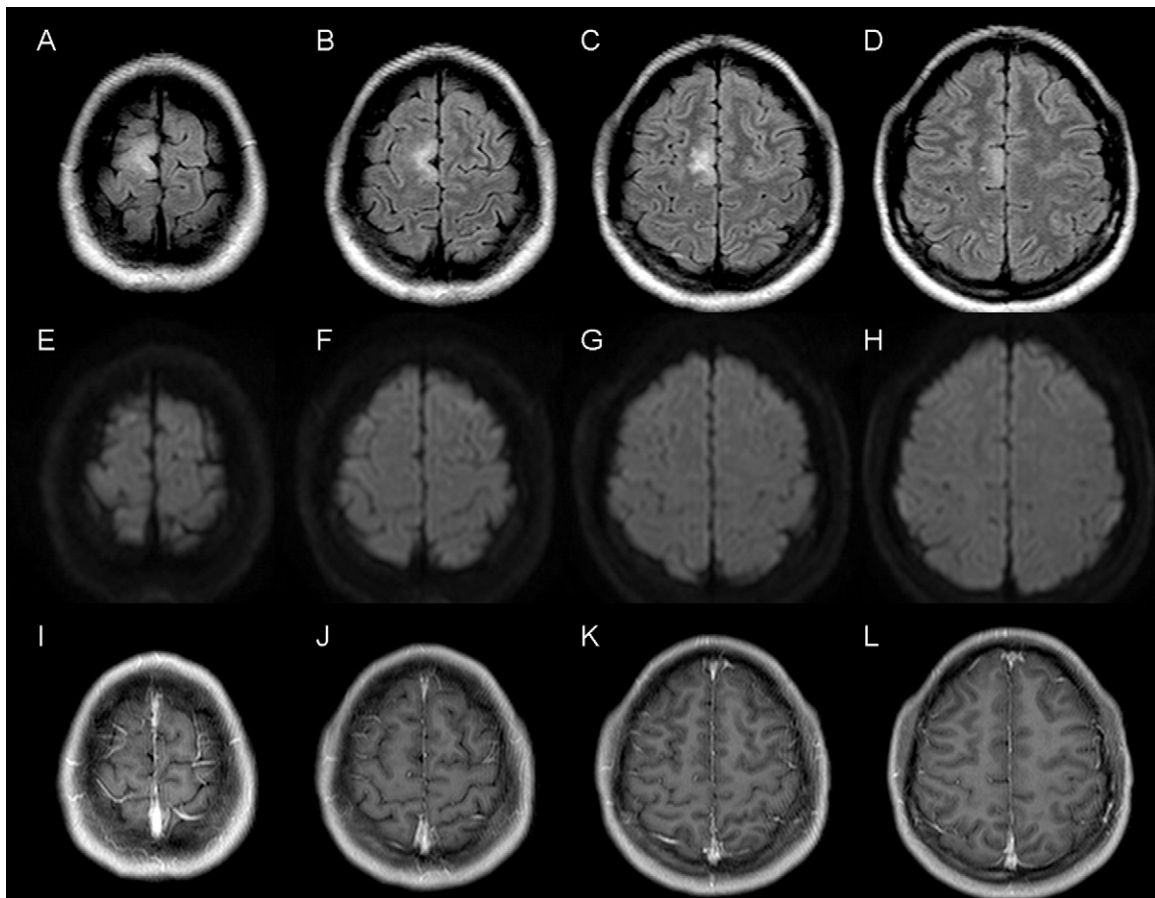
Her examination revealed a normal mental status and cranial nerves, and she lacked weakness, spasticity, rigidity, bradykine-

sia, sensory deficits, or cerebellar signs. Deep tendon reflexes were symmetrically 3+ but without Babinski signs. She could arise to a standing position but was incapable of initiating any steps to walk or turn. When a pen was placed on the floor perpendicularly in front of her and she was instructed to step over the object, with great effort she slowly took only two extremely short steps over the target. She could not simulate the leg movements of gait while lying in bed supine. She verbally reported that despite a lack of weakness, she was unable to move her legs to walk.

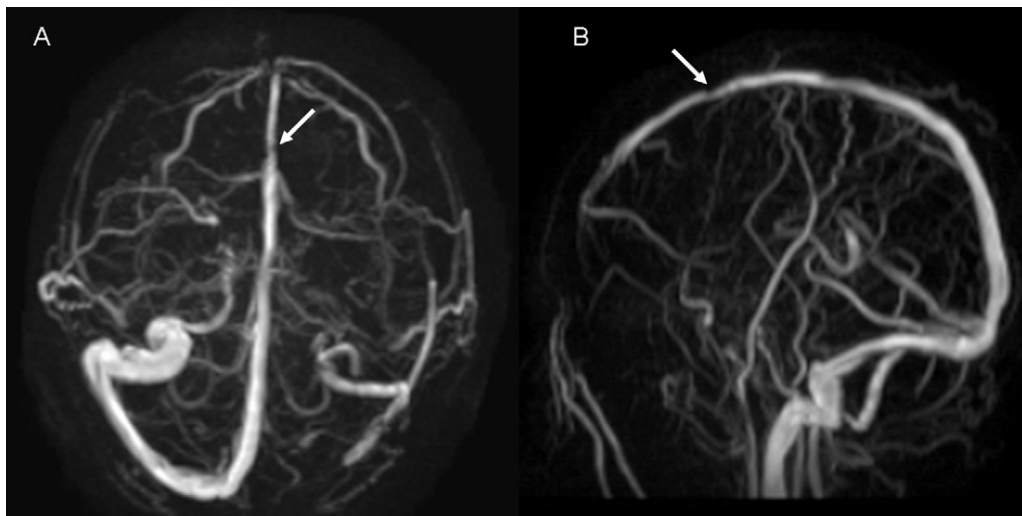
Routine laboratory testing including complete blood count, serum chemistries, and blood glucose were all within normal limits. Brain MRI without gadolinium revealed a right medial frontal lesion on fluid-attenuated inversion recovery and T2-weighted sequences without any correlate on diffusion-weighted imaging (DWI) (Fig. 1). Brain magnetic resonance venography (MRV) revealed irregularity of the superior sagittal sinus adjacent to the signal abnormality on MRI, with a dominant right transverse sinus (Fig. 2). On lumbar puncture, the opening pressure was 11 cm cerebrospinal fluid (CSF), with 0 red blood cells, 0 white blood cells, 33 mg/dL protein, and 68 mg/dL glucose. Serologies for genetic, acquired, and autoimmune causes of hypercoagulability were normal. She was diagnosed with a presumptive small cortical vein thrombosis based on the clinical presentation and imaging findings. She was anticoagulated with intravenous heparin and ultimately warfarin. Her gait normalized within 72 h, and she was able to initiate steps and turns unprompted. She delivered a healthy baby 2 weeks later. Repeat MRI and MRV with intravenous gadolinium 1 week after delivery revealed no contrast enhancement (Fig. 1) and was otherwise unchanged. After 3 months anticoagulation was discontinued without any symptom recurrence.

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**Fig. 1.** Axial magnetic resonance imaging (MRI) of the brain demonstrates a hyperintense lesion in the right parasagittal frontal lobe on fluid-attenuated inversion recovery (FLAIR) sequences (A, B, C, D). This lesion did not demonstrate a corresponding hyperintensity on diffusion-weighted imaging (E, F, G, H). Repeat MRI with intravenous contrast 3 weeks later demonstrated a lack of gadolinium enhancement (I, J, K, L).



**Fig. 2.** Magnetic resonance venography (MRV) of the brain demonstrates a slight irregularity of the superior sagittal sinus (white arrows) adjacent to the magnetic resonance imaging (MRI) abnormality in the right parasagittal frontal lobe.

### 3. Discussion

We present a patient who developed sudden onset isolated GA, with neuroimaging suggesting a causative right parasagittal frontal lesion. The MRI abnormality could have been incidental and the MRV abnormality was subtle. However, the syndrome's occurrence in the third trimester of pregnancy, where patients are

particularly hypercoagulable, and the lesion location in the high parasagittal frontal lobe, in the territory of a cortical vein draining into the superior sagittal sinus, suggest that the patient experienced a small cortical vein thrombosis. The semiology of an acute, maximal-at-onset, gradually resolving focal neurological syndrome over 72 h was also consistent with a cerebrovascular etiology. The lack of DWI positivity or contrast enhancement of the lesion

also suggested against arterial ischemia, neoplastic, and other etiologies.

This patient's focal right parasagittal frontal cortex lesion localized well to the supplementary motor area (SMA). Recently, three patients with GA having lesions including the SMA were described. One patient presented with a 10 month history of GA, and had lymphoma extending across the corpus callosum to the SMA in both hemispheres [1]. A second patient presented with an 8 month history of GA, and on MRI had focal atrophy of the parasagittal regions bilaterally, involving both the precentral gyri and the posterior portions of the superior frontal gyri [1]. In the third patient with SMA involvement, GA emerged progressively after recovery from paraplegia in simultaneous bilateral anterior cerebral artery infarctions [2]. Unlike our patient, these three cases manifested GA insidiously, and had more broad involvement of the SMA and neighboring structures bilaterally, not unilaterally.

In his report Della Salla also performed a comprehensive literature review regarding lesional GA [2]. GA manifested from a multitude of etiologies, including head trauma, intracranial tumors, cerebral abscesses, and stroke. No patient with cerebrovascular disease had an acute lesion solely in the right hemisphere, and the reported isolated left hemispheric lesions may have encompassed the SMA but had more broad involvement or were multifocal. A few cases of isolated right frontal tumors and abscesses also were reported to present with GA, but likely through direct tissue invasion or vasogenic edema developed more widespread injury rather than strictly SMA involvement.

This case lends support to the clinical observations and imaging studies that propose medial primary motor–sensory, supplementary motor, and premotor cortex participation in a higher level locomotion center controlling lower centers in the brainstem or spinal cord.[3,4] The SMA may be particularly active for the execution of gait rather than for maintaining a standing posture [5]. In

addition, the pre-SMA seems to be activated in functional imaging studies during the preparation and “ready-to-go” stages of motor tasks, whereas the more caudal supplementary motor area shows more activation in motor execution [5].

While two imaging studies in our patient did not reveal any other acute or chronic lesions to account for the GA, we cannot completely exclude the possibility of additional small lesions or involvement of other structures that lacked MRI abnormalities.

A salient feature of this case is the rarity of GA manifesting from an acute lesion. The demonstration of GA resulting from such a unilateral parasagittal frontal cortex lesion supports the presence of a cortical network subserving gait that includes the SMA. Finally, the isolated involvement of the right SMA in particular in a patient with acute GA is a unique observation that needs to be confirmed in future reports.

#### 4. Conclusion

GA may manifest from an acute right parasagittal lesion affecting the SMA. Such a lesion may disrupt a complex network of higher order cortical regulation of more caudal centers controlling locomotion.

#### References

- [1] Nadeau S. Gait apraxia: further clues to localization. *Eur Neurol* 2007;58:142–5.
- [2] Della Sala S, Francescani A, Spinnler H. Gait apraxia after bilateral supplementary motor area lesion. *J Neurol Neurosurg Psychiatry* 2002;72:77–85.
- [3] Fukuyama H, Ouchi Y, Matsuzaki S, Nagahama Y, Yamauchi H, Ogawa M, Kimura J, Shibasaki H. Brain functional activity during gait in normal subjects: a SPECT study. *Neurosci Lett* 1997;228:183–6.
- [4] Sato S, Hashimoto T, Nakamura A, Ikeda S. Stereotyped stepping associated with lesions in the bilateral medial frontoparietal cortices. *Neurology* 2001;57:711–3.
- [5] Wang C, Wai Y, Kuo B, Yeh YY, Wang J. Cortical control of gait in healthy humans: an fMRI study. *J Neural Transm* 2008;115:1149–58.