Cerebrovasc Dis 2013;36:462–463
DOI: [10.1159/000354861](https://doi.org/10.1159/000354861)**Mono-Limb Ataxia Caused by Motor Cortical Infarction: Case Report and Review of the Literature**

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Although small cortical infarctions in the primary motor cortex usually present with distal limb weakness [1], ataxia has been reported to be caused by such lesions [2–4]. However, due to the rarity of the cases, the lesion location and the presumed pathogenic mechanism have not been sufficiently discussed. We herewith describe a patient with isolated mono-limb ataxia caused by a stroke occurring at the motor cortex adjacent to the hand knob area, and review the previous literature.

Case Report

A 75-year-old hypertensive man with atrial fibrillation suddenly felt a sensation of clumsiness in the right hand while using chopsticks. His past history showed that he had developed aphasia and right limb weakness due to a left middle cerebral artery territory infarction 7 years before. After thrombolysis therapy, his neurological deficits completely resolved, and there was no functional impairment afterwards.

Neurological examination on admission showed normal articulation, muscle power, reflexes, and sensory perceptions. The finger-to-nose test showed a marked intention tremor and past-pointing in the right arm. Dysidiadochokinesia was also observed. However, no ataxia was observed in the lower extremities evaluated by the heel-to-shin test, and the tandem gait was normal. Diffusion-weighted MRI (DWI) performed 6 h after the onset showed a focal ischemic lesion located slightly medial to the precentral hand knob area (fig. 1). There was an old infarction in the left insular cortex, but no acute or chronic structural lesions were observed on the cerebellum or brainstem.

Discussion

The patient presented with mono-limb ataxia associated with cortical infarction. Although he had a previous infarction, the related neurological symptoms completely resolved afterwards, and the patient's current ataxia was caused by the new, cortical infarction. Through a literature review, we could identify 6 additional

patients who developed limb ataxia due to infarcts occurring at the motor cortex. Including ours, all patients showed a lesion located just medial to the hand knob area (fig. 1). In two patients (No. 6 and 7) who had hand clumsiness as well, the lesions were slightly extended to the hand knob area.

The cortico-ponto-cerebellar tract originates from the central regions of the cerebral cortex [5] and runs antero-medially adjacent to the corticospinal tract [6]. The characteristic lesion location, i.e. just medial to the hand knob area, in our patient and those described in the literature suggests that this tract may be selectively interrupted by the lesion, resulting in prominent ataxia. The symptom may also be explained by impaired blood flow and neuronal metabolism in the contralateral cerebellum due to supratentorial infarcts (crossed cerebellar diaschisis) [7].

Alternatively, the lesions are located in the transition zone between the primary motor and sensory cortex where short frontoparietal U shape tracts are located [8]. Disconnection of these fibers may interrupt the sensory input travelling to the motor output area, thereby producing limb incoordination. Due to the small size of the lesion, these affected fibers cannot be assessed by tractography, and the hypothesis remains to be proven. Nevertheless, our report and review of the literature provide solid evidence that mono-limb ataxia can be caused by a cortical lesion strategically located in the motor cortex.

References

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<div> <div> <div>Risk factors:</div> <div>Hypertension</div> <div>Atrial fibrillation</div> <div>Lesion location:</div> </div> <div> <div>Case 1</div> <div>75/M</div> <div>Clinical descriptions:</div> <div>Intention tremor and past-pointing on finger-to-nose test in the right arm</div> <div>Dysdiadochokinesia in the right arm</div> </div> </div> <div> </div>				
Patient No. and ref.	Age/sex	Risk factors	Clinical description	Schematic lesion location
2 [2]	69/M	Hypertension Diabetes	Oscillation of the left arm and shoulder during the finger-to-nose test Normal finger movements	<div> <div>Medial</div> <div>Rt.</div> </div>
3 [2]	67/M	Hypertension	Severe oscillation of the left arm and shoulder during the finger-to-nose test No loss of dexterity in the fingers and hand	<div> <div>Medial</div> <div>Rt.</div> </div>
4 [3]	76/M	Hypertension	Cerebellar ataxia in the left upper extremity	<div> <div>Medial</div> <div>Rt.</div> </div>
5 [3]	90/F	Hypertension	Cerebellar ataxia in the left upper extremity	<div> <div>Medial</div> <div>Rt.</div> </div>
6 [4]	43/M	Diabetes Smoking	Bilateral past pointing in the finger-to-nose test Bilateral hand dysdiadochokinesia	<div> <div>Medial</div> <div>Rt. Lt.</div> </div>
7 [4]	85/M	Hypertension Hyperlipidemia	Intention tremor on the finger-to-nose test in the right arm Right hand dysdiadochokinesia	<div> <div>Medial</div> <div>Lt.</div> </div>

Fig. 1. Demographics, risk factors, neurological deficits, and DWI findings of our patient (case No. 1) and 6 additional patients (case No. 2–7) from the literature review. The arrows in case 1 indicate the ischemic lesion adjacent to the hand knob area. In cases 2–7, the dark line is a schematic illustration representing the outline of the precentral motor cortex including the hand knob area. The black circles are the relevant infarcts. The gray circle in patient No. 4 represents an old infarct. Ages are presented in years. ref. = Reference; Rt. = right; Lt. = left.