

# Isolated Hemiataxia and Cerebellar Diaschisis after a Small Dorsolateral Medullary Infarct

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## Key Words

Hemiataxia · Cerebellar diaschisis · Inferior cerebellar peduncle · Stroke

## Abstract

Isolated hemiataxia after a medullary infarct is rare. We describe a case of isolated hemiataxia after a small infarct localized at the ipsilateral dorsolateral medulla. An 83-year-old man developed acute onset of ataxia in the left arm and in both legs. Speech and extraocular movement were normal, and he did not have any other neurological manifestations. Brain MRI showed a small infarct localized at the left dorsolateral medulla, which involved the inferior cerebellar peduncle. <sup>123</sup>ECD-SPECT showed hypoperfusion in the left cerebellar hemisphere without clear vascular territory. Neuroimaging findings for our patient suggested the involvement of the inferior cerebellar peduncle that projects to the cerebellum in our patient.

## Introduction

Ataxia is a common disabling symptom in patients with cerebellar or brainstem infarction [1]. Whereas the topography in accompanying symptoms, such as eye movement disorder, dysphagia and dissociated sensory patterns, has been studied extensively, the topodiagnostic implications of ataxia have not received as much attention [2]. Isolated hemiataxia of cerebellar type is usually attributed to lesions in the ipsilateral cerebellum [1]. Less frequently, isolated hemiataxia occurs in lesions of the contralateral pons [2], contralateral midbrain [3], and contralateral internal capsule [4], and some such lesions are regarded as an abortive form of ataxic hemiparesis. However, ipsilateral

hemiataxia and cerebellar diaschisis after a medullary infarct has not previously been known. Nevertheless, we recently had such a patient.

### Case Report

An 83-year-old, previously healthy, right-handed man suddenly noticed that when he walked straight, he unexpectedly went to the left side. Therefore, when walking, he started to support his body using his left hand against the wall. He did not notice any difficulty in using his hands, numbness in the face or other body parts, diplopia, dizziness, or nausea. Since his difficulty in walking did not change, at day 3 after the onset of disease, he visited our hospital and was admitted to the neurosurgery department. On admission, he was alert and cooperative. He had no paresis in the arms or legs. He was found to have mild limb ataxia in the left arm. However, axial, sagittal and coronal slices of brain magnetic resonance imaging (MRI) taken on day 3 and day 4 using diffusion, T1- and T2-weighted images and fluid attenuated inversion recovery (FLAIR) images showed no specific lesions, except for age-related, supratentorial ischemic changes. Therefore, he was referred to the neurology department on day 10. On referral, no apraxia, agnosia, or hemineglect was found. Cranial nerves and extraocular movements were normal, and no nystagmus was seen. He had no dysarthria or dysphagia. There was no deviation in the tongue when protruded. Tendon reflexes in his four extremities were normal. The patient had no muscle weakness in the four extremities, and there was no Babinski sign. However, he had dysmetria and decomposition in the left arm as determined by a finger to nose test. Similarly, he had dysmetria and decomposition in his legs, although there was no laterality. On standing, he was wide-based. The Romberg sign was negative when the patient was standing with his eyes closed. He could hardly walk without assistance. Body lateropulsion was positive bilaterally, but more prominent to the left side. A sensory examination showed normal pain sensation in the face as well as other parts of the body, as tested by a pin-prick (0–1 point in the affected area as compared with 10 points in the normal area). The joint position sensation of all his extremities was normal. Eye-tracking test and video-oculography showed mild hypometry to the right side. Visual suppression (a decrease indicates flocculonodular dysfunction) on caloric-induced nystagmus was evoked normally bilaterally. Somatosensory-evoked potentials induced by median nerve stimulation were normal bilaterally.

Axial slices of diffusion MRI on day 20 showed a small high-signal lesion in the left dorsolateral medulla oblongata, which spread slightly to the ventrum, involving the left inferior cerebellar peduncle ([fig. 1a, b](#)). Magnetic resonance angiography showed winding but patent basilar and cerebellar arteries, and no aneurysm or malformations were seen. Axial and coronal slices of <sup>99m</sup>Tc-labeled L,L-ethyl cysteinate dimer (ECD) -single-photon computed emission tomography (SPECT) on day 17 showed hypoperfusion in the left cerebellar hemisphere diffusely, without clear vascular territory ([fig. 2](#)). There was no other apparent diaschisis within the brain. These findings suggest that the lesion in the left dorsolateral medulla oblongata accounted for the patient's left-side dominant hemiataxia. His hemiataxia ameliorated slowly, together with mild antiaggregation therapy with 100 mg/day oral aspirin, which was started on day 3. He gradually became able to walk independently, and he was discharged on day 22.

### Discussion

Our patient was unique in that he acutely developed an isolated quasi-unilateral ataxia after a small infarct localized at the ipsilateral dorsolateral medulla oblongata. ECD-SPECT showed ipsilateral cerebellar hypoperfusion without clear vascular territory, indicating cerebellar diaschisis. To the best of our knowledge, no such case has previously been reported. Serial brain MRI of the patient on day 3 and day 4 was unable to show specific lesions, while brain MRI on day 20 clearly revealed the lesion. This time course is in accordance with the fact that diffusion imaging in acute infratentorial infarcts may have its peak at day 3 or later, which is in contrast to the earlier peak observed in acute supratentorial infarcts [5].

Ataxia is caused by lesions of the ipsilateral dorsolateral medulla oblongata, particularly when hemiataxia is accompanied by impairment of thermal and pain sensitivity in the ipsilateral face and contralateral body parts, Horner's syndrome, vertigo,

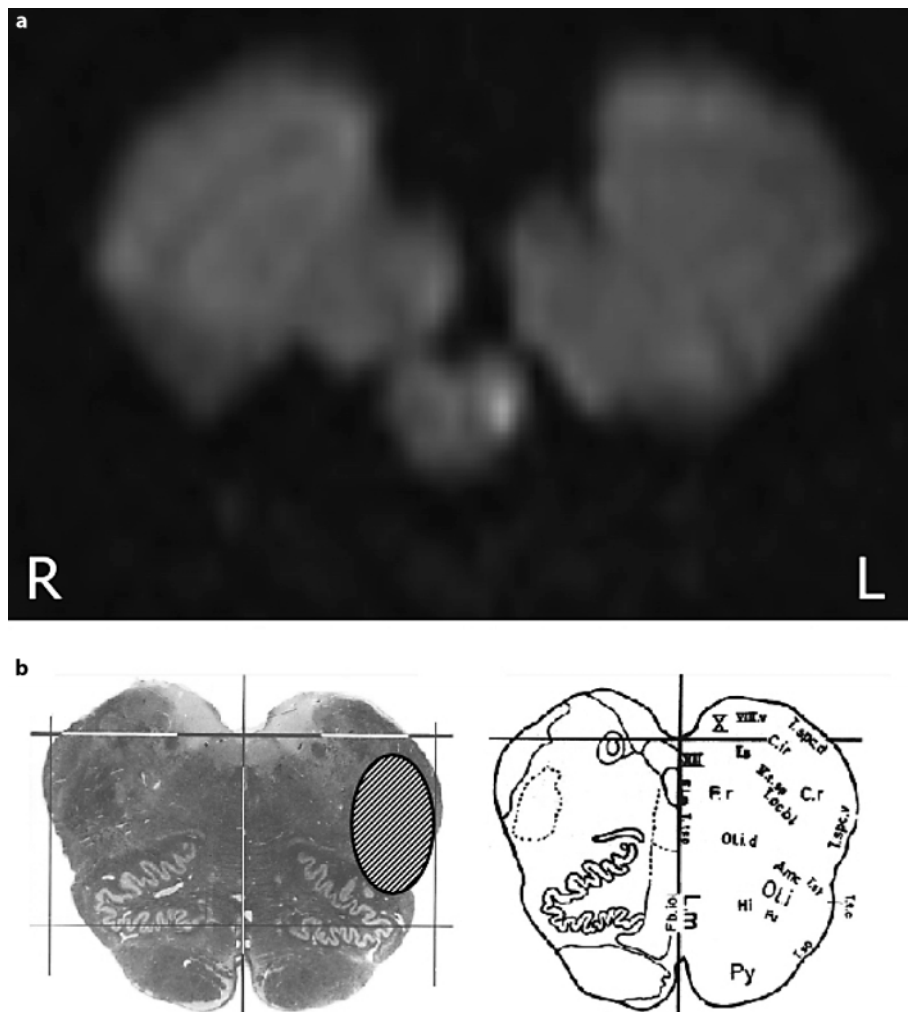
and dysphagia; the clinical signs are designated as Wallenberg's syndrome. The MRI lesion in our patient involved the inferior cerebellar peduncle, but spared the tegmental bulbar motor nuclei, the spinothalamic tract and the tractus spinalis nuclei trigemini as seen in Wallenberg's syndrome, thus leading to pure hemiataxic manifestation. This is in accordance with the results of a recent MRI mapping study showing that the inferior cerebellar peduncle is the main structure responsible for hemiataxia caused by medullary stroke [6]. Cerebellar ataxia syndrome in our patient included limb ataxia (due to the cerebellar hemisphere lesion [6, 7]) and gait ataxia (cerebellar vermis [6, 7]), but ocular ataxia was mild (upper paramedian [6, 7]); and dysarthria (upper paramedian [6, 7]), loss of visual suppression, spontaneous nystagmus or vertigo (flocculus, nodulus [6, 7]) were not observed in our patient. These clinical features in our patient possibly represent projection from the inferior cerebellar peduncle to the cerebellum in humans [2].

Whereas the MRI lesion in our patient was solely unilateral, his ataxia of the legs and gait and body lateropulsion were bilateral, although they were more prominent in the affected side. This may simply be the result of age-related, supratentorial ischemic change in the patient. However, another possibility is that the spinocerebellar tract, which lies adjacent to the spinothalamic tract within the medulla, is also involved in our case. This is because the ventral spinocerebellar tract is supposed to convey information from both sides of the body to the midline cerebellar structures in mammals [8, 9].

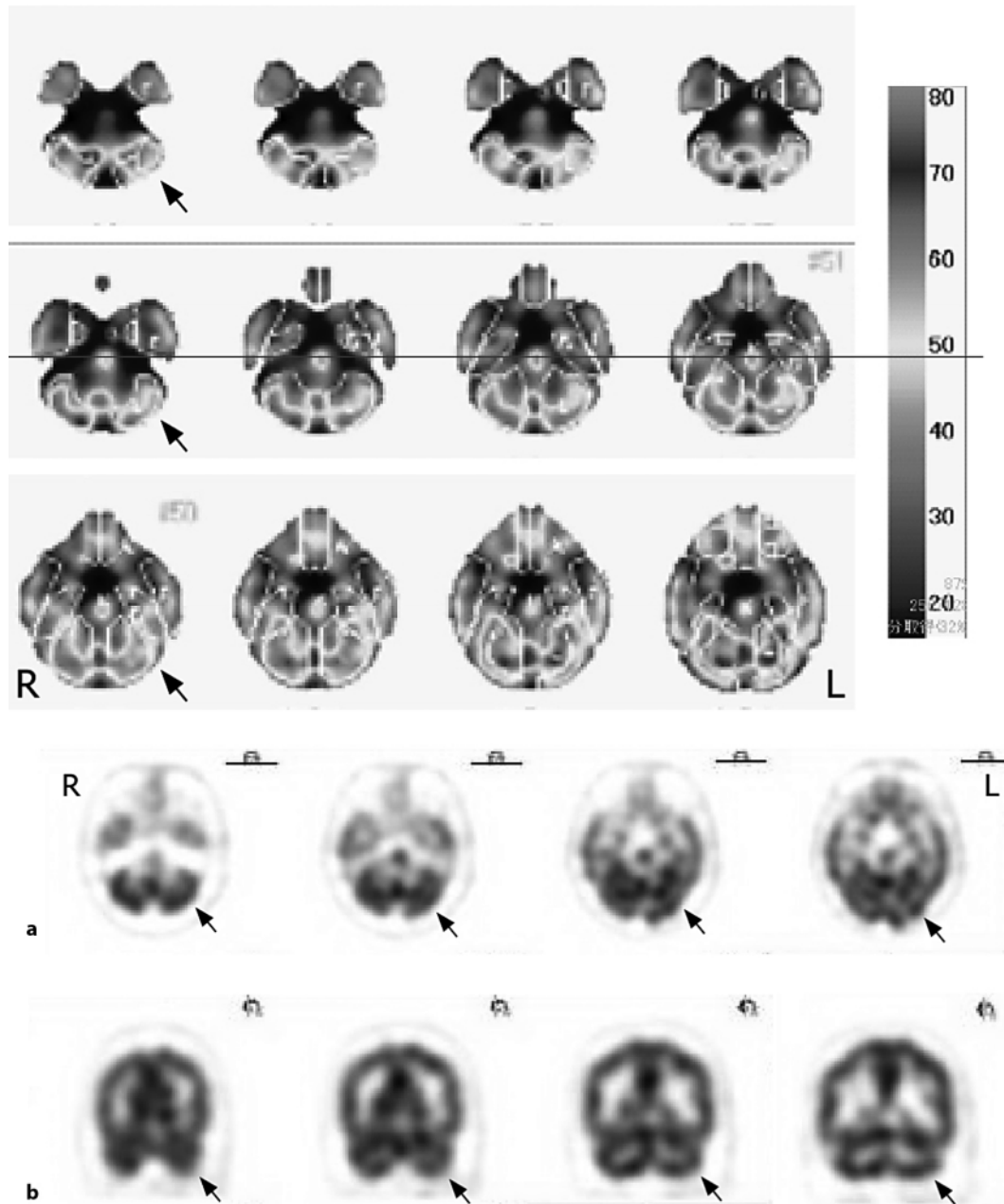
The term 'diaschisis' is used to describe a depression of regional neuronal metabolism and cerebral blood flow caused by dysfunction in an anatomically separate but functionally related neuronal region. While ataxia occurs in lesions of the pons [2], midbrain [3], and internal capsule [4], subclinical cerebellar diaschisis has been observed over a wider brain area, e.g., the frontoparietal cortex [10], thalamus [11], midbrain [12], pons [13], and medulla [14]. In our patient, ECD-SPECT could visualize hypoperfusion in the left cerebellar hemisphere diffusely. Combined with the findings by MRI, this hypoperfusion can be regarded as diaschisis, which most likely represents the anatomical connection between the inferior cerebellar peduncle and the cerebellum.

In conclusion, our patient quasi-unilaterally developed cerebellar ataxia because of a small infarct localized at the ipsilateral dorsolateral medulla. Neuroimaging findings suggested the involvement of the inferior cerebellar peduncle that projects to the cerebellum in our patient.

**Fig. 1.** MRI of the patient. **a** Axial slices of diffusion MRI on day 20 showed a small high signal lesion in the left dorsolateral medulla oblongata, which spread slightly to the ventrum, involving the left inferior cerebellar peduncle. **b** Hatched area indicates the lesion as imaged by the diffusion MRI. The anatomy of this slice was taken from Schaltenbrand and Wahren's Atlas for Stereotaxy of the Human Brain [15]. Am and Amc = Amiculum of the inferior olive; C.ir = juxtarestiform body; C.r = restiform body; Fb.IX = hypoglossal nerve fibers; Fb.iol = intraolivary fibers; Fo.c = foramen cecum; Fo.L = foramen of Luschka; F.L.m = medial longitudinal fasciculus; Fl.r.IX = rootlets of the glossopharyngeal nerve; F.r = tegmental reticular formation; Fu = fundus of the inferior olive; Gr.po = pontine grey matter; Hi = hilus of the inferior olive; L.m = medial lemniscus; Ol.I = inferior olive; Py = medullary pyramid; Ta = acoustic tuberculum; T.obc and T.ocbl = olivocerebellar tract; T.s = solitary tract; T.so = spinoolivary tract; T.spc.d = dorsal spinocerebellar tract; T.spc.v = ventral spinocerebellar tract; T.st = spinothalamic tract; T.t.c = central tegmental tract; T.tsp = tectospinal tract; V.t.sp = spinal trigeminal nucleus; VIII.c.v = ventral cochlear nucleus; VIII.v = vestibular nucleus; IX = glossopharyngeal nucleus; X = nuclei of vagus; and XII = hypoglossal nucleus.



**Fig. 2.** SPECT of the patient. Axial (**a**) and coronal (**b**) slices of  $^{99m}\text{Tc}$ -labeled ECD-SPECT on day 17 showed hypoperfusion (arrows) in the left cerebellar hemisphere diffusely, without clear vascular territory.



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