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Isolated Hemiataxia of the Cerebellar Type after a Small Internal Capsular Infarct

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Hemiataxia is classically ascribed to lesions involving the cerebellar system or interrupting the sensory pathways that control movement. Hemiataxia after supratentorial brain infarction is not uncommon. It, however, rarely occurs in isolation and most

Table 1. Case reports of isolated hemiataxia after a capsular infarct

Authors	Lesion	Cases
Luijckx et al. [1], 1993	haemorrhage in the posterior limb of the internal capsule	1 case
Luijcky et al. [2], 1994	infarct in the posterior limb of the internal capsule	2 cases

of the time is accompanied by either pyramidal or sensory defects, or a combination of both. Isolated hemiataxia after supratentorial brain lesion is infrequent. To our knowledge, only 3 cases of internal capsular lesions, of which 2 were ischaemic and 1 haemorrhagic in nature, have been described to date [1, 2] (table 1). The case reported here therefore provides further illustration of hemiataxia of the pure cerebellar type resulting from an ischaemic lesion restricted to the posterior limb of the internal capsule.

Case Report

A 65-year-old man with a history of hypercholesterolemia, hypertension and smoking suddenly developed clumsiness of the left arm and unsteadiness of gait, without weakness. Neurological examination performed on admission 24 h after onset of symptoms showed left hemiataxia consisting of left-sided dysmetria, dysdiadochokinesia and Holmes rebound phenomenon, hypermetria and intention tremor on finger-to-nose, finger-to-finger and heel-to-shin tests. This incoordination remained unchanged with the eyes open or closed. Ataxia of the arm was severer than that of the leg. Left-sided hypotony and decomposition of movement at tendon reflexes of the left leg were present. When standing, the patient showed a wide-based unsteady gait and a slight tendency to fall to the left. There was no weakness or pyramidal signs, and a normal plantar response. Position and vibration sense as well as touch, temperature and pain sensation were preserved. The patient had no signs of brainstem involvement, no pathological nystagmus or saccadic pursuit. There was no cognitive impairment. Computed tomography of the brain performed at admission disclosed a small hypodense lesion restricted to the posterior limb of the right internal capsule, compatible with a small deep infarct as well as an older lesion in the right centrum semiovale. MRI on day 3 confirmed these lesions with the internal capsular one appearing as new on diffusion-weighted MRI sequences and thus responsible for symptoms (fig. 1a, b).

There was a moderate improvement of the ataxia within the 3 days following the attack.

Discussion

Hemiataxia after supratentorial brain infarction is not uncommon and has been reported by several authors [3–7]. The latter, mainly related to a thalamic or capsular lesion, rarely occurs in isolation, being currently accompanied by either motor or sensory signs, or both [7–10]. Among hemiataxia secondary to supratentorial brain lesion, both ataxic hemiparesis and thalamic atax-

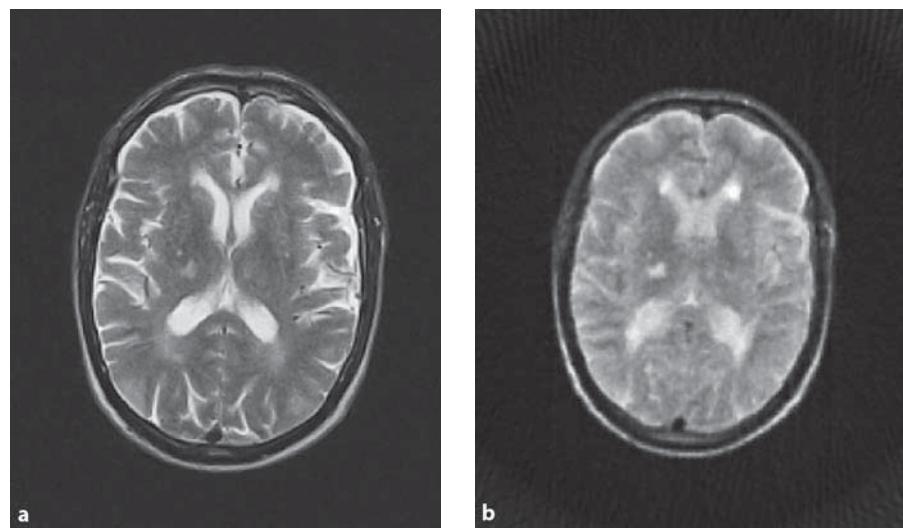


Fig. 1. **a** Transverse T₂-weighted MRI showing a small lesion of high signal intensity in the posterior limb of the right internal capsule. **b** Transverse diffusion-weighted MRI showing a small recent lesion in the posterior limb of the right internal capsule.

ia have been well described. Ataxic hemiparesis was coined by Fisher [3] and Fisher and Cole [11] and attributed to the infarct affecting either the internal capsule, the upper basis of the pons or the corona radiata. Ataxic hemiparesis has also been reported after a midbrain lesion [12].

While the associated weakness is due to interruption of the corticospinal fibres, the ataxia has been attributed to damage to the corticopontine fibres descending into the posterior limb of the internal capsule as well as to the interruption of the thalamocortical connections which run medially in the posterior limb of the internal capsule [4, 6].

Thalamic ataxia has been explained by lesions involving the ventral lateral nucleus of the thalamus, i.e. the thalamogeniculate territory. Interruption of the dentatorubrothalamicocortical fibres appears therefore responsible for the hemiataxia [7, 13]. Thalamic ataxia presents the main clinical characteristics of a cerebellar type of ataxia. It has been reported in association with either hemisensory loss (hemiataxia, hypoesthesia), weakness (ataxic hemiparesis) or both (hypoesthetic ataxic hemiparesis). The sensory loss is caused by damage to the ventral posterolateral and ventral posteromedial nucleus by interrupting the ascending spinothalamic and medial lemniscus fibres, respectively. When present, the weakness is usually mild and only transient. The potentially severe weakness observed in ataxic hemiparesis and hypoesthetic ataxic hemiparesis after thalamic stroke appears to result from larger lesions not only restricted to the thalamus, but also extending to the internal capsule, usually in the setting of a haemorrhage [13].

Crossed cerebellar diaschisis (CCD, i.e. decreased blood flow and cerebral metabolism in the cerebellar hemisphere contralateral to the side of a supratentorial lesion) [14] is frequently reported after supratentorial infarction in studies with positron emission tomography (PET) and/or single-photon emission computed tomography (SPECT).

Lesions in the internal capsule and thalamus have been described as a cause of CCD.

In both ataxic hemiparesis and thalamic ataxia, the CCD observed by either PET [15–17] or SPECT [18] strongly suggested that hemiataxia resulted from damage to both cerebellar pathways, not only the corticopontocerebellar as firstly described, but also the dentatorubrothalamicocortical connections.

CCD has also been described in correlation with transient ischaemic attack in a study where SPECT showed a focal reduction of perfusion during the presence of the clinical symptoms whereas the control after full clinical recovery was normal. This study shows that a temporary suppression of regional function is capable of producing secondary remote effects such as CCD [18]. In our case, the possibility of a concomitant transient ischaemic attack in the cerebellar hemisphere is highly unlikely since the ataxia persisted 3 days after the attack.

The patient described in our study is a case of CCD but with a particular representation.

By contrast, isolated hemiataxia of the cerebellar type after supratentorial brain lesion appears to be infrequent and has been only rarely reported [1, 2]. To our knowledge, only 3 cases of isolated hemiataxia after internal capsular lesion have been reported to date, and they were all by the same group [1, 2]. Two of them were due to a lacunar infarct restricted to the posterior limb of the internal capsule on MRI sequences [2] while one was secondary to a haemorrhage [1].

Classen et al. [19] described a case of visuomotor apraxia after a lesion in the right dorsal thalamus, the geniculate body and the adjacent retrothalamic and medial temporal white matter. The most posterior fibres of the posterior limb of the internal capsule were interrupted. Glickstein [20] described the participation of the descending corticopontocerebellar and ascending cerebello-thalamocortical projections in the visual guidance of movement.

Hemiataxia in our patient was clinically of the cerebellar type and cannot be attributed to disturbed proprioception or underlying weakness. There was no deep sensory loss on neurological testing, eye closure did not worsen the ataxia, optokinetic following was intact and we noted neither weakness nor pyramidal signs

suggestive of ataxic hemiparesis. In addition, MRI sequences clearly showed that the small deep lesion was restricted to the posterior limb of the internal capsule without involvement of the thalamus. Based on previous observations regarding ataxic hemiparesis and thalamic ataxia together with the 2 previously reported cases of isolated hemiataxia after capsular lacunar infarct, we strongly believe that in our patient, hemiataxia was more likely due to a lesion of the cerebellar pathways, either the ascending dentatorubrothalamicocortical tract or the descending corticopontocerebellar pathway at the level of the posterior limb of the internal capsule. Functional MRI, PET or SPECT would have been of great interest in this patient to demonstrate CCD and thus assess our hypothesis. Such investigations were, unfortunately, not performed.

To conclude, as previously suggested by Luijckx et al. [1, 2], as well as discussed in anatomical studies [21–23], the case reported here provides further clinical evidence of the anatomically segregated passage of the cerebellar fibres through the posterior part of the posterior limb of the internal capsule. Interruption of the cerebellar connections in the internal capsule may therefore cause isolated hemiataxia of the cerebellar type as observed in our patient. The determination of whether the descending or ascending pathway is severed and responsible for the cerebellar symptoms appears difficult. However MR diffusion tensor imaging may provide the answer to this question by studying the development of a descending or ascending wallerian degeneration in a given patient with an infarction in the posterior limb of the internal capsule.

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Floating Basilar Artery: An Interesting Clinical Dilemma

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Case Report

Our patient was a 79-year-old right-handed female. She presented with an event of speech and language disturbance, with right-sided weakness and numbness. Computed Tomography (CT) was performed in preparation for possible tissue plasminogen activator (tPA) administration but the event resolved completely within an hour. While being interviewed after admission, the patient had a second spell. She abruptly developed dysarthria, which progressed with right hemiplegia. At 10 min, her right-side function began to return and the event had resolved in 30 min. Magnetic resonant angiography (MRA) revealed minimal backfilling into the basilar artery apex (fig. 1B). Digital subtraction angiography (DSA) did not visualize the basilar artery (fig. 1A).