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## Gait and bilateral limb ataxia as isolated feature of a lower midbrain tegmental infarction

### A clinical-MRI study

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Sirs: The clinical picture of midbrain infarction is dominated by third-nerve palsy, conjugate or disconjugate gaze impairment, contralateral motor and sensory deficit and homolateral or contralateral limb ataxia [1–3]. Among various mesencephalic syndromes of vascular origin, those related to the lower midbrain lesion are rare [2, 3].

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We describe a patient with an isolated lower tegmental midbrain infarction, with gait and bilateral limb ataxia as isolated features.

A 71-year-old man complained of dizziness, imbalance, slight hypersomnolence, and bilateral clumsiness of arms and legs. There was a worsening of symptoms and 12 hours later at admission neurological examination revealed slight hypersomnolence, dysarthric speech and pronounced gait and bilateral limb ataxia. The NIHSS score was four. The patient walked with a wide-based, staggering gait without tendency to deviate to one side. The stance was on a broad base with impossibility standing with their feet together or in tandem position. Romberg sign was absent.

At the finger-to-nose and at the heel-to-shine tests important dysmetria was present. The movement path of the limbs was erratic with relevant hypermetria. Rapid alternating movements were irregular bilaterally. The rest of the neurological examination was normal.

One year before he suffered from an anterior circulation infarction involving the frontal branches of the right middle cerebral artery, without any residual deficit. Regarding conventional vascular risk factors, the patient suffered from hypertension and non-valvular atrial fibrillation. He was taking oral anticoagulants, with INR value ranging between 2 and 3.

Non contrast cranial CT showed only the previous right frontal infarction. Five days later brain MRI revealed a small ischemic lesion in the middle tegmental portion of the lower midbrain (Fig 1 A–C); multiple silent small lacunar infarctions were present in the white matter bilaterally. Extracranial duplex ultrasonography and MR-angiography of car-

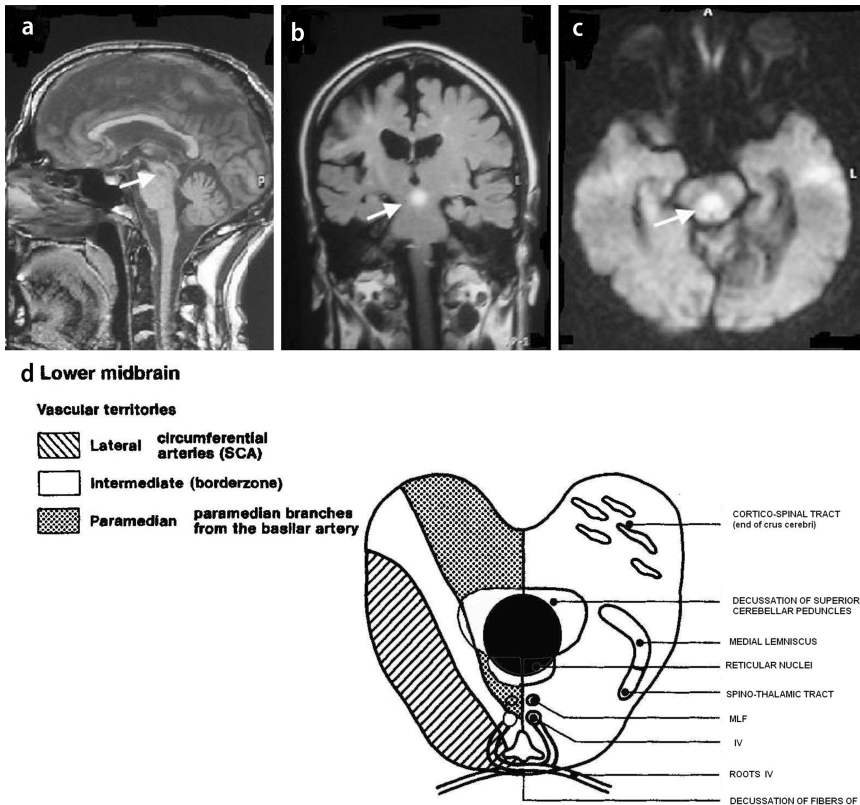
otid, vertebral and basilar arteries were normal.

Transthoracic and transoesophageal echocardiography failed in revealing intra-cardiac thrombi. Eight-months later gait and bilateral limb ataxia and dysarthria were the isolated and persistent neurological deficits. Palatal myoclonus was absent and the modified Rankin Scale was 3.

Our patient presented an infarction in the middle portion of the midbrain tegmentum, with gait and bilateral cerebellar limb ataxia as dominant and persistent symptoms. The vascularization of the midbrain regions is complex, and the region close to the midline is supplied by the paramedian branches from the basilar artery (Fig 1D). Generally, in midbrain infarction the cerebellar ataxia is overshadowed by upper-midbrain and thalamic-subthalamic signs [1–2].

A tetra-ataxia has been reported in patients with midbrain infarction [1–6] but in none of them it was the isolated feature. In these cases the bilateral ataxia was associated to other neurological deficits, such as hemiparesis, dysphagia, dysphonia, eye-movement disorders or palatal myoclonus.

Moreover, a bilateral ataxia was the sole manifestation of a rostral pontine tegmental infarction [7] and in patients with heat stroke [8]. In our patient the clinical picture may be considered a partial form of the Wernekink commissure syndrome where bilateral ataxia is associated to ocular abnormalities and palatal myoclonus. Indeed oculomotor deficit and delayed palatal myoclonus were absent and cerebellar signs were isolated except for the transient hypersomnolence related to the involvement of the reticular formation located dorsally to the superior cerebellar peduncles



**Fig. 1** Brain MRI. T1 excitation, sagittal scan (A); FLAIR axial scan (B); DWI axial scan (C). The ischemic lesion (arrow) involves the central portion of the lower midbrain (B–C), sparing the most dorsal portion of the tegmentum (A). The infarction is located in the central portion of the midbrain, at the anterior aspect of the aqueduct, corresponding to the superior cerebellar decussation (Wernekink commissure). (D) Axial anatomic drawing corresponding to the lower midbrain. The ischemic lesion (dark area) involves the decussation of the superior cerebellar peduncles and the reticular nuclei. The lesion spares the pyramidal tracts (ventral midbrain) and the medial lemniscus (lateral midbrain) as well as the oculomotor nuclei and the medial longitudinal fasciculus (dorsal midbrain portion close to the aqueduct)

decussation. The absence of sensory and pyramidal deficits, as well as of oculomotor disturbances, is explained by the strategic location of the infarct (Fig 1A–C). Indeed the lesion spares the mesencephalon, where sensory and pyramidal tracts run as well as the most dorsal portion where oculomotor nuclei and medial longitudinal fasciculus are located.

As clearly shown in Fig. 1, the infarction involves the middle portion of the lower midbrain sparing the most dorsal aspect close to the aqueduct as well as the ventral and lateral portion. This region corresponds to the Wernekink decussation where the cerebello-thalamic fibers, running in the superior cerebellar peduncles, cross the midline (Fig 1D) before reaching the thalamus. Notwith-

standing the small amount of the infarction, such strategic location may have damaged both the right and the left cerebellar-thalamic fibers, thus causing the gait and bilateral limb ataxia. Differently from the cases previously reported [1–6], in our patient the infarct was not lateralized and was located in the central portion of the midbrain, explaining the severity of the cerebellar manifestation.

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