Limb ataxia and proximal intracranial territory brain infarcts: clinical and topographical correlations

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Background: Limb ataxia is classically attributed to cerebellar hemispheric lesions, although isolated lesions of the inferior cerebellar peduncle (ICP) in the medulla may also cause this sign. It is still unclear why only some patients with acute cerebellar infarcts in the posterior inferior cerebellar artery (PICA) territory present with limb ataxia. The proximal intracranial posterior circulation (P-PC) territory includes structures fed by the intracranial vertebral arteries (ICVAs): the medulla, supplied by small ICVAs branches, and posterior inferior portion of the cerebellum, fed by PICA. ICP and PICA territory cerebellar infarcts most often occur independently but occasionally occur together.

Objective: To identify structures responsible for limb ataxia in acute P-PC brain infarcts, correlating clinical and topographical findings.

Methods: Sixteen patients (8 women) were included, aged 30–82 years (mean 62 years), with isolated acute strokes in the P-PC territory.

Results: The cases reported here indicate that limb ataxia in acute P-PC territory infarcts may be associated with damage to the ICP in the dorsolateral medulla, regardless of a hemispheric cerebellar lesion. In fact, among the nine patients with PICA stroke, limb ataxia was observed only in the two patients who also presented damage to the dorsolateral medulla involving the ICP. Of the seven patients with isolated dorsolateral medullary infarct, only five patients with ICP damage had limb ataxia.

Conclusions: When correlating limb ataxia and acute P-PC infarcts, it is important to take into account the entire ICVA territory.

Limb ataxia is characterised by dysmetria, dyssynergia and intention tremor, and is clinically assessed by the finger-to-nose and heel-to-knee tests.¹ It is classically associated with hemispheric cerebellar lesions² and rarely with brainstem lesions.³ Previous reports on cerebellar strokes focused on the frequency of neurological signs in patients with different cerebellar arterial territory infarcts.^{4–5} Limb ataxia was reported in only approximately 50% of cases of posterior inferior cerebellar artery (PICA) infarcts but it was not related to the histological or neuroradiological findings.^{4–5} A very recent clinical and MRI study⁶ reported limb ataxia in only 4 of 13 patients with acute PICA stroke. The authors correlated this finding with impairment of the intermediate and lateral cerebellar cortex or of the cerebellar nuclei, which are involved in the control of limb coordination. However, it is well known that lesions of the inferior cerebellar peduncle (ICP) without involvement of the cerebellar hemisphere may also cause limb ataxia.⁷

The proximal intracranial posterior circulation (P-PC) territory includes structures fed by the intracranial vertebral arteries (ICVAs), the medulla and the posterior inferior portion of the cerebellum. The blood supply of the posterior inferior cerebellum and the ICP derive from different branches of the ICVAs. The lateral medulla is supplied by small branches that originate from the ICVA and course through the lateral medullary fossa to supply the ICP and the dorsolateral medulla. The ICVA also gives rise to the PICA. The medial branch of the PICA supplies a small portion of the dorsal medulla but not the ICP. ICP and PICA territory cerebellar infarcts most often occur independently but occasionally occur together. Only 1 in 5 proximal territory infarcts include both the lateral medulla and the PICA territory cerebellum.⁸ When both occur together it is usually caused by a long occlusion of the ICVA which blocks flow in both the PICA and lateral medullary penetrators.

Here we describe 16 patients with acute infarcts in the ICVA territory in order to understand the role of the cerebellum and of the ICP in the occurrence of limb ataxia.

RESULTS

Clinical features

Based on clinical (presence or absence of limb ataxia) and MRI findings, we identified four patterns (table 1). In fig 1, an axial T2 MRI—at the level of the inferior olivary nucleus in the medulla—of the four patients, each one representative of the four patterns, is illustrated.

Pattern 1: posterior–inferior cerebellar infarct (ICVA territory: PICA)

Seven patients (patient Nos 1–7) were categorised into this group.

MRI showed an infarct in the cerebellar hemisphere in the PICA territory (in 2 of 7 patients the infarct involved both the cerebellar hemispheres in the PICA territory), in the absence of brainstem lesions.

All the patients presented with vomiting and vertigo. On examination, there was gait ataxia associated or not with nystagmus. Limb ataxia was absent.

Pattern 2: posterior–inferior cerebellar (ICVA territory: PICA) and dorsolateral medullary infarct involving the ICP (ICVA territory: small branches)

Two patients (patient Nos 8 and 9) were included in this group.

Abbreviations: ICP, inferior cerebellar peduncle; ICVA, intracranial vertebral artery; mRS, Modified Rankin Scale; PICA, posterior inferior cerebellar artery; P-PC, proximal intracranial posterior circulation; SCA, superior cerebellar artery

Table 1 Clinical–MRI correlations in acute proximal intracranial territory brain infarcts

	Lesion topography	Limb ataxia
Pattern 1 (Patients 1–7)	Posterior–inferior cerebellar infarct (ICVA territory: PICA)	Absent
Pattern 2 (Patients 8, 9)	Posterior–inferior cerebellar infarct (ICVA territory: PICA)	Present
Pattern 3 (Patients 10–14)	Dorsolateral medullary infarct involving the ICP (ICVA territory: small branches)	Present
Pattern 4 (Patients 15, 16)	Dorsolateral medullary infarct sparing the ICP (ICVA territory: small branches)	Absent

ICP, inferior cerebellar peduncle; ICVA, intracranial vertebral artery; PICA, posterior inferior cerebellar artery.

MRI showed an infarct in the cerebellar hemisphere in the PICA territory and in the ipsilateral dorsolateral medulla involving the ICP.

The patients presented with vertigo, vomiting and some symptoms of Wallenberg's syndrome (dysphagia, dysphonia, dysarthria). On examination, there was limb ataxia on the ipsilateral side of the bulbar infarct, gait ataxia and signs of involvement of the nucleus ambiguus (dysphagia, dysphonia, dysarthria).

Pattern 3: dorsolateral medullary infarct involving the ICP (ICVA territory: small branches)

Five patients (patient Nos 10–14) were categorised into this group.

MRI showed an infarct in the dorsolateral medulla involving the ICP.

Patients presented with vertigo and vomiting. On examination, there was limb ataxia on the ipsilateral side of the bulbar infarct and gait ataxia, associated or not with nystagmus. There were also signs of Wallenberg's syndrome (dysphagia, dysphonia, dysarthria, Horner's syndrome, crossed sensory loss including the face on one side and the limb on the contralateral side).

Pattern 4: dorsolateral medullary infarct sparing the ICP (ICVA territory: small branches)

Two patients (patient Nos 15 and 16) were included in this group.

MRI showed an infarct in the dorsolateral medulla sparing the ICP.

Both patients presented with dysphagia. On examination, there were signs of Wallenberg's syndrome (dysphagia, dysphonia, dysarthria, Horner's syndrome, crossed sensory loss including the face on one side and the limbs on the contralateral side). There was no limb ataxia.

Additional characteristics of the patients

Demographic data

Among the 16 patients there were 8 women and 8 men with a mean age of 62 (15) years (range 30–82).

Aetiology

The presumed cause of infarct was: cardioembolism in three patients; large artery (ICVA) occlusive disease—haemodynamic mechanism—in four patients; intra-arterial embolism in four patients; and unknown in five patients.

Risk factors

The most common risk factors were hypertension (10 patients), current smoking (3 patients), diabetes mellitus (3 patients), hypercholesterolaemia (4 patients) and previous myocardial infarction (1 patient).

Prognostic factors and outcome

We obtained follow-up information in 13 surviving patients after a mean time of 32 (SD 26) months by telephone interviews. To describe the outcome, we used the Modified Rankin Scale (mRS). One patient (No 15) was lost to follow-up. One patient (No 8) died during the acute period of gastrointestinal bleeding and one patient (No 13) died during follow-up of lung carcinoma. At follow-up, most of the remaining patients (13 patients) had no symptoms or only slight symptoms that caused no or slight disability: four patients (Nos 2, 3, 7, 10) had a mRS score of 0; five (Nos 4, 5, 12, 14, 16) had a mRS score of 1; three (No 6, 9, 11) had a mRS score of 2 and one (No 1) had a mRS score of 3.

DISCUSSION

The results of the present study indicate that limb ataxia in acute P-PC territory infarcts may be associated with damage to the ICP in the medulla, regardless of a hemispheric cerebellar lesion. In fact, among our patients with acute PICA stroke, limb ataxia was not observed in patients with an isolated hemispheric cerebellar lesion (patient Nos 1–7), whereas it occurred only in patients who also presented damage to the dorsolateral medulla involving the ICP (patient Nos 8 and 9). This suggests that the ICP, which contains the spinocerebellar, vestibulocerebellar and olivocerebellar afferent pathways, appears to be crucial for the occurrence of limb ataxia. It is noteworthy that of the seven patients with dorsolateral medullary infarct, only patients with ICP damage (patient Nos 10–14) had limb ataxia while those without ICP damage (patient Nos 15 and 16) had not.

Most studies in PICA stroke have dealt with the frequency of limb ataxia, reporting this sign only in about 50% of patients.^{4,5} However, the relationship between this sign and the topographical findings has not been systematically assessed. Therefore, it is still unclear why patients with an acute PICA stroke may or may not present with limb ataxia. Very recently, clinical findings and three dimensional MRI based cerebellar lesion sites were compared in a large sample of patients with cerebellar lesions.⁶ Acute PICA stroke was reported in 13 patients, but only four had limb ataxia. The authors correlated the presence of limb ataxia with impairment of the intermediate and lateral cerebellar cortex or of the cerebellar nuclei (dentate and interposed), which are involved in the control of limb coordination. This may be consistent with the observation that infarcts in the superior cerebellar artery (SCA) territory are usually associated with limb ataxia, because the SCA almost always supplies the dentate and interposed nuclei and the superior cerebellar peduncle, which are the only cerebellar efferent pathways. In our patients with a cerebellar lesion in the PICA territory, only 2 of 7 cases showed limb ataxia. In both cases the ICP in the medulla was damaged. This suggests that this structure must also be considered when evaluating limb

Figure 1 Comparison of lesions with and without limb ataxia. Axial T2, 1 T MRI findings, at the level of the inferior olivary nucleus in the medulla, representative of the four patterns. (A) Pattern 1, patient No 1, without limb ataxia. T2 weighted MRI showing an infarct in the right cerebellar hemisphere in the posterior inferior cerebellar artery (PICA) territory, sparing the medulla. (B) Pattern 2, patient No 8, with limb ataxia. T2 weighted MRI showing an infarct in the right cerebellar hemisphere in the PICA territory and in the dorsolateral medulla, involving the inferior cerebellar peduncle (ICP). (C) Pattern 3, patient No 10, with limb ataxia. T2 weighted MRI showing an infarct in the dorsolateral medulla, involving the ICP. (D) Pattern 4, patient No 15, without limb ataxia. T2 weighted MRI showing an infarct in the dorsolateral medulla, sparing the ICP. (E) Schematic presentation of important structures in a transverse section of the medulla through the inferior olivary nucleus. The crucial structure for the occurrence of limb ataxia is the ICP in the medulla.

ataxia in PICA stroke. When looking at studies of PICA infarct cases, where it was possible to correlate the presence or absence of limb ataxia with the histopathological or MRI findings,^{9–11} it appears that, in the majority of cases, limb ataxia is present when PICA infarct is associated with a dorsolateral medullary infarct involving the ICP. Only very few cases of PICA stroke presented with limb ataxia without clear evidence of ICP lesions.¹¹ There are two explanations for the presence of limb ataxia in isolated cerebellar hemispheric lesions. Firstly, it may depend on the involvement of the cerebellar output—namely, the cerebellar nuclei—as suggested by the authors, because of a variant boundary between the PICA and the SCA territory.⁶ Alternatively, it might be due to indirect involvement of the cerebellar input, the ICP, because of compression, from swelling of the infarcted cerebellar hemisphere, or because of decreased perfusion. Several lines of evidence indicate that the ICP is an important structure in the control of limb coordination. Indeed, lesions of the ICP in mammals produce limb ataxia.¹² In the clinical domain, limb ataxia and impairment of the ICP have been correlated in Wallenberg's syndrome without associated cerebellar involvement.⁷ Among our four cases of Wallenberg's syndrome, two had limb ataxia and ICP involvement.

The data of the present study suggest that limb ataxia in acute P-PC territory infarcts occurs not only when the efferent pathways from the intermediate and lateral cerebellar cortex are impaired (ie, when PICA stroke involves the dentate/interposed nuclei, which are usually supplied by the SCA). Rather, it is also associated with impairment of the afferent pathways (ie, when PICA stroke is associated with a dorsolateral medullary infarct, involving the ICP, which is supplied by small branches which arise from the ICVA).

Our findings cannot be regarded as conclusive because the patient sample was too small in number, the vascular lesions were not systematically assessed and the aetiology remained unknown in some cases. This issue has to be further addressed in a large series of patients using highly sensitive MRI techniques to better evaluate the entire P-PC.

An important clinical implication from the present study is that the absence of limb ataxia cannot exclude the presence of a PICA territory infarct, a serious event which may be complicated by cerebellar oedema, brainstem compression which requires a neurosurgical intervention.^{9–13} In this condition, the more reliable clinical test is to assess gait because gait ataxia is a common sign associated with stroke in the PICA territory. In fact, the PICA supplies structures, such as the nodulus and the inferior half of the vermis, which are ancient in the phylogenesis and are involved in the control of balance. All of our patients with PICA stroke (patient Nos 1–9) presented with gait ataxia.

The outcome in our patients was favourable. Among the patients whose follow-up was available (13 patients), 12 had no symptoms or only slight symptoms that caused no or slight disability (mRS 0–2). This is consistent with the observation reported by Graf and colleagues⁸ showing no or minor disabilities from their P-PC territory infarcts in a large series of patients.

In conclusion, when correlating limb ataxia and acute P-PC territory infarcts, it is important to take into account the entire ICVA territory (ie, the posterior inferior cerebellar (supplied by the PICA)) and the dorsolateral medullary (fed by the small branches which arise from the ICVA).

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