

Pure motor hemiparesis due to occlusion of the internal carotid artery

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To the Editor-in-Chief,

Pure motor hemiparesis (PMH), also known as pure motor stroke, is one of the classical lacunar syndromes, along with pure sensory stroke, sensorimotor stroke, ataxic hemiparesis, and dysarthria-clumsy hand. It is considered as the most common lacunar syndrome in clinical practice, accounting for one-half to two-thirds of cases. The clinical criteria for diagnosis of PMH include the presence of unilateral partial or complete paresis involving at least two of three body areas (face, upper limb, or lower limb) and no evidence of aphasia, apraxia, and agnosia, or visual field defect, eye movement disturbance, ataxia, sensory loss or evidence of bilateral weakness [1]. This lacunar syndrome is commonly due to lacunar infarct resulting from small vessel disease. We present a rare case of PMH not due to a lacunar infarct, following an occlusion of the contralateral internal carotid artery.

An 83-year-old man with a previous history of arterial hypertension, heavy smoking (>20 cigarettes/day), obesity and dyslipidemia presented to our department with motor disturbance affecting his right brachio-crural distribution, lasting 24 h. Symptoms developed suddenly, and there

were no headache, nausea, vomiting, or speech disturbances. Sensory disturbances in upper or lower extremities or in ipsilateral thoraco-abdominal anatomy were absent. He had neither nuchal rigidity nor semiology of meningeal syndrome. The patient had a proportional right arm and leg weakness with movement against gravity, and Babinski sign, compatible with pure motor hemiparesis (modified Rankin Scale = 2, NIHSS = 5).

The following investigations were normal or unremarkable: chest roentgenography, 12-lead electrocardiography, brain CT scan, two-dimensional echocardiography, complete hematological screening, routine biochemical profile, urinalysis, serology for syphilis (VDRL), immunological blood test (including ANA, ENA, lupus anticoagulant, IgG and IgM anticardiolipin antibodies, and rheumatoid factor), and basic hemostasis study.

Cerebral magnetic resonance imaging (MRI) at day 3 post-stroke showed increased signal on FLAIR and T2-weighted sequences, and diffusion-weighted imaging revealed restriction involving, partially, the left frontoparietal cortex compatible with an acute infarct in the primary motor cortex (Fig. 1). Echo-Doppler of the supra-aortic trunks and angio-MRI showed thrombotic occlusion at the origin of the left internal carotid artery, with mild right carotid artery stenosis (<30 %) clinically silent.

Antithrombotic therapy with bempiparine (7500 UI/24 h sc) was prescribed for secondary stroke prevention together with strict control of cardiovascular risk factors, and intensive physiotherapy. The clinical course was favorable. A gradual regression of symptomatology was observed, and 21 days after the onset of stroke, focal neurological deficit was partially recovered.

PMH has been reported from autopsied cases as a focal small (<20 mm in diameter), deep infarction, involving the

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Fig. 1 *Left* cortical frontoparietal infarct revealed on the MRI performed 3 days post-stroke: increased signal on FLAIR (a), and T2-weighted sequences (b), restriction on diffusion-weighted imaging (c), and *left* extracranial internal carotid artery occlusion on MRI angiography (d)



Table 1 Pure motor hemiparesis not due to lacunar infarcts

Non-lacunar ischemic stroke ^a
Cardioembolic infarct
Atherothrombotic infarct
Internal carotid artery occlusion in the neck
Ventromedial pontine infarction due to a propagating thrombosis of the basilar branch
Complex aortic atheroma plaques
Ischemic stroke of unusual etiology
Ischemia-edema after craniotomy for postoperative bleeding
Intracerebral hemorrhage
Subdural hematoma
Brain abscess of the motor cortex
Cerebral metastasis
Demyelinating disease

^a Cerebral cortical surface infarction or large deep infarction (>20 mm diameter)

internal capsule, corona radiata, pons, and medullary pyramid [1]. Other topographies are centrum semiovale, basal ganglia, and mesencephalon. Rare causes of PMS

[2, 3] are shown in Table 1. Among these unusual causes, we highlight the internal carotid artery occlusion in the neck, as the clinical case we describe. These unusual causes can, nowadays, be easily detected by imaging techniques.

In a previous clinical study [4] of 222 patients with PMH, we found 189 (85 %) lacunar infarcts, whereas ischemic lacunar syndromes not due to lacunar infarcts occurred in 23 (10.4 %) (atherothrombotic stroke in 12, cardioembolic stroke in 7, infarction of undetermined origin in 3 and infarction of unusual etiology in 1), and hemorrhagic lacunar syndromes in 10 (4.5 %). However, in that study, no pure motor hemiparesis was due to occlusion of the carotid artery.

Aleksic and George [5] presented two similar case reports of pure motor hemiplegia with occlusion of the extracranial internal carotid artery and suggested that hemodynamic cerebral ischemia was likely to be an important manifestation of this process.

In conclusion, our case shows that extracranial internal carotid artery occlusion is a rare etiology of PMH, and may

play a role in the pathogenesis of the clinical lacunar syndrome of pure motor hemiparesis.

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Compliance with ethical standards

Conflict of interest None declared.

Ethical approval All procedures performed in this study were in accordance with the ethical standards of the institutional research committee and with the Helsinki declaration; no formal ethical review was required.

Informed consent Informed consent was obtained from the individual participant included in the study.

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