SHORT REPORT

Middle cerebral artery territory infarction sparing the precentral gyrus: report of three cases

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We report three patients with large middle cerebral artery infarctions in the non-dominant hemisphere, with striking recovery of motor function. In each case this excellent functional outcome correlated with selective sparing of the motor cortex in the precentral gyrus. We discuss some of the possible circulatory variants that might underlie this pattern of infarction.

Infarctions in the middle cerebral artery (MCA) territory may present with different clinical features depending on which divisions or branches are occluded and on the extent of the infarct. If the anterior (superior) division is involved, the most common consequences are contralateral hemiparesis and hemisensory loss. In addition, aphasia usually accompanies lesions in the left hemisphere, whereas sensory neglect phenomena and anosognosia accompany right hemispheric lesions.^{1,2} Here we provide clinical descriptions of three cases of large MCA infarctions in the non-dominant hemisphere that spare the motor strip (precentral gyrus; PCG) resulting in surprisingly little or no weakness within a few days after the initial onset of symptoms.

CASE 1

A 54 year old right-handed smoker with hypertension and diabetes presented with acute onset of right gaze deviation, lethargy, and left hemiparesis. He had prominent visual neglect and sensory loss over the left side and could not move his left arm or leg on command (NIHSS=22). However, when his arm was placed in his preserved field of vision he could move it with near normal (4+/5) strength. A head CT revealed no acute infarcts. He was given intravenous tissue plasminogen activator within three hours of symptom onset. Follow up brain MRI showed a full territory right MCA infarct, with sparing of the PCG (fig 1A, B). Twenty-four hour Holter, carotid ultrasound, and magnetic resonance angiography (MRA) of the head and neck were unremarkable. Echocardiography showed a dilated left ventricle with an ejection fraction of 0.37 and focal areas of hypokinesia. On transfer to a rehabilitation hospital, he could look past the midline towards the left and move his left side spontaneously with full strength (Barthel Index=65).

CASE 2

A 67 year old right-handed woman with a history of hypertension and previous strokes, was found in a state of collapse at home. In the emergency room she had slurred

Figure 1 Three cases of MCA territory infarction sparing the precentral gyrus (PCG). (A) Case 2, brain MRI-DWI reveals an infarct in the right MCA territory that spares a strip of grey matter corresponding to the PCG. (B) Schematic outline of the extent of MCA infarcts for all three patients. The perimeter of the infarcts was traced on MRI or CT scans for each patient. Shaded areas represent infarcted tissue. Arrows point to the contralateral central sulcus.

Abbreviations: MCA, middle cerebral artery; MRA, magnetic resonance angiography; PCG, precentral gyrus

Figure 2 Diagram of the normal and variant PCG arterial supply. Inferofrontal views are depicted. The right temporal lobe has been retracted in order to reveal the proximal branches of the middle cerebral artery. The frontal lobes have also been retracted laterally to reveal the course of the anterior cerebral arteries and their branches. In the variant PCG arterial supply an extension of the medial Rolandic artery (also known as paracentral artery), a branch of the anterior cerebral artery, extends laterally to supply the region of the motor strip, normally supplied by branches of the lateral Rolandic artery.

speech without aphasia, right gaze preference with incomplete left hemianopsia, left sensory hemi-neglect, and moderate (4–/5) weakness of the left arm but milder (4/5) weakness of the left leg (NIHSS=12). Head CT and brain MRI revealed two small old infarcts (left cerebellum, right posterior parietal area), and a new complete right MCA infarct, with sparing of the PCG (fig 1B). Head MRA showed patent anterior cerebral arteries (ACA), and attenuated flow-related signal in the right MCA beyond the M1 segment, while its superior division was not visualised. Twenty-four hour Holter, neck MRA, and carotid ultrasound were unremarkable. Echocardiography demonstrated mild dilation of the left atrium with left ventricular hypertrophy, and ejection fraction of 0.57. When she was discharged to a rehabilitation facility, her strength was 4–/5 in the left arm and 4+/5 in the left leg. (Barthel Index=30).

CASE 3

A 32 year old right-handed smoker with AIDS and a history of migraines with aura, presented seven hours after the acute onset of left hemiparesis and headache. She admitted to having used cocaine the night before. She was somnolent and followed commands intermittently. She was dysarthric and had left facial weakness as well as left visual and sensory hemineglect, but without anosognosia. Her left arm strength was severely decreased (2/5), while her left leg strength was less severely affected (4/5 proximally, 3/5 distally; NIHSS=14). A head CT showed a large right fronto-parietal hypodensity consistent with an infarct in the anterior division of the right MCA. A brain MRI three days later showed significant sparing of the right PCG (fig 1B) and a head MRA showed a sharp cut off of the right MCA. Echocardiography was normal except for the presence of a patent foramen ovale. Lower extremity doppler studies did not show deep venous thrombosis. In addition to possible cocaine induced vasospasm, she may also have had systemic hypercoagulability due to a protein S deficiency. On transfer to a rehabilitation facility, her strength had improved markedly (left arm 4/5 proximally and 4+/5 distally; left leg 4/5 throughout). (Barthel Index=85).

DISCUSSION

Good functional motor recovery after large MCA territory infarctions has been shown to depend at least in part on the sparing of the premotor cortex, presumably because of its involvement in motor function and particularly its role in the control of the contralateral proximal musculature.^{4,5} For example, in a series of seven cases of good recovery after MCA strokes, the preservation of motor function was postulated to be due to sparing of the premotor cortex.⁶ We report three patients with non-dominant MCA infarctions in whom excellent motor recovery correlated with sparing of the primary

motor cortex. A thorough evaluation of MCA territory infarctions from a large registry (including 238 patients with superior division strokes) did not reveal any cases of motor strip sparing.¹

The lateral PCG contains the motor representation for the contralateral face and arm and is normally supplied by a distal branch of the MCA, the lateral Rolandic artery.² This territory is normally included in all infarctions involving the superior division of the MCA. In order to explain the sparing of PCG observed in three cases of large MCA infarctions presented here, one might invoke at least four possible mechanisms. In one scenario, a shower of small emboli resulting from the break up of a large proximal embolus would block distal branches of the MCA, with the exception of the lateral Rolandic branch that supplies the PCG. This is the proposed mechanism for the so-called “spectacular shrinking deficit” phenomenon (SSD),^{3,4} resulting in small and scattered infarcts seen on neuroimaging.⁷ However, in the three patients presented, the size of infarct as assessed by MRI, was much larger, occupying nearly the entire MCA territory (fig 1B). Moreover, in previously reported SSD cases the hemiparesis was dense initially and improved subsequently,^{3,4} whereas in two of our cases (cases 1 and 2) the motor deficit was minimal at presentation.

In a second scenario, spontaneous local recanalisation of one of the occluded vessels, with or without additional good leptomeningeal collateralisation, could lead to selective restoration of flow to the PCG through the lateral Rolandic branch of the MCA. Recanalisation occurring within the first few days after the acute event is known to improve outcome after MCA infarction. However, this mechanism generally spares tissue in the ischemic penumbra² and not along a strip of gray matter in the core of the infarct as seen in our patients. Moreover, in case 3, a follow up MRA several months after the initial stroke showed a persistent cut off of the right MCA, suggesting that spontaneous recanalisation of the vessel could not explain her rapid recovery or the sparing of the motor strip. For this reason, we feel that the presence of pre-existing collateral circulation, rather than revascularisation, better explains the protection of the precentral gyrus from ischemia.

The PCG could have been preserved due to the presence of an accessory MCA branch. Accessory MCAs arising most frequently from the ipsilateral (or contralateral¹⁰) anterior cerebral artery have been described in approximately 3% of autopsies or angiograms.^{8–10} Interestingly, even though these “anomalies” were felt to be incidental findings, those reports suggested that they might have clinical relevance in the setting of MCA occlusion because they would represent potential means of collateral blood supply.^{9,11} Yet, although the accessory MCAs were felt to supply regions normally supplied by the MCA, these usually corresponded to the lateral part of the orbital surface of the frontal lobe, and therefore do not fit the pattern of sparing we observed in our three patients.⁹

Thus, we propose a fourth possible scenario whereby a variant of the medial Rolandic (paracentral) artery, which is a distal branch of the ACA,¹⁰ would extend laterally over the convexity of the brain to supply the PCG (fig 2). Indeed, anastomoses between this vessel and the lateral Rolandic branch of the MCA are among the known cerebral arterial anastomoses.¹² Unfortunately, we were unable to demonstrate any such variant vessel, probably because its caliber was below the resolution of the imaging modality (MRA), and none of the patients underwent conventional angiography. Thus, the other potential scenarios, including the possibility of an embolus break up with distal migration (scenario 1), cannot be entirely ruled out. Future prospective studies of similar patients with angiography are necessary because they might reveal such circulatory variants that could be exploited for the treatment of stroke.

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