Acute dysarthria, dense left hemiparesis and left sensory neglect: is it striatocapsular infarction?

Osama SM Amin

Department of Neurology, Sulaimaniya General Teaching Hospital, Sulaimaniya City, Iraq

Correspondence to Dr Osama SM Amin, dr.osama.amin@gmail.com

DESCRIPTION

A 52-year-old right-handed hypertensive patient presented with sudden onset of dense flaccid left-sided weakness. There was atrial fibrillation, dysarthria, moderate left-sided facial weakness, left sensory hemineglect and left extensor planter. Her CT brain scan showed a hypodense comma-shaped area at the right basal ganglia. Her brain MRI (figure 1) and MR angiography (figure 2) were obtained on day 2 of admission.

Clinically, the patient seemed to have developed an embolic stroke of the main stem of the right middle cerebral artery (MCA) and one might expect that the whole cerebral territory supplied by the right MCA would have been infarcted.

However, radiologically, the patient had developed right-sided striatocapsular infarction (SCI), which is defined as a comma-shaped infarction at the basal ganglia area of at least 3 cm in length and 1 cm in width as a result of occlusion of the lateral lenticulostriate arteries which stem off the posteriolateral surface of the MCA’s main stem. The overlying cortical areas are spared; although the maximum ischaemia is found at the depth of the hemisphere, but several patients (including ours) demonstrate cortical signs (such as hemineglect). A compromise in the overlying cortical areas, that is not seen by conventional imaging studies, was the suggested mechanism behind the development of cortical signs.

In most patients, dense hemiplegia occurs; the arm weakness is usually more severe than the leg, and this ratio usually persists upon recovery. SCI of the non-dominant hemisphere can result in subcortical dysarthria or hypophonia and hypokinetic speech.

Figure 1 Axial T2-weighted MRI of our patient with right-sided striatocapsular infarction at the level of basal ganglia on day 2 of admission. Note the right-sided hyperintense comma-shaped sign that represents the infarcted right putamen–globus pallidus, anterior limb of internal capsule and part of the head of caudate. On the T1-weighted image, this ‘comma’ appears hypointense. The adjacent thalamus and the overlying cortical areas are ‘normal-looking’. The normal left putamen–globus pallidus (yellow arrow), anterior limb of internal capsule (black arrow) and caudate’s head (red arrow) were labelled for comparison.

Figure 2 Coronal section of the patient’s brain magnetic resonance angiography, who had developed right-sided striatocapsular infarction. Note that the right middle cerebral artery becomes non-visualised suddenly, just beyond its origin (yellow arrow) when compared with the left one (red arrow).
Learning points

▸ Striatocapsular infarction is an uncommon, but special, form of deep hemispheric ischemic stroke with resultant subcortical neurological deficits with dense hemiplegia. The damaged basal ganglia areas display a highly characteristic comma-shaped infarction.

▸ Associated cortical signs are common but conventional imaging studies (eg, CT or MRI) fail to demonstrate their causative cortical ischaemia.

▸ The site of the arterial occlusion is either the proximal part of main stem of the middle cerebral artery or the carotid T junction; therefore, lateral lenticulostrate arterial ischaemia occurs (with resultant extensive basal ganglia infarction) while the pertinent cortical areas are maintained via trans-cortical and trans-dural arterial anastomoses.

Competing interests  None.

Patient consent  Obtained.

REFERENCES