An unusual clinical presentation of ischemic stroke due to carotid dissection: The wrist drop

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Abstract

We report a patient with sudden onset of right wrist drop mimicking radial nerve palsy, found to be due to a borderzone infarct due to spontaneous carotid artery dissection (CAD).

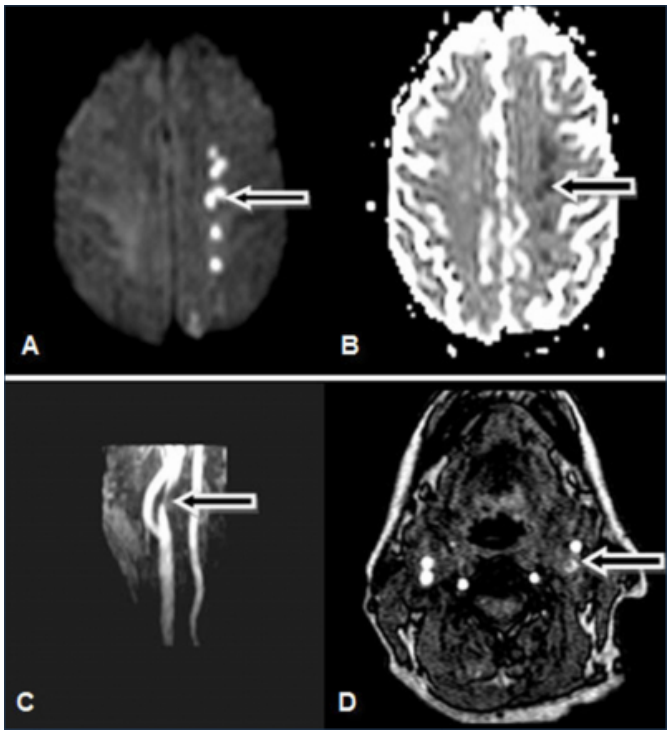
CASE REPORT

A 69-year-old hypertensive woman had sudden onset of right hand weakness. One week before she had experienced left facial and neck pain for which she did not seek medical attention. On admission she had severe paresis of right wrist and finger extensors, along with less severe weakness of finger flexors and interossei muscles. She could not actively extend her interphalangeal joints when the wrist and metacarpophalangeal joints were passively placed in neutral position, which in addition did not lead to improved finger flexion strength. Fine finger movements were slow, and the deep tendon reflexes were brisk on the right side. Sensory exam revealed decreased sensation to pinprick and light touch below the elbow on the right.

Computed tomography (CT) on the day of admission showed a small area of decreased attenuation in the left frontoparietal cortex. Magnetic resonance imaging (MRI) on the same day revealed multiple foci of restricted diffusion consistent with acute ischemic infarcts in the borderzone of left middle/anterior cerebral arteries (Figure 1A and 1B). Magnetic resonance angiography (MRA) showed severe stenosis of the proximal left internal carotid artery (ICA) (Figure 1C), with decreased flow-related signal intensity of the entire left ICA. MRI of the neck (T1-weighted) showed focal dissection in the proximal left ICA (Figure 1D).

Figure 1

Figure.1



A. DWI (+) infarct along the MCA/ACA borderzone (arrow)

B. ADC maps with corresponding low signal (arrow)

C. MRA of left ICA with tapered, “flame-like” stenosis about 2.5 cm distal to its origin (arrow)

D. Axial T1-weighted MRI demonstrating a “crescent sign” (arrow)

The patient was treated with anticoagulation for 6 months, during which time she improved to the point of having

minimal weakness of wrist extension, which was fully resolved by the next 3 months' visit. The CAD remained unchanged on follow-up MRA after 6 months from symptom onset, when she was switched from warfarin to daily aspirin treatment.

DISCUSSION

Spontaneous carotid artery dissection (CAD) accounts for about 2 % of all ischemic strokes. Clinical history and physical examination features that should raise consideration of the diagnosis are the combination of an acute focal neurologic deficit with pain in the neck, face and head, or Horner's syndrome ipsilateral to the affected carotid artery.1 Isolated hand weakness is a rare presentation of ischemic stroke, often mistaken for a peripheral lesion. Although reported as a result of small embolic or large-artery atherosclerotic infarctions involving the representation of the hand in the motor cortex, isolated hand weakness has not been described in the setting of CAD.2-5

The frequency of isolated hand weakness due to acute ischemic stroke is unknown, but it is likely that many cases are initially mistaken for a peripheral lesion. Our patient presented with an isolated wrist drop suggestive of a radial nerve lesion, but left-sided carotydinia suggested the possibility of CAD. More importantly, several clinical features on physical examination led to the recognition of the central origin of the wrist drop: the patient's inability to extend her fingers was accompanied by similar limitation for finger flexion and adduction/abduction, in contrast with the improved activation of median and ulnar nerve innervated muscles in subjects with isolated radial nerve palsy. Similarly, such patients show marked improvement in finger flexion strength with passive positioning of the wrist in the neutral position, which did not occur in our patient. These maneuvers eliminate most of the mechanical disadvantages in radial neuropathy, but do not improve a central type of weakness, which affects hand extensors more than flexors.

Isolated hand weakness has been previously reported as a result of embolic stroke involving the hand knob area,2 large-artery atherosclerotic infarct of vascular borderzones,3 and small subcortical lacunar infarct.4 It has been suggested that "fractional weakness of the hand" is a strong predictor of atherosclerotic infarction affecting vascular borderzones.5 Our case adds another non-atherosclerotic cause of isolated hand weakness by a mechanism of "artery-to-artery" embolism from CAD. The mechanism of such a small and isolated cortical infarct reflects the likely small diameter of the emboli, allowing them to be arrested in the smallest caliber arteries of the distal MCA territory, in close proximity to the anatomical MCA/ACA borderzone.6

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