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Aortic Dissection Extending Into Bilateral Common Carotid Arteries

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A 56-year-old man presented with extensive Stanford type A aortic dissection (Figure 1A&B). The right common carotid (CCA; Figure 1C) was noted to have a false lumen without significant intracranial arterial stenosis (Figure 1D) or cerebral infarction (not shown).

Two days after emergent repair of his ascending aorta and resuspension of the aortic valve, he was noted to have transient 3/5 weakness in his right arm as well as neglect to his right side. Repeat CT imaging demonstrated a left parietal infarct (not shown) and now there was a new dissection of the left CCA (Figure 1E&F). He was anticoagulated with heparin but continued to have fluctuating 0/5 to 3/5 hemiparesis, right-sided neglect, and global aphasia. Repeat CTA of the head on post-operative day four demonstrated flow attenuation in respective A1 and M1 segments of the left anterior and middle cerebral arteries (Figure 1G) as well as occlusion of several branches of the left middle cerebral artery (not shown). CT and CT-perfusion revealed an extensive area of ischemia in the previously non-infarcted MCA territory without new infarction (Figure 1H-K).

On follow-up 4 months postoperatively, he displayed residual moderate right hand dyspraxia and paresthesia as well as almost full recovery of aphasia. Repeat CT of the head demonstrated previously noted parietal infarction (Figure 1L) and brain MRI showed two previously not visualized small lesions (Figure 1N&O). MR-angiogram showed complete recanalization of the left MCA (Figure 1M), and left CCA dissection without definite right CCA dissection (not shown).

Aortic dissection is a rare and potentially fatal disease, with an estimated incidence of 5-30 individuals per million per year.[1,2] Cerebral ischemia is the most frequently observed neurological complication and ischemic stroke has been referred to the anterior circulation in up to 81% of cases.[1] Carotid dissection, frequently bilateral, may be observed in as many as 15%-41%; however, signs of focal cerebral ischemia do not develop in the majority of cases.[1,2] Though successful emergent revascularization of carotid arteries after

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aortic dissection repair has been reported,[3,4] medical treatment remains the first-line management such as for isolated spontaneous cervical artery dissection with probable clinical equipoise for antiplatelet and anticoagulant therapy.[5]

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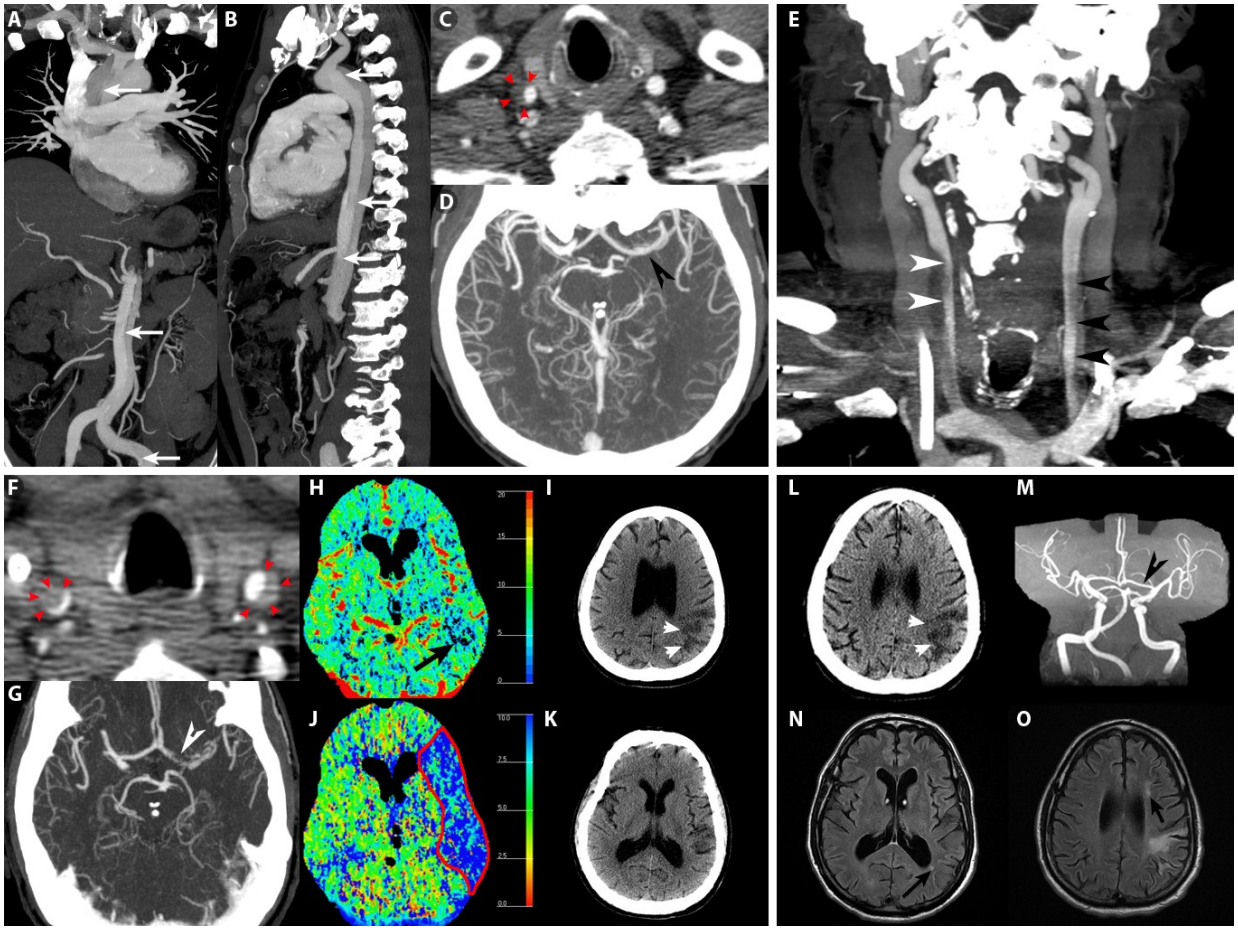

Figure 1: (A, B) Stanford type A aortic dissection (white arrows) on CT-angiogram. (C) Right common carotid artery (CCA) dissection with false lumen denoted by red arrowheads. (D) Patent left middle cerebral artery (MCA, arrowhead). (E) Old right (white arrowheads) and new left CCA dissection (black arrow heads) with (F) false lumen (red arrowheads) on postoperative day two. (G) Left MCA stenosis (white arrowhead). (J) Mean transit time (MTT) indicates extensive ischemia with (H) corresponding small ischemic core on the cerebral blood volume map (CBV, black arrow). (I, K) small subacute parietal infarct on postoperative day four. Four months postoperatively, CT demonstrates (L) previously noted parietal infarct and (M) MRA shows complete MCA recanalization. (N, O) MRI identifies two small new infarcts (arrows) in addition to previously visualized infarcts on CT.