Corpus Callosum Stroke

A Rare Localization for an Isolated Cerebral Infarction

A rare case of isolated corpus callosum infarction secondary to recurrent atherosclerotic carotid disease.

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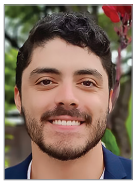


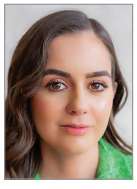
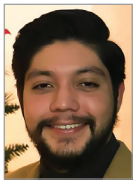

Case Presentation

NM, aged late 60s, a taxi driver and current smoker, presented to the emergency department with a 6-day evolution of right-sided facial and limb weakness and moderate holocranial headache. Clumsiness in both hands in performing daily tasks such as eating or dressing also was reported as well as an overall impaired speech output that was interpreted by the family as being emotionally related. NM had a history of untreated chronic hypertension and nonreperfused left middle cerebral artery infarction, which had been diagnosed 3 months previously at another institution without etiologic identification, but NM had experienced an almost complete recovery of motor symptoms.

On physical examination, NM was awake and alert. Speech was labored and slow with phonemic paraphasias. There were no impairments in general language comprehension or various memory modalities. Right-sided mild hemiparesis of the face and limbs with ipsilateral exteroceptive and proprioceptive hypoesthesia was noted as well as bilateral ideomotor apraxia of the upper limbs with marked dominance of the left hand as evidenced by the inability to perform transitive and intransitive gestures triggered by verbal, visual, or tactile modalities. National Institutes of Health Stroke Scale score was calculated as 3 points (1 point for facial weakness, 1 point for motor function of the right upper hand, and 1 point for aphasia). No signs of meningeal irritation were elicited, and no other abnormalities were noted on general examination.

Diagnostic Process

Vital signs were normal on admission, and apart from mild hyperglycemia (146 mg/dL), no other relevant abnormalities were noted on blood test results, including C-reactive protein and erythrocyte sedimentation rate. SARS-CoV-2 antigen test and polymerase chain reaction testing were negative. Initial CT angiography of the head showed a mildly hypodense area in the left white matter with watershed distribution and a large vessel occlusion in the first segment of the left internal carotid artery, with predominant visualization of the right anterior circulation, suggesting detour of blood flow away from the infarct site (Figure 1). The contralateral carotid system showed only mild (30%) calcified plaque in the distal right common carotid artery.

T2-weighted MRI and T2-weighted fluid-attenuated inversion recovery sequences showed a subacute ischemic stroke in the corpus callosum (CC), predominantly on the left side, involving all its portions except the rostrum (Figure 2), as well as a left hemispheric luxury perfusion phenomenon (Figure 3).

Case Resolution

After complementary studies were obtained, the diagnosis of left anterior watershed cerebral infarction with isolated recurrence in the CC, secondary to carotid occlusion caused by atherosclerosis, was concluded. Secondary prevention with smoking cessation, hypertension treatment, salicylic acid 100 mg once daily, and atorvastatin 80 mg once daily was initiated. Carotid revascularization was not considered because total occlusion of the proximal internal portion

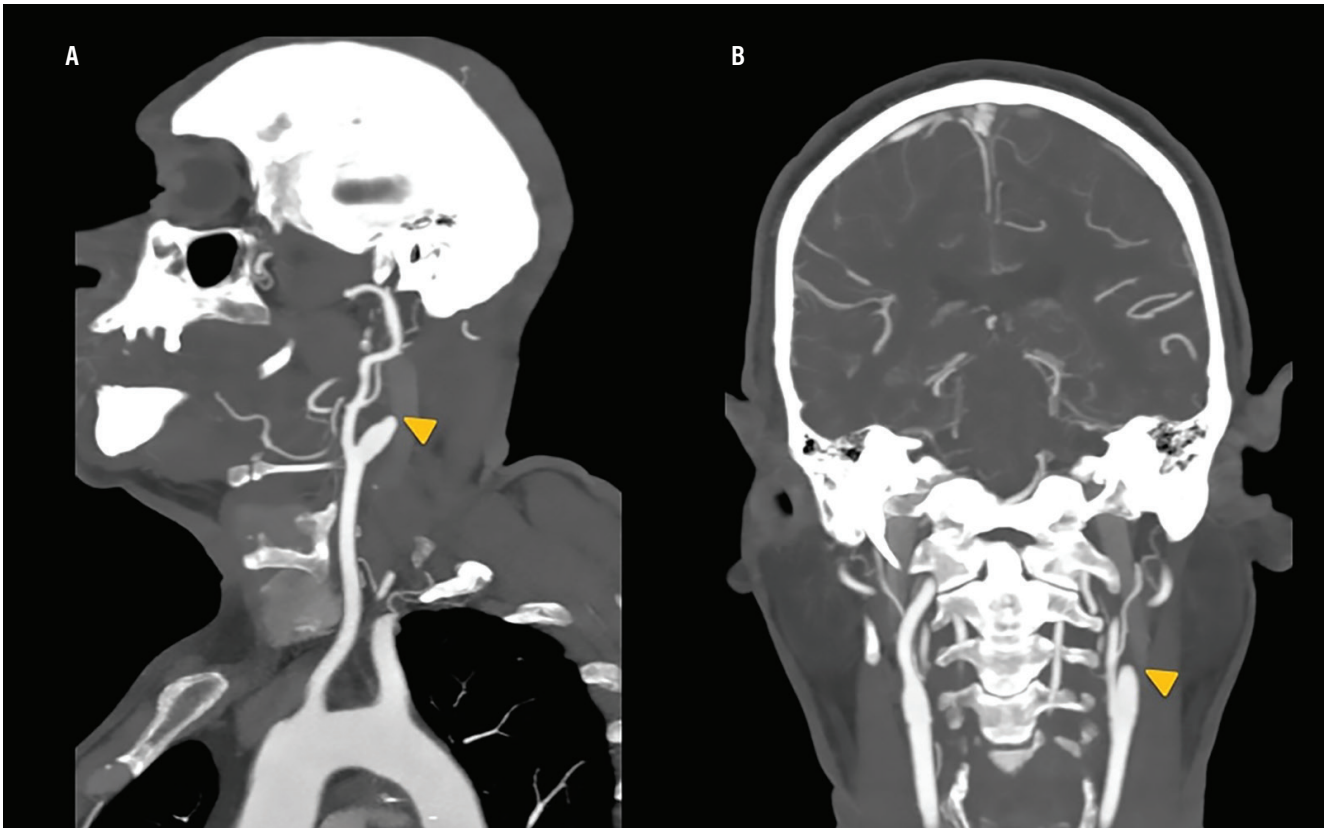

Figure 1. Sagittal (A) and coronal (B) CT angiography show a complete occlusion of the proximal cervical segment of the left internal carotid artery (arrowheads).

was documented and there was no further neurologic deterioration. The presence of callosal apraxia—a peculiar limb apraxia, which contrary to most is more evident in the nondominant hand—is a typical finding in lesions involving the body and posterior parts of the CC. The mild right hemiparesis and hypoesthesia findings were probably related to NM's nonreperfused left middle cerebral artery infarction.

The apraxia eventually compromised the NM's functional state. After the fourth day of hospitalization, NM requested a voluntary discharge and did not attend the outpatient clinic for follow-up.

Discussion

The CC is rarely infarcted in isolation because it is considered a borderline arterial irrigation zone with a broad number of anastomoses between the anterior and posterior cerebral arterial systems.[1] CC infarction has an estimated prevalence of 0.49% to 7.9% of all ischemic strokes.[2,3]

Pathogenesis

The internal carotid artery irrigates most of the CC through the pericallosal branch, which originates from the anterior cerebral artery. The exception is the splenium of the CC, which is supplied by the terminal and choroidal branches of both the posterior cerebral artery and the anterior cerebral artery.[4] Anastomoses between both anterior and posterior systems create the pericallosal pial plexus, originating from perforator branches that follow an almost right-angle path from proximal arteries. This particularity, associated with the intrinsic resistance of the small arteries to atherosclerosis, could explain the low frequency of isolated CC infarcts attributable to lacunar syndromes and embolism.[4] The primary etiology of CC infarction is large vessel atherosclerosis; however, other pathogeneses have been recognized.[2]

After an ischemic stroke, the luxury perfusion phenomenon is associated with failure of autoregulation mechanisms and blood–brain barrier dysfunction.[5] This is described as a state of increased brain flood flow caused by the excessive oxygen and metabolic demands of an infarcted brain zone. It can occur spontaneously or as a result of revascularization procedures. Other findings associated with hyperperfusion include the fogging effect seen in subacute chronic stroke.[6] Perfusion abnormalities have been reported to correlate with a worse prognosis, as post-thrombolysis perfusion-weighted imaging showing a good perfusion is associated

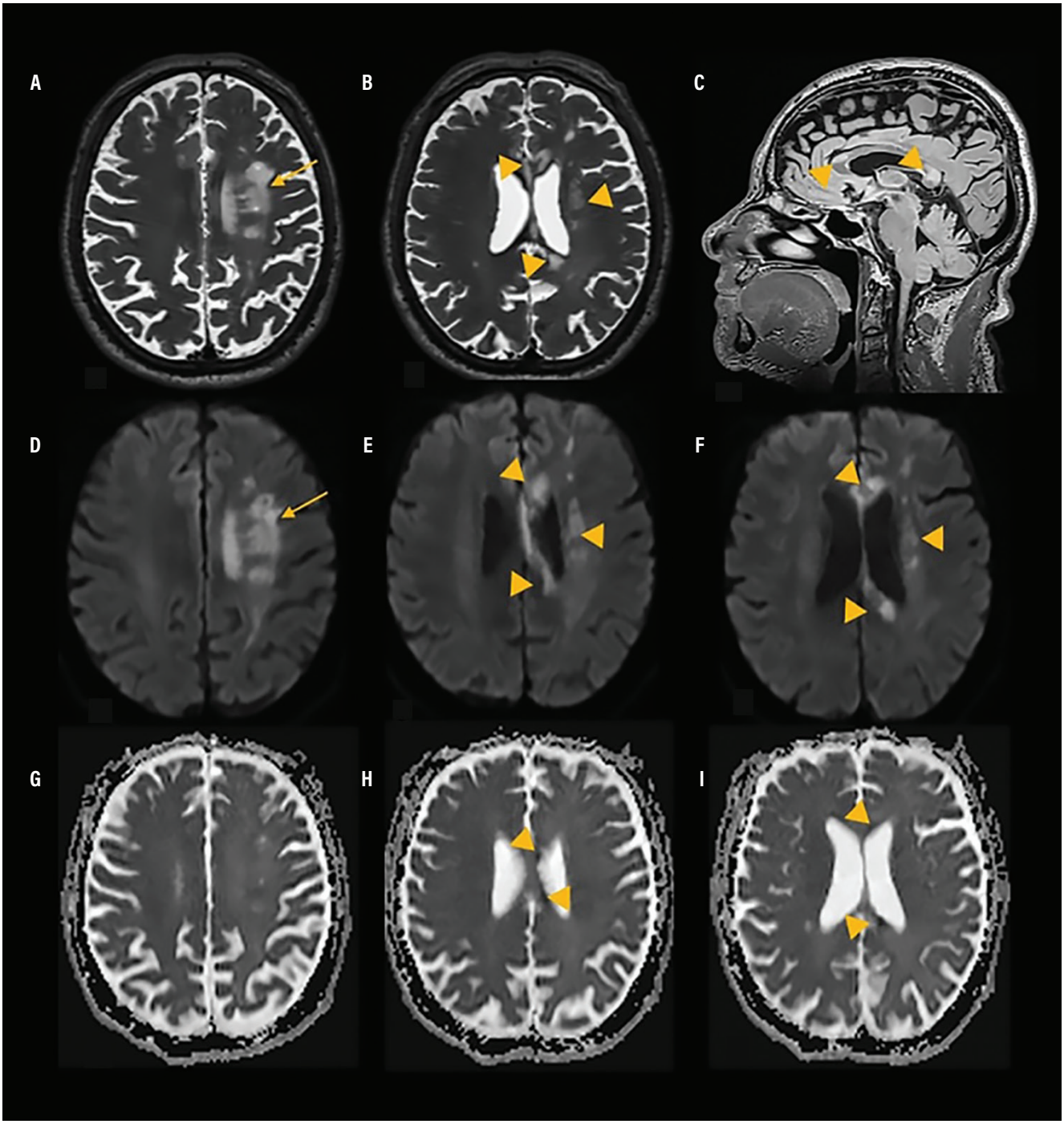

Figure 2. Axial T2-weighted fluid-attenuated inversion recovery (A, B) and T2-weighted MRI (C) show hyperintense lesions involving the left centrum semiovale (arrow) and genu, body, and splenium of corpus callosum (CC; arrowheads). Axial diffusion-weighted imaging (D-F) demonstrates restricted diffusion of the left centrum semiovale (arrow) and CC (arrowheads). Apparent diffusion coefficient mapping (G-I) shows normalized restricted diffusion but a mild persistence of signal reduction in CC (arrowheads).

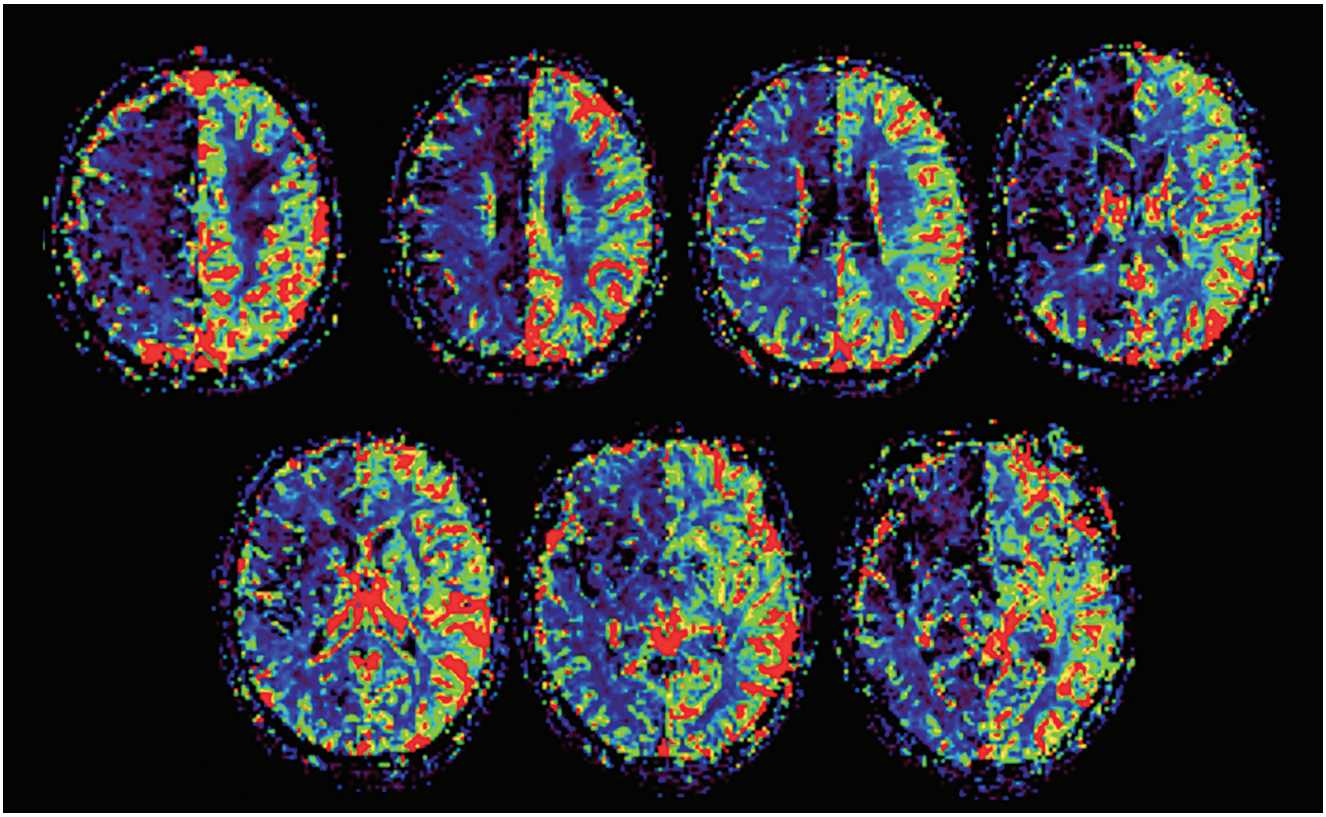

Figure 3. Axial MRI perfusion images of cerebral blood flow show substantially increased perfusion in the left cerebral hemisphere attributable to luxury perfusion.

with recovery, even in patients with non-early clinical improvement.[7]

Caution is needed when excluding other possibilities regarding isolated lesions of the CC, even if there is a suspicion of cerebral infarction. Central nervous system vasculitis is an important differential diagnosis in patients with recurrent cerebral infarcts and indolent headaches. Despite the usually heterogeneous clinical presentation, most cases have a subacute course, with headache, cognitive decline, or focal neurologic deficits, although hyperacute onset secondary to arterial stroke is rarely described.[8] The presence of symptoms distributed on only 1 side and associated with a major arterial pathway in an individual with multiple high-level cardiovascular risk factors and imaging signs of a more common etiology (eg, large vessel atherosclerosis, watershed distribution of infarction), and the absence of signs associated with a vasculitic etiology (eg, only 1 affected side without hemorrhages and without segmental stenosis of multiple arterial branches), may help exclude central nervous system vasculitis with certainty.[8] Cerebral venous thrombosis (mainly superior sagittal sinus and deep vein thrombosis) must be considered in patients with acute motor and cognitive symptoms, especially in the context of the COVID-19 pandemic, and with headache as an accompanying symptom.[9,10] Our patient, however, did not have any data suggestive of cerebral venous thrombosis on MRI venogram or any positive test or clinical sign of COVID-19. Other conditions frequently involving the CC include multiple sclerosis and other demyelinating disorders (eg, transient lesions of the splenium[10]), central nervous system lymphoma, traumatic brain injury, seizures, and carbon monoxide poisoning, among others. Nonetheless, the lack of clinical, imaging, and laboratory findings supporting these diagnoses made them less feasible in our case.

Clinical Particularities

Isolated CC neurologic syndromes are a rare manifestation of stroke attributable to the common coexistence of ipsilateral or contralateral lesions elsewhere. Classic CC disconnection syndromes, characterized by a combination of apraxia of the limbs (predominantly of the nondominant side), accompanied by alexia without agraphia and alien hand syndrome, have been rarely described in vascular lesions of the CC.[11] An individual with a left occipital infarction that

involved the ipsilateral splenium of the CC attributable to posterior cerebral artery occlusion and an ipsilateral transverse and sigmoid venous sinus thrombosis recently was reported in the context of active SARS-CoV-2 infection.[12]

In the largest cohort of individuals with CC infarction to date, Sun et al[13] reported on 127 participants (with or without extracallosal involvement), in whom up to 44% presented with cognitive, psychiatric, or sleep disturbances, in addition to the motor and sensory symptoms commonly reported with any other localization. The median time to referral to a specialized center was 24 hours, and a negative head CT scan in the first 24 hours had a false-negative rate of 76.4% compared with diffusion-weighted MRI. The most common localization in isolated CC infarctions was the splenium.[13]

Conclusion

Despite the difficulty in establishing a clinical diagnosis of a vascular injury of the CC, physical examination (to seek upper limb ideomotor apraxia with predominance in the nondominant hand) and imaging features (to examine T2-sequence hyperintensities with restricted diffusion in diffusion-weighted imaging and apparent diffusion coefficient) can help illuminate the correct diagnosis. A careful review of MRI findings and changes in brain perfusion can lead physicians to an accurate diagnosis for individuals who present with ischemic stroke in the setting of atypical clinical manifestations or arterial territories. ■

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Disclosures

The authors report no disclosures.