Hypoglossal nerve damage following carotid endarterectomy

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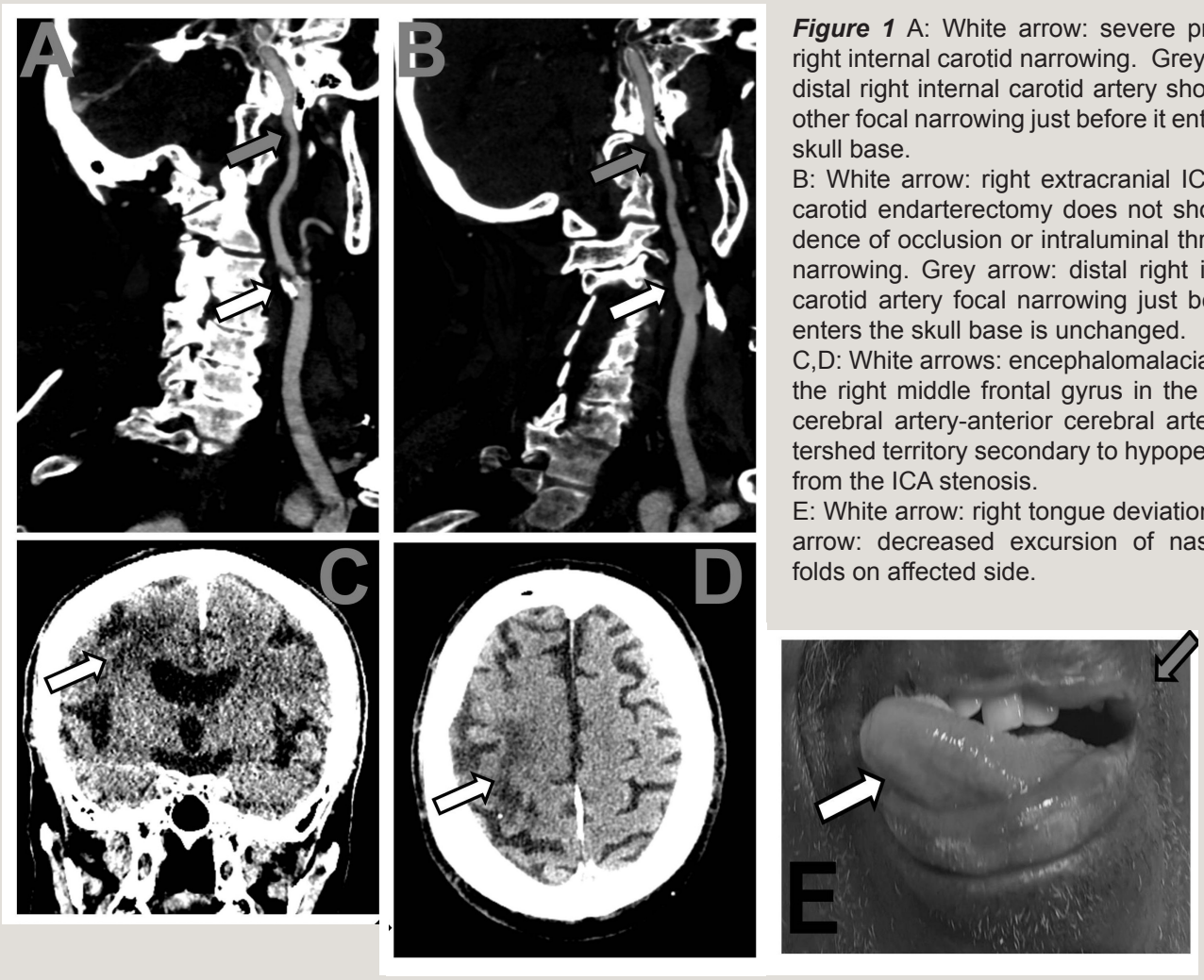

Figure 1 A: White arrow: severe proximal right internal carotid narrowing. Grey arrow: distal right internal carotid artery shows another focal narrowing just before it enters the skull base.
B: White arrow: right extracranial ICA after carotid endarterectomy does not show evidence of occlusion or intraluminal thrombus narrowing. Grey arrow: distal right internal carotid artery focal narrowing just before it enters the skull base is unchanged.
C,D: White arrows: encephalomalacia within the right middle frontal gyrus in the middle cerebral artery-anterior cerebral artery watershed territory secondary to hypoperfusion from the ICA stenosis.
E: White arrow: right tongue deviation; Grey arrow: decreased excursion of nasolabial folds on affected side.

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An 88-year-old man was admitted with left facial droop (Figure E grey arrow) and left upper extremity weakness. The patient had several risk factors for carotid atherosclerosis, including high cholesterol, hypertension, history of smoking, and diabetes. A computed tomography (CT) angiogram showed tandem stenosis (one area of stenosis followed by another area of stenosis) of right

internal carotid artery (Figures A, B) likely caused by atheromatous plaque formation. The tandem stenosis of right internal carotid artery resulted in a middle cerebral artery-anterior cerebral artery watershed territory infarct secondary to hypoperfusion (white arrows, Figure C, D). The patient had worsening weakness on the left upper extremity at systolic blood pressures less than 140 indicating a perfusion-dependent neurological examination. He underwent carotid endarterectomy (CEA) with resolution of the intraluminal thrombus narrowing of the right ICA (Figures, white arrows, A: before; B: after CEA). He had right-sided tongue deviation post-surgery indicative of hypoglossal nerve injury (Figure E, white arrow).

New neurological deficits from cranial nerve or other nerve injuries can arise as an immediate post-operative complication of CEA; new focal neurological deficits can also arise from acute ischemic stroke or cerebral hyperperfusion syndrome following CEA. The incidence of cranial injury following CEA reportedly ranges from 3% to 30%.[1] The Carotid Revascularization Endarterectomy vs Stenting Trial (CREST) trial reported relative frequencies of nerve injuries as hypoglossal nerve in 13%, facial nerve in 16%, glossopharyngeal and vagus nerves in 22%, and sympathetic nerves in 2%.[1] Myocardial infarction and postoperative neck hematoma are non-neurological immediate post-operative complications of CEA.

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