

Cardioembolism: A Rare Cause of Jaw Claudication

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Jaw claudication (JC) results from ischemia of the masticatory muscles, typically caused by temporal arteritis¹ and other arteriopathies affecting the external carotid artery (ECA).^{2,3}

We documented a case of JC resulting from cardioembolic occlusion of the ECA and searched both English and French literature on the Medline database (1966-October 2006) to identify previous reports. We combined the keywords "jaw claudication" with either "cardiogenic embolism", "cardiac embolism", "cardioembolism", "etiology", "cause", or "carotid occlusion".

A 77-year-old, right-handed man with chronic atrial fibrillation discontinued his oral anticoagulant and underwent toe surgery. Four days later, he experienced new-onset, right-sided jaw pain which was repeatedly provoked by mastication while eating breakfast. This pain did not recur at the next meals. Seven hours after breakfast, he suddenly developed left-sided homonymous hemianopia, paresis, hypoesthesia, and spatial neglect. He denied previous neurological symptoms, shoulder pain or stiffness, and constitutional symptoms. On palpation, his temporal arteries were painless, non-nodular, and pulsated normally. His temporomandibular joints were unremarkable. No carotid artery or cardiac murmurs were detected. Brain CT showed a subacute right middle cerebral artery infarct. Ultrasonography of the right ECA failed to reveal a patent lumen at grey-scale B-mode scanning and demonstrated absence of flow at spectral, power and color Doppler imaging techniques, consistent with total occlusion.⁴ The left ECA was normal. Bilateral internal carotid artery stenoses of $\leq 20\%$ were present. An EKG confirmed atrial fibrillation. Echocardiography did not reveal other sources of embolism or thrombus. C-reactive protein (CRP) was 7.0 mg/L (normal ≤ 5.0). Erythrocyte sedimentation rate (ESR) and levels of white blood cells, hemoglobin, platelets, and fibrinogen were normal. We diagnosed JC resulting from cardioembolic occlusion of the right ECA and cardioembolic stroke. Anticoagulant therapy was reinitiated one week after presentation. This individual did not receive anti-inflammatory or immunosuppressive drugs. Three months after stroke, his CRP had decreased to 5.15 mg/L. Repeat ultrasonography by the same operator revealed normal temporal and external carotid arteries, including a re-canalized right ECA without residual stenosis. This individual experienced no additional symptoms during a clinical follow-up of 24 months. He remains with hemianopia, mild hypoesthesia and sensory neglect on the left side.

Our literature review did not identify any previous reports of JC resulting from cardioembolic occlusion of the ECA.

DISCUSSION

Jaw claudication is typically attributed to temporal arteritis,¹ and more rarely to other chronic arteriopathies affecting the ECA and its branches, including atherosclerosis, medial arterial calcification, amyloid angiopathy, Takayasu's arteritis, Churg-Strauss syndrome, Wegener's granulomatosis, polyarteritis nodosa, and hypersensitivity vasculitis.^{2,3} Several features of this case are consistent with cardioembolic occlusion of the ECA as a mechanism for JC. First, as this patient has atrial fibrillation and had stopped his anticoagulation therapy before surgery, he was at increased risk of cardioembolism. Second, the short interval between his JC and cardioembolic stroke suggests a common pathogenic mechanism for the two processes. Third, the limited duration of JC and the presence of a palpable pulse in the temporal artery suggest rapid collateralization of arterial branches distal to an abruptly occluded ECA. In particular, the internal maxillary artery supplies the masticatory muscles and has numerous important anastomoses with the internal carotid and ophthalmic arteries.⁵ In chronic arteriopathies, JC would generally recur or worsen over time. Fourth, clinical manifestations of systemic vasculitides⁶ and nonspecific laboratory markers of inflammation (e.g., anemia, increased ESR and fibrinogen level) were absent, except for slightly increased CRP at presentation, which can be attributed to a recent surgery. Finally, ultrasonography revealed right ECA occlusion on initial assessment and complete re-canalization on follow-up assessment by the same experimented operator using power and color-flow Doppler techniques. These techniques provide

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complementary color-coded information on blood flow (i.e., density versus direction and mean velocity of flowing blood cells) combined to real-time grey-scale display of the vessel walls and lumen. Because both techniques are highly sensitive for blood flow detection, even in the context of near occlusion,⁷ it is unlikely that a normal flow was missed on initial assessment. Re-canalization without residual stenosis is inconsistent with an underlying chronic stenosing arteriopathy. We therefore suggest that cardioembolic occlusion of the ECA should be considered as a mechanism in individuals with a limited duration of JC, known or suspected emboligenic cardiopathies, and in those lacking evidence of temporal arteritis or other chronic arteriopathies affecting the ECA.

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