

Hyoid bone impingement contributing to symptomatic atherosclerosis of the carotid bifurcation

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ABSTRACT

Symptomatic carotid artery disease caused by hyoid bone compression is rare, although scant reports describe cerebrovascular events due to this mechanical interference leading to entrapment, embolism, dissection, atherosclerotic stenosis, and pseudoaneurysm formation. This report describes a patient presenting with left-sided paresis and paresthesia who was found to have focal right carotid stenosis secondary to impingement of the carotid bulb by an elongated hyoid bone. Whereas previous cases describe hyoid bone resection, we describe successful management with endarterectomy and carotid mobilization without hyoid resection, with long-term follow-up demonstrating a widely patent carotid system without recurrent impingement. (*J Vasc Surg Cases and Innovative Techniques* 2020;6:89-92.)

Keywords: Hyoid bone; Carotid artery; Carotid stenosis; Atherosclerosis; Cerebral infarction

Symptomatic carotid artery disease caused by hyoid bone intrusion is an extremely rare phenomenon. Scant reports have detailed the cerebrovascular events due to mechanical interference of the hyoid bone on the carotid artery leading to entrapment,¹⁻³ embolism,⁴⁻⁹ dissection,^{6,10} atherosclerotic stenosis,¹¹ and pseudoaneurysm formation.¹² In some cases of embolism or dissection, the patients were treated medically,^{4,7,10} whereas other patients with compression, pseudoaneurysm, or stenosis underwent surgical management with partial resection of the hyoid bone. Herein, we present a case of a symptomatic right internal carotid artery stenosis due to unilateral hyoid bone compression of the carotid bifurcation. This was successfully treated with surgical mobilization of the carotid bifurcation, endarterectomy, and avoidance of bone resection. The patient consented to publication.

CASE REPORT

An 81-year-old man with a history of right-sided Hollenhorst retinal plaques, coronary artery disease, hypertension, and hyperlipidemia was evaluated for symptomatic right carotid

stenosis after an episode of left facial and upper extremity paresthesia and paresis. When he was initially evaluated, he was noted to have decreased sensation to light touch in V1, V2, and V3 distributions; there was mild flattening of the left nasolabial fold and mild decreased strength in the left arm. At the time of surgical evaluation, his symptoms had mostly resolved except for some residual difficulty with left hand dexterity.

Magnetic resonance angiography of the brain had revealed cortical infarcts within the right precentral and postcentral gyri. Carotid duplex ultrasound demonstrated moderate to severe atherosclerotic disease at the right carotid bulb (*Fig 1*). The peak systolic velocities corresponded to a 50% to 69% stenosis. As part of preoperative workup, computed tomography angiography of the neck was performed, which, in retrospect, demonstrated proximity of the hyoid bone to the carotid bulb (*Fig 2*). Selective cervicocerebral angiography demonstrated an eccentric, focal stenosis of the carotid bulb (*Fig 3*). The decision was made to proceed with a right carotid endarterectomy.

During dissection of the carotid bifurcation, a large bone prominence was noted to be impinging on the carotid bulb directly at the site correlating with the focal stenosis (*Fig 4*). Further dissection revealed an elongated hyoid bone impinging on the carotid bulb at its posteromedial surface. The fibrous bands connecting the bone to the adjacent vasculature were carefully dissected. This mobilized the artery away from the bone prominence and allowed the remainder of the operation to be performed without further bone intrusion. Endarterectomy revealed focal atherosclerotic plaque that was removed with smooth tapers proximally and distally. No further foci of disease were noted. Completion Doppler interrogation confirmed an adequate high-flow, low-resistance signal in the internal carotid artery and a biphasic external carotid artery signal. After mobilization and dissection of the extensive fibrous bands attaching the bone to the carotid bulb, the final orientation of the endarterectomized and patch angioplastied carotid bifurcation revealed a relaxed configuration without further impingement by the bone in various orientations and positioning of the head. Thus, a decision was made to leave the hyoid bone in place without resection.

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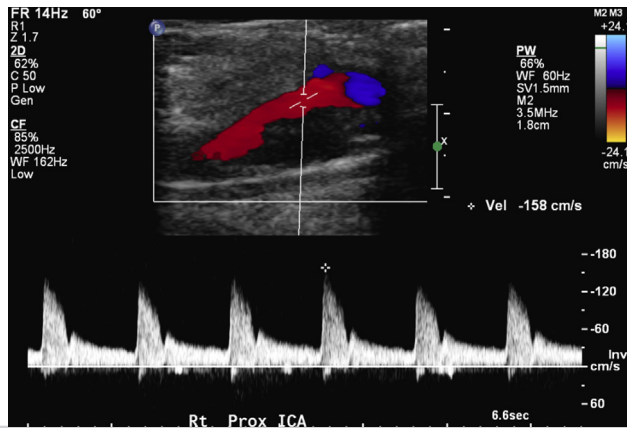


Fig 1. Preoperative Doppler spectrum with color flow image of right proximal internal carotid artery showing elevated peak systolic velocity and internal carotid artery-common carotid artery ratio of 1.9 corresponding to a 50% to 69% stenosis.



Fig 2. Three-dimensional reconstruction of computed tomography angiogram showing the right carotid artery and the proximity of the hyoid bone to the carotid bulb.

The patient tolerated the procedure well without complications and was discharged home on postoperative day 1 per standard protocol. Histologic evaluation of the plaque demonstrated calcified atheroma. On follow-up ultrasound 3 and 15 months after surgery, a widely patent carotid system was noted without recurrent stenosis or impingement.

DISCUSSION

In this report, we describe a case of cerebral infarction presumably related to hyoid bone impingement on the internal carotid artery causing atherosclerotic plaque



Fig 3. Preoperative selective cervicocerebral angiography image demonstrating a bandlike, focal stenosis of the carotid bulb (arrowhead).

formation. Workup after the cerebrovascular event demonstrated internal carotid artery stenosis on duplex ultrasound, and computed tomography angiography of the neck showed a discrete, bandlike narrowing on the internal carotid artery that corresponded with the hyoid bone impingement on the artery visualized during surgery. It is speculated that the hyoid accelerated plaque formation at the location of its impingement in a patient with risk factors for stroke.

The hyoid bone, a horseshoe-shaped bone that forms attachments with several muscles, aids in tongue movement and swallowing. Its position in the neck relative to the carotid arteries can shift, as evidenced by reports of arterial injury by the hyoid bone after repetitive trauma, such as carrying heavy weights and playing golf.^{2,8,12} The compression of the carotid artery by the hyoid can cause abrupt geometric wall changes to the artery that result in disturbed flow and high wall shear stress

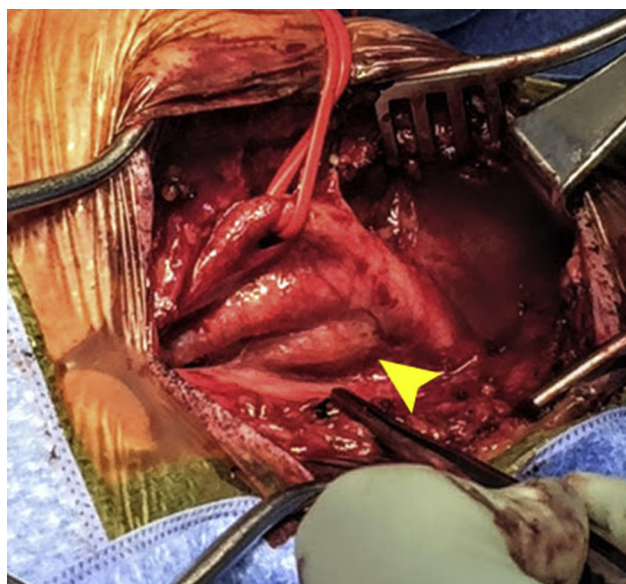


Fig 4. Intraoperative photograph showing a large bone prominence (arrowhead) impinging on the carotid bulb.

gradients.¹³ The susceptibility to atherosclerosis at the carotid bifurcation, a location where the vessel geometry changes from straight to branched, is an example of the correlation between wall stress and atherosclerosis. Previous studies in bovine coronary arterial branches have shown three to four times greater intramural stresses at the branch compared with straight segments,¹⁴ and models of the carotid bifurcation find significantly higher localized circumferential wall stress at the branch point.¹⁵ These mechanical stresses can locally injure the endothelium, which is thought to be the initiating event in atherogenesis.¹⁶ Furthermore, another cause of vasculopathy is the direct compression and subsequent injury of the arterial wall by the hyoid bone; histologic analysis of the arterial wall from patients who have had carotid artery compression by the hyoid show fibrosis⁸ or necrosis and inflammatory changes⁵ in the media. Injury to the arterial wall then accelerates atherogenesis in the setting of a patient with existing hyperlipidemia by increasing endothelial permeability to lipoproteins and smooth muscle mitogens.¹⁷ Increased hyoid bone proximity to the internal carotid artery has also been noted as a risk factor for carotid artery dissection.¹⁸

CONCLUSIONS

Because of the scarcity of reported cases of symptomatic carotid artery stenosis from hyoid impingement, the optimal treatment is unclear. For this patient, after extensive circumferential dissection of the carotid sheath off of the hyoid bone and endarterectomy, the decision was made to forego hyoid resection when we noted that the vasculature was free from all attachments to the adjacent hyoid bone and was no longer experiencing impingement. Previously published cases have

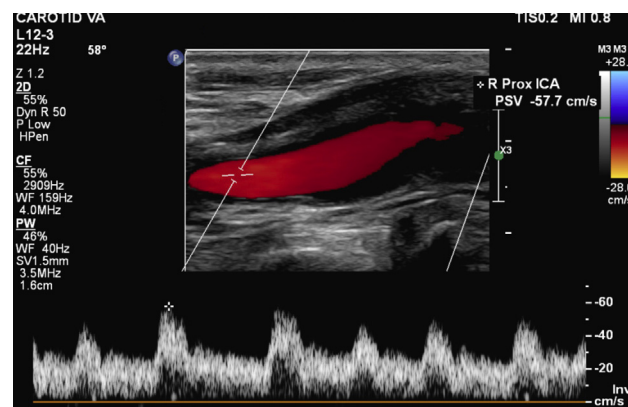


Fig 5. Follow-up Doppler spectrum with color flow image of right proximal internal carotid artery at 15 months after surgery showing a widely patent system without recurrent stenosis.

described partial resection of the hyoid in these situations^{5,11}; however, this patient remains without hemodynamically significant stenosis at 15 months of follow-up (Fig 5). Whereas bone resection remains a viable option, anatomic variants may justify alternative, less invasive approaches such as the one presented, particularly in a patient with other atherosclerotic risk factors contributing to carotid stenosis and for whom bone impingement may have been an exacerbating factor rather than the sole cause. Thus, hyoid bone resection may not always be necessary, although longer term follow-up with surveillance imaging would be helpful to explore this rare phenomenon.

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