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Saccular Aneurysm of the Proximal Left Common Carotid Artery

James E. Knake,¹ Trygve O. Gabrielsen,¹ and Robert M. Zwolak²

Aneurysms of any part of the extracranial carotid artery are uncommon lesions [1–6], and those involving just the common carotid segment may appropriately be considered rare. Although fusiform enlargement of the mediastinal segment of the left common carotid artery can occur in atherosclerotic disease and in association with syphilitic and other forms of aortitis, a saccular form of aneurysm in this location has been recorded only in a few instances after blunt chest trauma [7–9], and all have been designated as pseudoaneurysms. We report an apparently unique case of saccular aneurysm of the left common carotid artery root in a patient without history of trauma or sepsis.

Case Report

A 60-year-old man experienced occasional "dizziness" and worsening memory. Arch aortography and cerebral angiography at another hospital revealed a 1 × 2 cm saccular aneurysm of the most proximal part of the left common carotid artery (fig. 1A). The patient was referred to the University of Michigan Hospitals, where he appeared to be a healthy man in no obvious distress. His blood pressure, peripheral pulses, and cardiac rhythm were normal. His medical history included hospitalization only for uncomplicated lumbar laminectomy. Specifically, there was no history of chest trauma or other significant bodily trauma, nor of sepsis or other major illness. He took no medications and had no allergies.

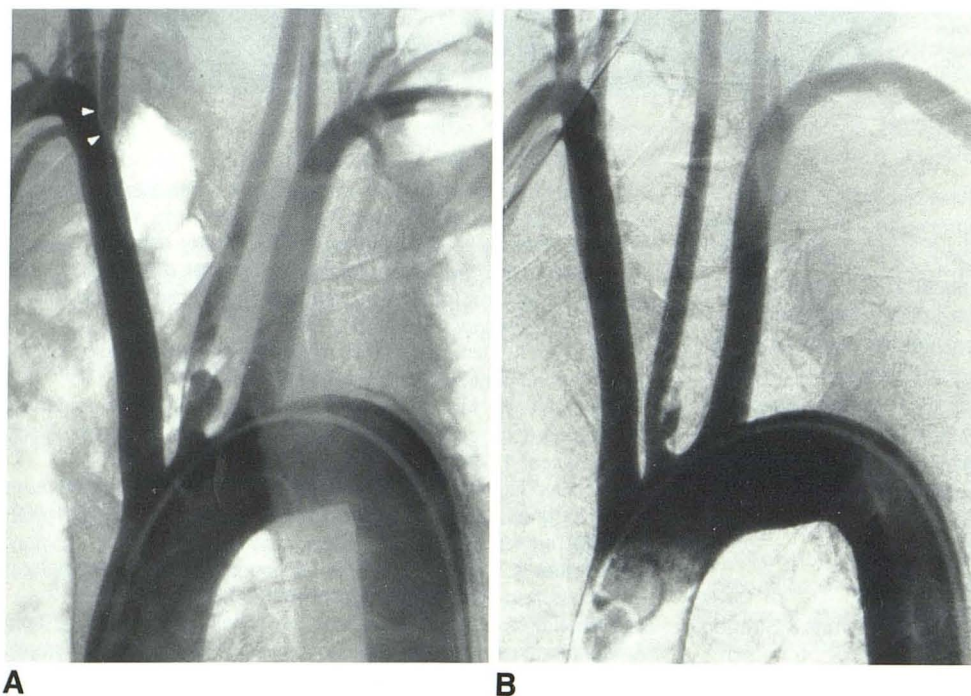


Fig. 1.—A, Initial arch aortogram in standard right posterior oblique projection. The 1 × 2 cm saccular aneurysm appears to arise directly from base of left common carotid artery, and its bulk compromises lumen of parent vessel. Only evidence of atherosclerotic disease is shallow defect in right subclavian artery just beyond right vertebral artery origin (arrowheads). B, 7 weeks later. Volume of patent lumen of aneurysm has been reduced by intraluminal thrombosis. Aneurysm clearly arises from left common carotid artery itself and not from aortic junction. On this occasion, aneurysm could not be detected on aortography in usual right posterior obliquity, and considerably steeper degree of obliquity was required.

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¹ Department of Radiology, Box 13, Division of Neuroradiology, University of Michigan Hospitals, Ann Arbor, MI 48109. Address reprint requests to J. E. Knake.

² Department of Surgery, University of Michigan Hospitals, Ann Arbor, MI 48109.

Repeat arch aortography after 7 weeks demonstrated a decrease in size of the patent lumen of the aneurysm, indicating that some intraluminal thrombosis had occurred (fig. 1B). No thrombus visibly protruded into the parent vessel. Five days later, surgery was done to interpose a Dacron graft between the supraclavicular segment of the left common carotid artery and the left subclavian artery, and the stump of the proximal left common carotid artery was oversewn. The patient was discharged on the third postoperative day.

Discussion

Although about 950 aneurysms of the extracranial carotid artery have been reported in the medical literature since 1687, groups of 20 or more cases have been presented only four times [3–6]. The incidence of these lesions is, therefore, inaccurately determined, but estimates have been that they represent 0.4%–4.0% of all peripheral arterial aneurysms [3, 6]. In 1949, Kirby et al. [10] reported that at least 90% of all common carotid aneurysms were syphilitic in origin. Atherosclerosis is now the most commonly quoted etiology, and most such aneurysms are fusiform and involve the common carotid artery bifurcation region [1, 11]. Saccular carotid aneurysms most often involve the midsegment of the cervical internal carotid artery, and trauma has been reported to be the most common cause of such aneurysms [12]. The few saccular aneurysms reported to arise from the common carotid artery have been classified as secondary to atherosclerosis [13], infection [14], tumor erosion [13], trauma including angiographic needle puncture [15, 16], or an unspecified etiology [11, 14]; none of these involved the mediastinal segment.

Nonfusiform aneurysm involving the mediastinal segment of the common carotid artery has been recorded only in the form of dissection, with pseudoaneurysm formation in young individuals who had just sustained major blunt chest trauma [7–9]. Thus, the aneurysm described and illustrated in our case report appears to be unique. Of interest, the aneurysm showed internal thrombotic change in the 7 week interval between angiographic examinations, indicating an actively evolving process rather than a long-standing indolent lesion. That peculiarity only adds to the difficulty of classifying this aneurysm accurately. In the absence of any elicitable history of antecedent cause, the best recourse is to attribute it, like most cerebral aneurysms, to a developmental defect in the vascular tunic.

The relation of the aneurysm to this patient's symptoms of memory impairment and spells of "dizziness" is certainly conjectural. However, the propensity for carotid aneurysms of all sorts to act as sources of thromboemboli has been emphasized [1, 17], and this alone justifies surgery to exclude possible embolization to the cerebral circulation.

The possibility of discovering such a lesion exists only when arch aortography is included in the angiographic evaluation of cerebral ischemia. Although the likelihood of spontaneous hemorrhage from such an aneurysm is indeterminable, the

aneurysm could quite possibly have been entered and even perforated if selective left carotid catheterization had been attempted without prior arch aortography, a practice advocated by some authors [18].

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