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*AJNR Am J Neuroradiol* 1988, 9 (3) 604-606

http://www.ajnr.org/content/9/3/604.citation

This information is current as of August 23, 2024.
Right Subclavian Steal Associated with Aberrant Right Subclavian Artery

David M. Rowe,1,2 Gary J. Becker, John A. Scott, Dewey J. Conces, Jr.

The subclavian steal syndrome is a well-recognized clinical and radiographic entity that involves the left subclavian artery far more commonly than the right. Atherosclerotic stenoses are the most common underlying cause [1-3]. Right subclavian stenosis with associated steal is markedly less common.

Although an aberrant right subclavian artery is one of the most common anomalies of the great vessels [4, 5], its association with and contribution to the right subclavian steal syndrome in adults has not been elucidated. In this report, we describe two cases of aberrant right subclavian artery associated with right subclavian steal syndrome.

Case Reports

Case 1

A 53-year-old woman presented with signs and symptoms of diffuse atherosclerotic peripheral vascular disease and occasional chest pain. She had noted increasingly severe right lower extremity claudication. In addition, she complained of recurrent episodes of both vertigo and ataxia. The right radial and brachial pulses were diminished, and her blood pressure was 90/60 mm Hg in the right arm and 130/80 mm Hg in the left. The patient denied arm claudication and paresthesias.

Transaxillary coronary arteriography revealed minimal coronary occlusive disease. Intraarterial DSA of the aortic arch and brachiocephalic vessels disclosed smooth, mild (<50%) atherosclerotic carotid bifurcation stenoses bilaterally, a dominant left vertebral artery, and an aberrant right subclavian artery with its origin distal to that of the left subclavian. Also noted was a severe stenosis of the proximal right subclavian artery with distal filling via retrograde flow in the right vertebral artery (Figs. 1A and 1B).

A right common carotid-right subclavian bypass graft was constructed. The patient tolerated surgery well and remains asymptomatic in the initial postoperative period.

Case 2

A 54-year-old obese woman had endured right arm claudication for 1 year. At the time of admission, she described severe pain and weakness with minimal exertion. The patient's medical history included previous diagnoses of non-Hodgkin lymphoma and insulin-dependent diabetes mellitus. The right radial pulse was markedly diminished. Blood pressure was 78/7 mm Hg in the right arm and 150/80 mm Hg in the left. A transfemoral intraarterial DSA of the aortic arch and brachiocephalic vessels revealed an aberrant right subclavian artery with its origin distal to that of the left subclavian artery. The right subclavian was occluded proximally and reconstituted distally via retrograde flow in the right vertebral artery (Figs. 2A and 2B). The left vertebral artery was dominant. There was also a moderate stenosis at the origin of the right common carotid artery and moderate irregularity of the left subclavian.

Owing to the previous history of lymphoma, a contrast-enhanced CT of the mediastinum (Fig. 2C) was obtained. This nicely demonstrated the aberrant course of the right subclavian and the proximal thrombus. There was no evidence of an extrinsic compressive mass. The patient remains undecided regarding surgical intervention at this time.

Discussion

Modern awareness of the subclavian steal syndrome was facilitated by the works of Contorni [6] and Reivich et al. [7] in the early 1960s. Since that time a well-defined constellation of clinical signs and symptoms has evolved in adults presenting with this syndrome. The presenting complaints usually include either neurologic symptoms or ischemic arm changes. Neurologic symptoms are variable and include vertebrobasilar problems, such as vertigo, paresthesias, and limb paresis. Less common presenting symptoms include ataxia, visual problems, and syncope [1-3]. Exercise-induced symptoms are thought to be more specific but less common [1]. Arm ischemia produces claudication, fatigability, diminished peripheral pulses, and a systolic blood pressure differential of 20 mm Hg or more between the upper extremities [1-3]. Concomitant disease in the other brachiocephalic vessels is the rule [1].

Among all the large series of subclavian steal, a predominance of left-sided lesions is well demonstrated. In the large series of Fields and Lemak [1], brachiocephalic trunk or right
subclavian lesions accounted for only 30%, or 51 of 168 cases, including patients with bilateral disease. A review of 22 cases by Larrieu et al. [2] disclosed only one patient with an isolated right obstruction and steal. At our institution, a review of 50 cases revealed only seven with right-sided steal (P. Bendick, personal communication, May 1986). Although atherosclerotic occlusive disease is by far the most commonly described cause, other documented causes include arteritis, trauma, tumor thrombus/encasement, previous surgical procedure such as Blalock-Taussig shunt, and congenital aortic interruption [3, 8, 9]. Isolated left subclavian artery and right aortic arch with associated congenital steal has been described [10, 11]. However, there has been only one report of a congenital right subclavian steal in a patient with a normal left aortic arch and isolated right subclavian artery arising from a small aortic diverticulum [12]. None of the angiographically demonstrated cases in adults has been associated with an aberrant right subclavian artery.

The aberrant right subclavian artery is considered the most common variant of innominate-right subclavian development [4, 5]. Reported frequencies range from 0.4–2.0% [4]. Most commonly the aberrant vessel originates from the posterior wall of the aortic arch distal to the origin of the left subclavian artery [4, 5]. This is well demonstrated on the CT scan in case 2 (Fig. 2C). The aberrant vessel usually crosses posterior to the esophagus, although it may course between the esophagus and trachea or anterior to the trachea [4]. In our case 2, the occluded portion of the right subclavian lies posterior to the esophagus.

Although Felson et al. [4] alluded to a probable case of subclavian compromise with diminished pulses in their classic review article of aberrant right subclavian artery in 1950, there
was no angiographic evaluation. Most discussions of the aberrant right subclavian artery have been limited to either anatomic description or to its controversial role in the production of dysphagia symptoms (dysphagia lusoria). Obvious surgical implications exist, particularly in those patients with other congenital cardiac or vascular lesions. However, the association of this common anomaly with the clinical symptoms and radiographic demonstration of subclavian steal, manifested by delayed filling of the distal right subclavian via the right vertebral artery, has not been well documented.

Isolated involvement of occlusive disease severe enough to produce subclavian steal is unusual for reasons that are not entirely clear. One potentially important factor is the relatively larger diameter of the brachiocephalic trunk as compared with that of the left subclavian. Presumably, atherosclerotic plaques of similar size would be more likely to produce earlier symptomatic narrowing in the smaller left subclavian than in the brachiocephalic trunk. Because of their similar sizes, aberrant right subclavian arteries should be at the same risk as left subclavian arteries for the development of early symptomatic occlusive disease.

In summary, isolated right subclavian steal syndrome is unusual. Demonstration of an aberrant right subclavian artery as an associated condition or contributing factor may be difficult if the proximal artery is totally occluded. However, contrast-enhanced CT may demonstrate the aberrant course of the occluded right subclavian. In our two cases the aberrant right subclavian arteries were easily demonstrated on DSA, and late frames showed the steal. An aberrant right subclavian artery should be considered a contributing factor in patients presenting with isolated right subclavian steal syndrome.

REFERENCES

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