Accessory renal arteries involved in atherosclerotic occlusive disease at the aortic bifurcation

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ABSTRACT

Accessory renal arteries (ARAs) are embryonic remnants found in more than one-third of patients and occurring bilaterally in 10% of the population. Very few reports have documented such vessels arising near or at the level of the aortic bifurcation. Furthermore, the presence of ARAs has yet to be described in the context of atherosclerotic disease. Here, we present a unique case of large bilateral ARAs originating above the aortic bifurcation concurrent with symptomatic aortoiliac atherosclerotic disease. We highlight the embryologic and clinical significance of these vessels as well as discuss their potential role in accelerating atherosclerotic disease processes. (J Vasc Surg Cases and Innovative Techniques 2020;6:425-9.)

Keywords: Accessory renal arteries; Atherosclerosis; Aortic bifurcation

An accessory renal artery (ARA) is a vestigial structure that forms during the ascent of the kidney from the pelvis to the lumbar region.[1] In the pelvis, the primitive kidney is supplied by vessels branching off the common iliac arteries. As the kidneys ascend, they are supplied by successively more superior vessels branching off the aorta until the main renal artery develops at the second lumbar vertebral body. Concurrently, inferior primordial branches involute and disappear. Failure of these inferior embryonic vessels to degenerate results in ARA.[1,2]

The reported prevalence of ARAs ranges from 24% to 42%.[3] Bilateral ARAs occur in approximately 10% percent of the population.[4] Compared with main renal arteries that measure 4 and 5 mm in diameter, ARAs are generally <2 mm.[5,6] With respect to surgical interventions involving the abdominal aorta, damage to the ARAs may carry an increased risk of postoperative renovascular complications, including renal infarction and decline in function.[7,8]

Although previous studies attempted to address the clinical significance of ARAs in the context of hypertension, perioperative bleeding, and impaired renal function,[3,7,9] there is currently scant literature describing this aberrant anatomy in the setting of atherosclerotic disease. Because ARAs rarely arise close to the aortic bifurcation,[6,7,10] it is unclear whether the presence of ARAs would hasten the rate of plaque formation in this region by means of further disrupting blood flow dynamics. In this case, we describe a fairly young patient presenting with severe, symptomatic aortoiliac stenosis, in whom operative repair was influenced by the presence of large, bilateral ARAs above the aortic bifurcation. The patient agreed to publication of the case details and images.

CASE REPORT

A 47-year-old man presented with complaints of severe, disabling bilateral claudication in the lower extremity, buttocks, and thighs at less than half a block. The patient was an active smoker with a body mass index of 23 kg/m^2. Past medical history was significant for hypertension, hyperlipidemia, and diabetes mellitus. There was no history of previous cardiac or cerebrovascular ischemic events. On physical examination, the patient had very weak femoral and nonpalpable pedal pulses.

Computed tomography angiography of the abdomen and pelvis revealed atherosclerotic occlusive disease of the aorta extending into the proximal common iliac arteries bilaterally (Fig 1, *a-c*; Supplementary Video). There were also bilateral ARAs >4 mm in diameter that arose 8 mm above the aortic bifurcation (Figs 1, *d* and *e*, and 2).

Given the patient's age and restrictive anatomic criteria, open surgical revascularization was recommended. Through a transperitoneal incision, the distal abdominal aorta and proximal common iliac vessels were resected, and a Hemashield bifurcated graft (Getinge AB, Rastatt, Germany) was sewn in place. To avoid potential compromise of kidney function, given the large size of the ARAs that were observed entering the kidneys at the inferior pole, operative repair was directed to preserve the ARAs by excising a large aortic patch encompassing the origins of these vessels. The ARA island patch was then reimplanted onto the main body of the Hemashield graft (Fig 3). At

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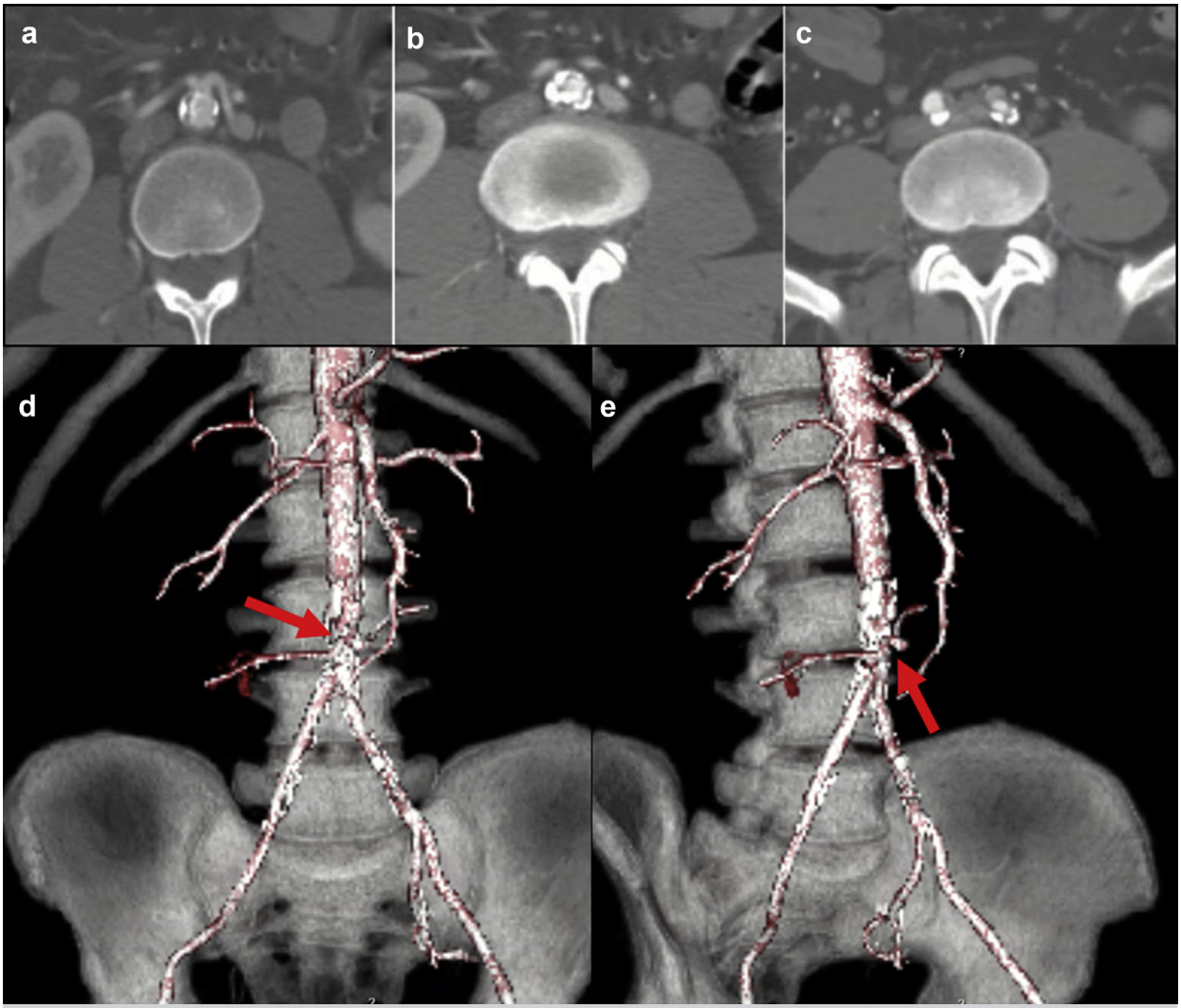

Fig 1. a-c, Preoperative computed tomography scan demonstrating calcifications beginning proximally at **(a)** accessory renal artery (ARA) origin and extending distally into **(b)** aorta and **(c)** aortic bifurcations. **d** and **e,** Three-dimensional reconstructed computed tomography images; the *arrows* indicate ARA origin in **(d)** anteroposterior and **(e)** anterolateral views.

the conclusion of the procedure, the patient had palpable femoral and pedal pulses before leaving the operating room.

The patient needed to be taken back to the operating room 6 hours after the procedure for thrombectomy after development of bilateral lower extremity numbness and diminished pulses. The cause of the graft limb thrombosis was believed to be embolic shower from the highly dense, calcified aortic plaque at the proximal anastomosis of the graft. The postoperative course was otherwise uncomplicated, and kidney function was clinically and objectively undisturbed (Figs 4 and 5).

DISCUSSION

Because of the complex nature of renal embryogenesis, anomalous variations in renal vasculature are common. ARAs are additional arteries that may either join the main artery at the hilum or penetrate the parenchyma separately. ARAs with "extrahilar" penetrations (as observed intraoperatively in our patient at the inferior pole) typically serve as functional end-arteries, which are tissue-sustaining vessels devoid of anastomoses. Therefore, damage or ligation of these ARAs functioning as end-arteries will inherently lead to infarction of the renal parenchyma that it supplies.[1,2,11] Although numerous observational studies offer varying and sometimes conflicting recommendations, no comprehensive or concrete guidelines specifying when it is appropriate to preserve these vessels have been developed. For example, Majos et al[10] reported successfully occluding extrarenal arteries up to 3 mm in diameter without compromising renal function in patients with normal and horseshoe kidneys. A study by Devirgilio et al[12] that focused on patients with chronic kidney disease advocated for ARA reimplantation of all sizes. Additional studies in patients who underwent endovascular

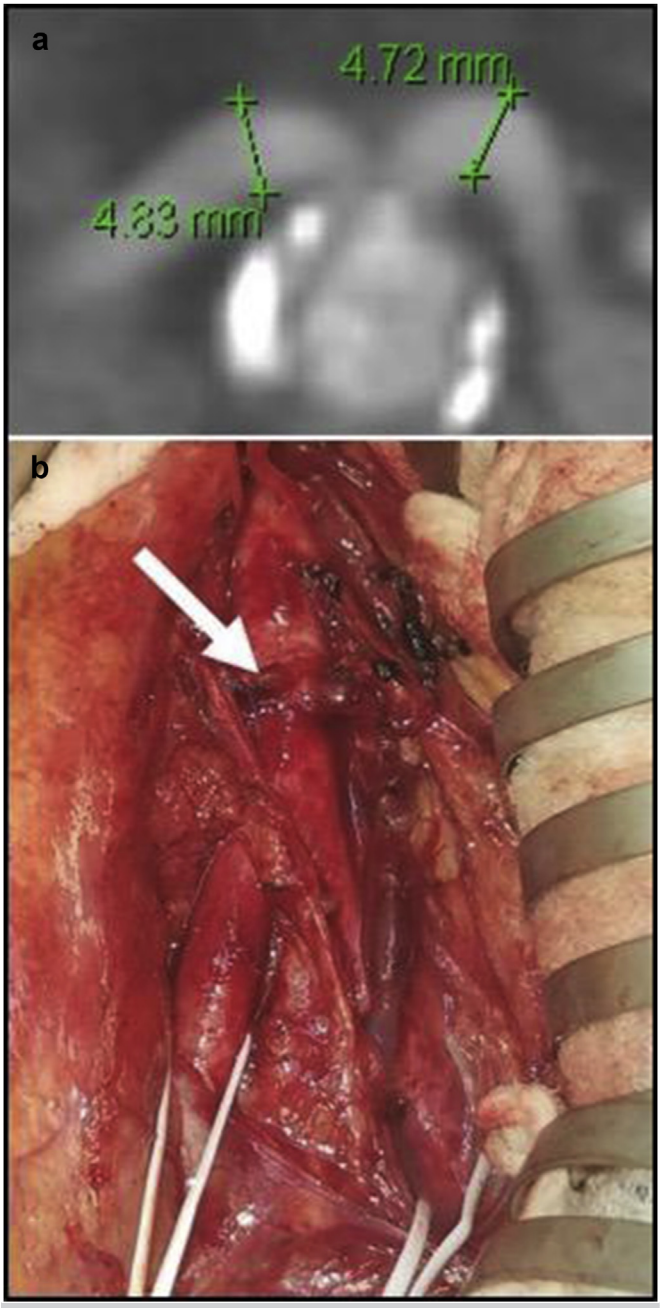

Fig 2. a, Accessory renal artery (ARA) diameter measured by computed tomography. **b,** Exposure; the *arrow* indicates ARA origin.

aneurysm repair with coverage of the ARA reported renal infarction in 67% to 84% of patients.[7,8] Ultimately, the decision of whether to sacrifice or to reimplant ARAs rests on the surgeon's discretion and clinical judgment, which is plagued by a combination of patient-specific factors (including vessel size, parenchyma involvement, and renal function). The decision to preserve the ARAs for this patient was mainly based on the considerable size of the vessels (>4 mm) and their extrahilar penetration, albeit good renal function.

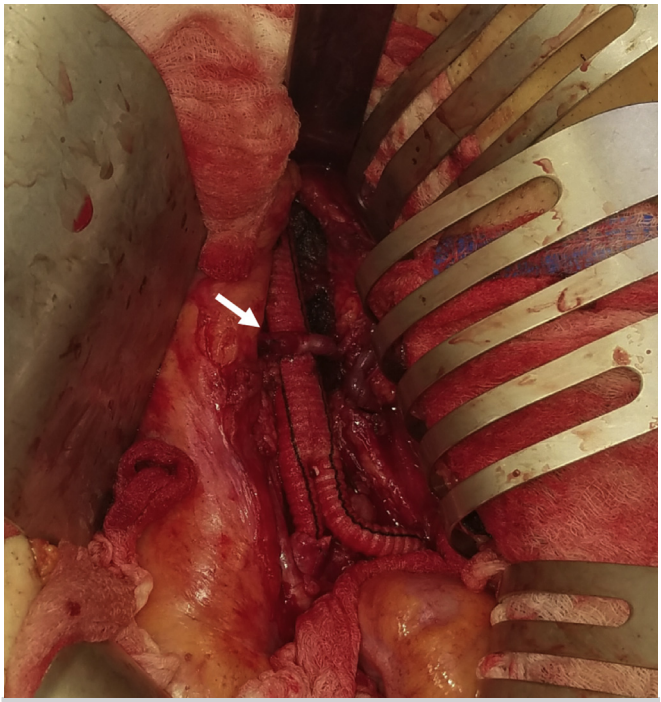

Fig 3. Aortic patch encompassing accessory renal artery (ARA) origins (indicated by *arrow*) reimplanted onto Hemashield graft.

Literature discussing key morphologic features in renal vasculature anomalies has cited rare cases of ARAs originating near or below the aortic bifurcation.[6,7,10,13] The majority of these cases have been described in the setting of other renal anomalies, such as horseshoe kidney and ectopic kidneys.[10,13] Çilingiroğlu et al[14] reported a case in which a 46-year-old man presenting with an atherosclerotic plaque occluding the left common iliac artery was found to also have a left ectopic kidney with a single renal artery arising from the aortic bifurcation. It is possible that subsidiary renal vessels predispose adjacent regions to atherosclerosis by means of altering blood flow dynamics.

Hemodynamic changes have long been characterized in areas of vessel curvature, branching, and bifurcations.[15] Because the endothelium is in direct contact with flowing blood, it is constantly exposed to the biomechanical forces that blood exerts on the vessel wall.[16] Straight, unbranched vessels exhibit undisturbed, uniform, and unidirectional flow that is dominated by laminar forces. Bends, branches, and bifurcations introduce turbulent and oscillatory forces that decrease blood flow velocity. Altering flow dynamics as such lowers shear stress, which is the frictional "drag" force exerted by blood on the endothelial or intimal lining.[17] The mechanical cues generated by low shear stress trigger vasomotor and inflammatory responses that eventually impair endothelial integrity. Disruptions in the intimal lining ultimately

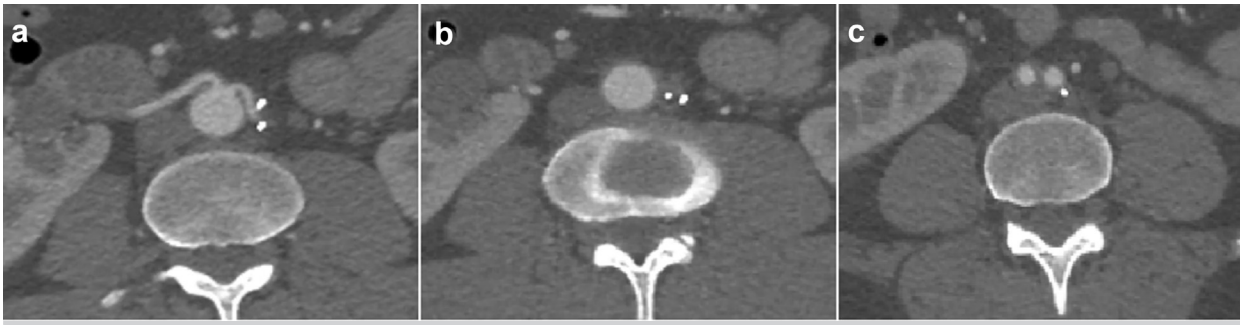

Fig 4. Postoperative computed tomography scan beginning proximally at **(a)** accessory renal artery (ARA) origin and going distally into **(b)** aorta as well as **(c)** aortic bifurcations. Images were captured at the same level as preoperative imaging shown in Fig 1.

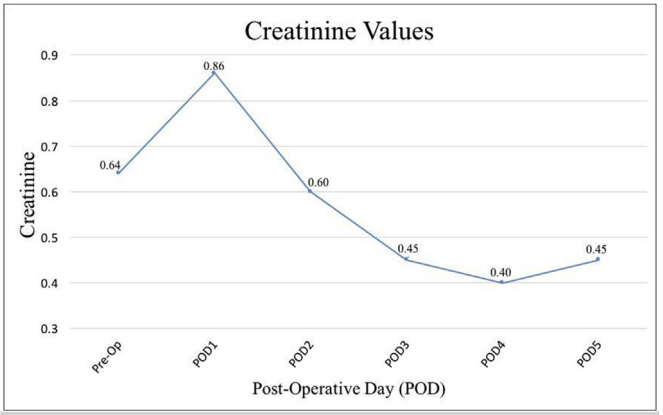

Fig 5. Preoperative and postoperative serum creatinine values.

become loci susceptible to further derangements, such as atheroma formation.[16,18]

Regions of extensive tortuosity and branching in external, internal, and common carotid arteries have demonstrated diminished flow velocity and shear stress.[19,20] Histologic analysis at these regions revealed development of atherosclerosis, with larger measurements in plaque thickness directly linked to the degree of reduction in shear stress.[21] Similarly, this hemodynamic phenomenon of proatherogenic shear stress has been described at the aortic bifurcation[22] but not in the presence of supernumerary vessels. Whereas this may be speculative, we believe the extent of debilitating atherosclerotic disease at such a young age, as seen in our patient and in the case of Çilingiroğlu et al,[14] may in part be explained by the disturbed blood flow rendered by the presence of anomalous renal vessels. Therefore, we believe that identifying this variant anatomy in conjunction with atherosclerotic disease may be imperative moving forward.

CONCLUSIONS

In our case, the presence of ARA not only posed a challenge to our operative repair but also raised the question of whether these vessels increase risk of accelerated atherosclerotic plaque formation. Our patient presented with atherosclerosis of the aorta and iliac arteries at a young age. Although this is not unheard of and our patient presented with other atherosclerotic risk factors, his variant anatomy may have played a role in the acceleration of the atherosclerotic process. Further studies are warranted in an effort to explore this relationship as it may potentially identify critical values at which ARA diameter would constitute increased risk for atherogenesis. Future studies in patients with unique ARA morphologic features (such as vessel size and origin as seen in our patient) would also benefit from arterial duplex ultrasound measuring flow patterns and velocities, specifically at the aortic bifurcation and ARA origin. Last, additional studies among young patients with otherwise healthy renal function, such as our patient, may offer valuable contributions to the eventual development of comprehensive guidelines providing an optimal approach for patients with ARAs.

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