Renal artery aneurysms

Dawn M. Coleman, MD, and **James C. Stanley, MD,** *Ann Arbor, Mich*

Renal artery aneurysms are rare in the general population, although the true incidence and natural history remain elusive. Controversy over criteria for repair persists across decades. Indications for repair presently include aneurysm size >2 cm, female gender within childbearing age, symptoms like pain and hematuria, medically refractory hypertension including that associated with functionally important renal artery stenosis, thromboembolism, dissection, and rupture. Conventional surgical reconstruction options are variable and continue to offer technically sound and durable results. Endovascular therapies with novel devices also offer technical success with few major adverse events, and are increasingly employed as indications for intervention broaden. This review summarizes the accumulated evidence on true renal artery aneurysms with a particular focus on contemporary treatment criteria, natural history, options for repair and outcomes following such. (J Vasc Surg 2015;62:779-85.)

Renal artery aneurysms (RAAs) are rare. Controversy over criteria for repair persists across decades. In addition, conventional treatment strategies have come into question with evolving endovascular technologies. A review of the existing information surrounding RAAs is appropriate in contemporary practice.

INCIDENCE AND NATURAL HISTORY

The widely accepted incidence of RAA is 0.1% in the general population, although the true incidence and natural history remain elusive. These aneurysms are infrequently discovered during gross autopsy, as modest attention is often paid to the distal vessels (Table I). Angiographic and computed tomography studies report an incidence from 0.3% to 2.5%, acknowledging that diagnostic imaging incurs some degree of bias in pursuing such studies and may overestimate RAA prevalence.[2-6]

The natural history of RAAs is that of slow to null growth (Table I). While historic series describe rupture rates as high as 14% to 30% with associated mortality of 80%, this is not supported by contemporary data.[2] Most ruptures are diagnosed at the time of presentation, and several authors have supported no rupture during the surveillance of nonoperative RAAs.[1,2,4,5,7-11] Contemporary series estimate a median annualized growth rate of 0.06 to 0.6 mm.[9-11] The most recent and largest multi-institutional series of nonoperative RAA surveillance found no difference in growth rate based on aneurysm morphology or calcification.[10] These same authors also report the successful surveillance of 88 aneurysms measuring 2 to 3 cm and seven aneurysms measuring >3 cm without complication or rupture during a mean of 49 months. Contemporary rupture rates are estimated at 3% to 5% with nongestational mortality <10%.[2,5,12-15]

From the Section of Vascular Surgery, University of Michigan Cardiovascular Center.

Author conflict of interest: none.

Correspondence: Dawn M. Coleman, MD, University of Michigan Cardiovascular Center, 1500 E Medical Center Dr, Ann Arbor, MI 48109-5867 (e-mail: dawnbarn@umich.edu).

The editors and reviewers of this article have no relevant financial relationships to disclose per the JVS policy that requires reviewers to decline review of any manuscript for which they may have a conflict of interest.

0741-5214

http://dx.doi.org/10.1016/j.jvs.2015.05.034

CLINICAL PRESENTATION AND DIAGNOSIS

RAAs typically present in the sixth decade. Some authors suggest that males present up to a decade later in life than females.[2,13] Women are more commonly afflicted with RAA, likely due to the high incidence of associated fibromuscular dysplasia. A minority of patients will present with symptoms, and clinical exam may reveal hypertension (HTN). Renal bruit or a palpable abdominal mass are inconsistent and unreliable physical findings. The majority of patients lack traditional cardiovascular risk factors other than HTN. Less than one-third of patients smoke.

Typical RAA anatomy is outlined in Table I. Single unilateral aneurysms appear to favor the right side, with the average reported RAA size being 1.3 to 3.8 cm, and operative series reporting larger diameters.[2,7-10,12,15-18]

Computed tomography is the most common contemporary diagnostic modality, followed by magnetic resonance imaging, ultrasonography, and catheter-based arteriography.[10] A RAA may appear as a signet-ring calcification on roentgenogram. Conventional preoperative arteriography is warranted, given the frequency of multiple aneurysms affecting distal branches that may be missed on conventional cross-sectional imaging.

INDICATIONS FOR INTERVENTION

Currently accepted indications for RAA intervention include size >2 cm, female gender within childbearing age, symptoms like pain, hematuria, medically refractory HTN including that associated with functionally important renal artery stenosis, thromboembolism, dissection, and rupture.

Women of child-bearing age. Pregnancy is thought to be associated with a higher rate of rupture secondary to increased vascular flow and hormonal changes, resulting in weakening of the vessel wall elastic tissue. Although no large scale studies detail the true incidence, in a series of 180,000 pregnancies brought to term, no RAA ruptures

Table I. Features of renal artery aneurysms (RAAs)

Incidence	• Autopsy rates, <0.01%-0.09%[1] • Arteriogram rates, 0.3%-2.5% (up to 9.7%)[2-5] • CT rates, 0.7%[6]
Natural history	• Large autopsy series demonstrate no rupture[3,4] • Most report no rupture during surveillance out to 270 months[1,2,4,5,7-11] • Growth rate 0.06-0.6 mm/y[9-11] • Rupture rate 3%-5% with nongestational mortality <10%[2,5,12-15]
Clinical presentation and risk factors	• Sixth decade of life (range, 46-62 years of life)[2,4,7,9-11,13,15,16] • Female predominance up to 72%[1,7,9-13,15,17] • Association with FMD up to 68%[1,7,9-13,15,17] • Symptoms rare (4%-23%): abdominal and/or flank pain, hematuria • Clinical exam may identify: HTN, renal bruit, and palpable abdominal mass • Majority of patients are hypertensive • Chronic renal insufficiency has been identified in 4-14% of patients[1,12,15,16] • Alternate arterial aneurysms (ie, aortic, iliac, visceral) in 7%-30% of cases
Anatomy and radiographic features	• Most saccular • Two-thirds affect arterial bifurcations • Often multiple, 10%-20% bilateral, non-renal arterial aneurysms (7%-30%)[1,2,7,9,11,12,15-17] • 18%-68% calcified • 8%-11% demonstrate thromboembolism

CT, Computed tomography; *FMD,* fibromuscular dysplasia; *HTN,* hypertension.

Table II. Summary of evidence on hypertensive outcomes following renal artery aneurysm (RAA) reconstruction

Series	*No.*[a]	*Mean follow-up, months*	*Clinical improvement or cure in HTN*
Martin[1]	14	4.8-7.8 years	76%
Hupp[17]	17 (8 patients described with 'refractory' HTN)	NR	100%
Henke[7]	40	94	60% taking fewer meds
Pfeiffer[13]	75	46	47%
English[16]	55	47	75%
Chandra[8]	7	11	Collective decrease in antihypertensive meds following surgery (1.6 vs 2.7; $P = .03$)
Robinson[12]	24	99	100% 82% with severe HTN taking fewer meds (1.1 vs 2.6; $P = .0128$)
Klausner[9]	14	36	28%
Klausner[10]	76 described with 'difficult to control HTN'	NR	32% (no significant change in number of meds postoperatively)

HTN, Hypertension; *NR,* not recorded; *meds,* medications.
[a]Number of patients with HTN undergoing reconstruction.

were identified.[3,19] Ruptures typically occur in the third trimester with only a few case reports of rupture post-delivery.[19] Rupture during pregnancy has been described in aneurysms as small as 1 cm.[1] Historic reports imply dismal consequences (56%-92% maternal mortality and 82%-100% fetal mortality).[20] Contemporary outcomes for both mother and fetus may be improving, as there are anecdotal reports of gestational rupture resulting in both maternal and fetal survival.

HTN. Approximately 70% of patients with RAA have HTN, with up to 100% affected in some series.[1,2,7,9,11-13,16,17] Hypotheses for the mechanism of HTN include (1) coexistent renal artery occlusive disease, (2) distal parenchymal embolization, (3) compression or kinking of associated renal artery branches, and (4) hemodynamic changes from turbulent blood flow within the aneurysm resulting in decreased distal renal artery perfusion.[2,16]

Most series suggest improvement or cure in the majority of hypertensive patients undergoing RAA reconstruction (Table II). Martin et al evaluated patients for renovascular HTN preoperatively and demonstrated that 100% of those operated upon with documented renovascular HTN improved or were cured of HTN, while only 60% of those without arterial stenosis were cured or improved.[1] Similarly, Pfeiffer et al noted a differential improvement in HTN following aneurysm repair in those