

Renal artery aneurysms

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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}

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

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Table I. Features of renal artery aneurysms (RAAs)

Incidence	<ul style="list-style-type: none"> • Autopsy rates, <0.01%-0.09%¹ • Arteriogram rates, 0.3%-2.5% (up to 9.7%)²⁻⁵ • CT rates, 0.7%⁹
Natural history	<ul style="list-style-type: none"> • 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	<ul style="list-style-type: none"> • 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	<ul style="list-style-type: none"> • 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; <i>P</i> = .03)
Robinson ¹²	24	99	100% 82% with severe HTN taking fewer meds (1.1 vs 2.6; <i>P</i> = .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.

^aNumber 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

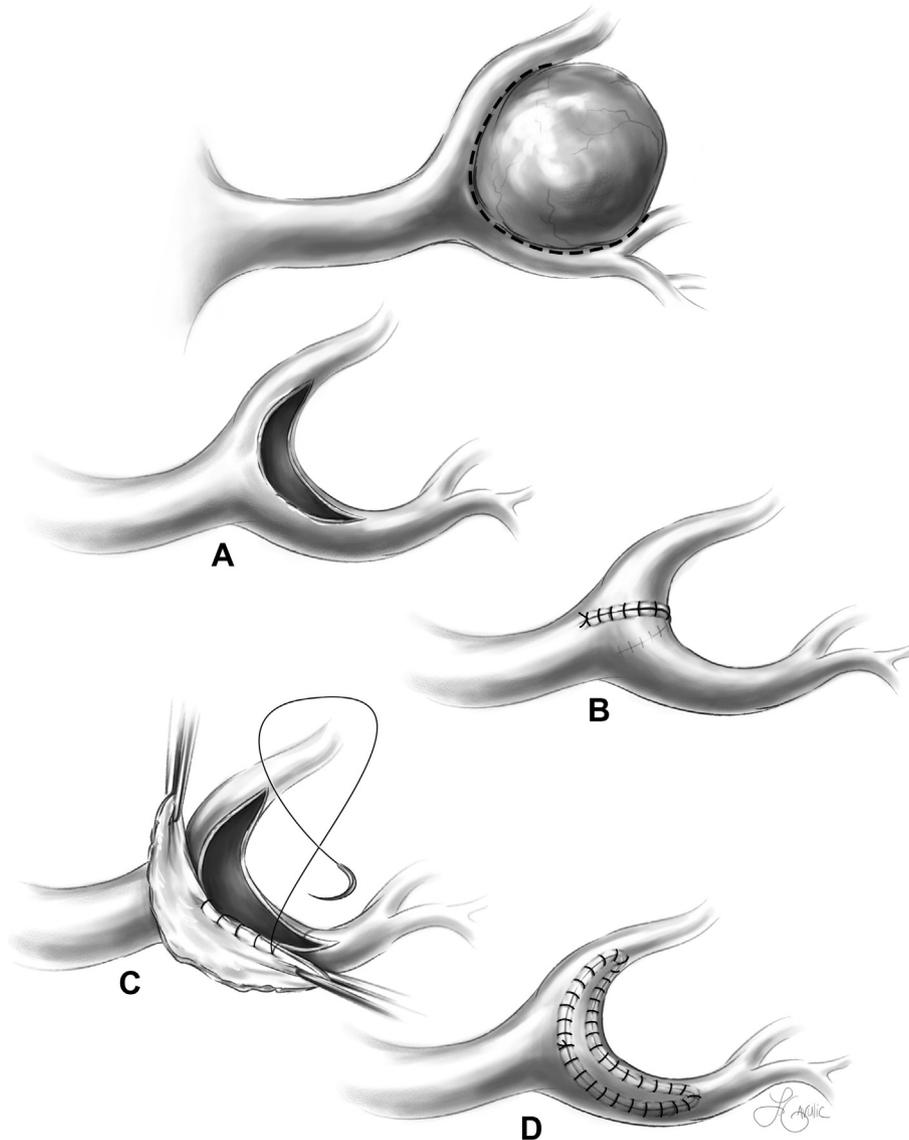


Fig 1. Following the longitudinal excision of a large saccular aneurysm located at the primary branch point (A), primary angioplasty closure of the defect in a transverse (Heineke-Mikulicz) fashion (B) or patch angioplasty (C and D) may be considered.

with documented renal artery stenosis (67%) in comparison to those without stenosis (29%).¹³

A functionally important associated renal artery stenosis in the presence of a RAA remains a valid indication for intervention. Coexistent renal artery stenosis has been identified in 7% to 66% of patients with RAA, but it does not explain every case of improvement following surgery.^{1,7,9,12,13} HTN may be related to distal parenchymal embolization, which has been described in 8% to 11% of patients with RAA and may be a consequence of dislodged mural thrombus from within the aneurysm sac.^{13,16}

Computational fluid studies that modeled renal flow for selected renal aneurysms have demonstrated that

pressure on the aneurysm wall supports the presence of forces that both deform and may obstruct the renal artery, resulting in pathologically relevant pressure losses, which can induce renin-dependent HTN.²¹ Among the modeled geometries, a saccular aneurysm located at the main renal artery branch, a common shape and location for RAA, appears to lead to the largest risk of occlusion.²¹

MANAGEMENT OPTIONS

Surgical reconstruction. Conventional in situ reconstructions (Figs 1-5) include aneurysm resection with (1) primary angioplasty closure with or without branch reimplantation, (2) patch angioplasty, (3) primary

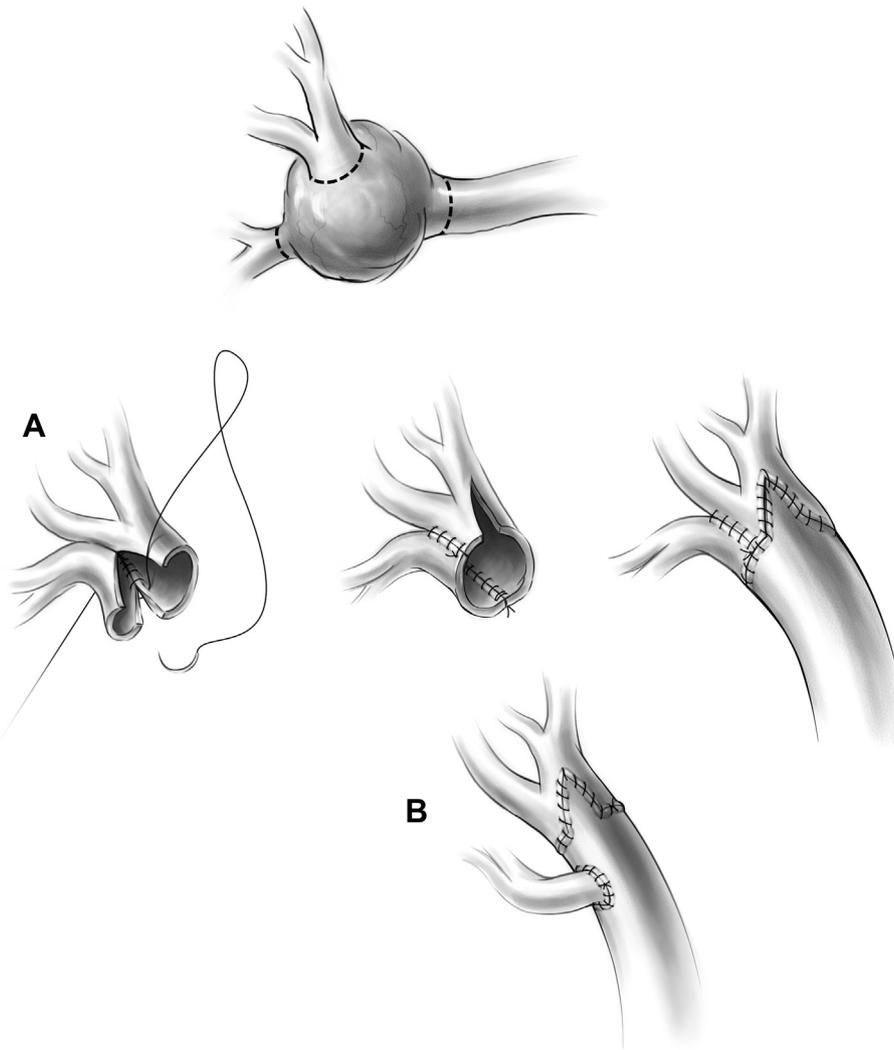


Fig 2. Aneurysms may have several outflow vessels of variable caliber. **A**, These vessels may be spatulated together to provide a common outflow target for a bypass graft; or **B**, branch vessels may require separate reimplantations onto the graft.

re-anastomosis, (4) interposition bypass, (5) aorto-renal bypass, (6) splanchno-renal bypass, and (7) plication of small aneurysms. Conventional open surgery offers low morbidity, negligible mortality, and durable patency (Table III). The young age of many patients and excellent projected long-term survival (up to 91% at 10 years) reinforces the importance of performing a technically sound procedure.^{12,16,18}

Henke et al noted no difference in long-term event-free outcome between those patients undergoing aneurysmectomy with angioplastic closure or aneurysmectomy with bypass, with mean lifespan calculated at 108 and 130 months, respectively.⁷ Pfeiffer et al demonstrated superior patency rates for angioplastic repairs in comparison to those reconstructions requiring saphenous vein interposition (100% vs 73%).¹³ Moreover, angioplastic

repair yields no recurrent aneurysmal degeneration with follow-up.^{7,13}

While complex distal branch lesions were historically treated with nephrectomy, they may best be approached with ex vivo repair and auto-transplantation. Murray et al described a 92% success rate with in situ bifurcation and ex vivo multi-branch replacement with branched and unbranched internal iliac artery autograft in 12 patients with aneurysms without mortality or major morbidity.²⁶ Gallagher et al reported seven ex vivo reconstructions for complex aneurysmal disease with excellent technical success.²⁷ Chandra et al compared in situ and ex vivo reconstructions and noted no significant difference in hospital length of stay, morbidity, mortality, or need for reoperation at follow-up.⁸ Importantly, 100% of reconstructions in the latter study were patent by imaging

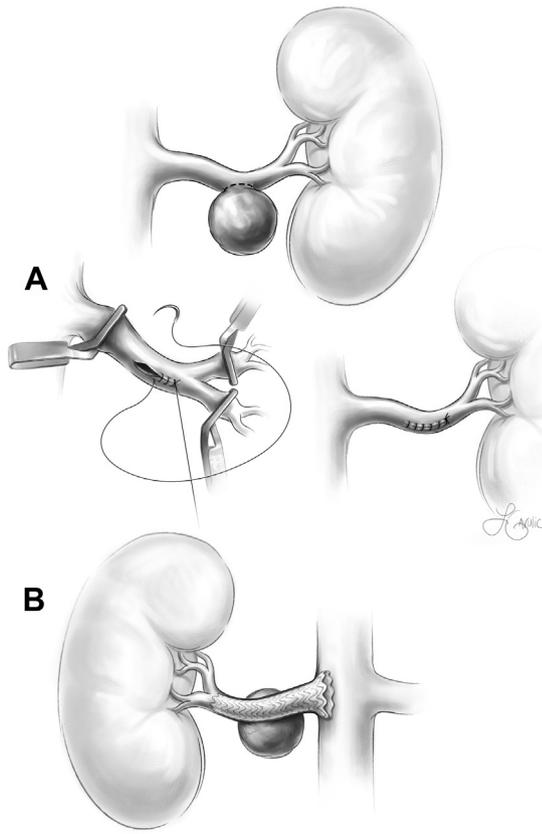


Fig 3. Large aneurysms of the main renal artery may be treated by way of excision with primary repair (A) or excluded by endovascular means using a covered stent graft (B).

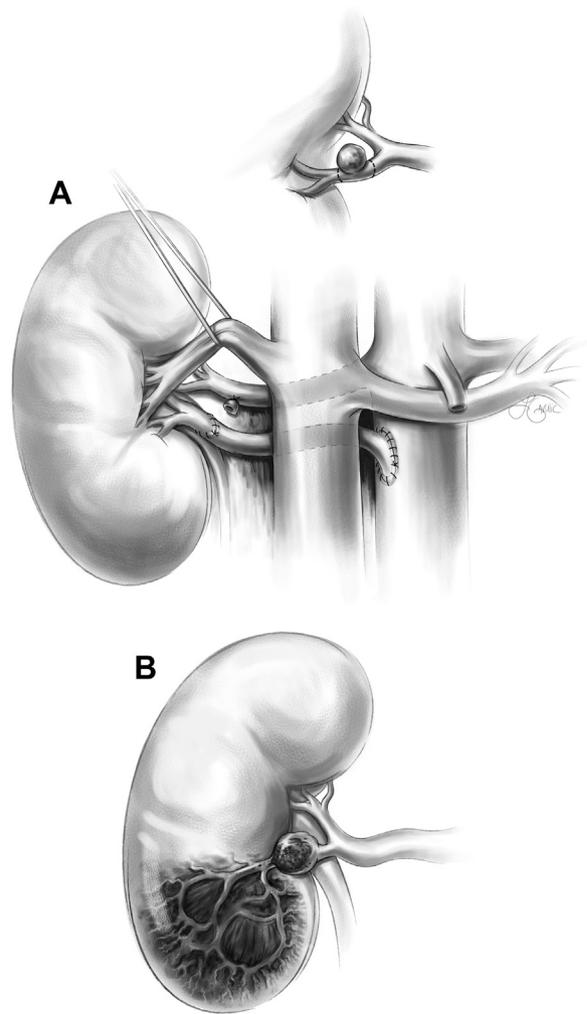


Fig 4. A, Distal aorto-renal bypass may be appropriate for segmental renal artery aneurysms (RAAs). B, Select aneurysms of this type may be appropriately treated by endovascular embolization.

obtained during the first year of follow-up. Robotic techniques have more recently been introduced that may become more prevalent in future practices.^{23,28-30}

Cooled (4°C) renal perfusion supplemented with mannitol or prostaglandin E has been advocated by several authors either routinely or when >30 to 40 minutes of warm renal ischemia is anticipated, to reduce the risk of acute tubular necrosis.^{7,13,16} Most authors advocate completion imaging before hospital discharge and long-term follow-up with surveillance imaging.^{2,7,12,13,16,26}

Endovascular interventions. Traditional endovascular therapies have utilized coil embolization for distal and parenchymal aneurysms (Fig 4, B) and stent graft exclusion (Fig 3, B) for main renal artery lesions. The indications for endovascular repair have broadened with the introduction of three-dimensional detachable coils, non-adhesive liquid embolic agents (ie, Onyx), remodeling techniques (which include balloon- and stent-assisted coiling), and flow diverter stents (ie, the CardiaStis multi-layer stent).^{22,24,25} Technical success across larger series have been reported as 73% to 100%, with highly variable rates of morbidity (13%-60%) that include primarily radiographic evidence of end-organ malperfusion from

thromboembolism and subsequent postembolization syndrome (Table III). These reported series describe no incidence of access-related complications, arterial dissections, or renal compromise, along with low rates of recanalization requiring reintervention (4%-13%).³¹⁻³³

Comparisons of open surgical and endovascular procedures have reported no significant difference in mortality, perioperative morbidity, freedom from re-intervention, decline in renal function, or length of stay.^{10,14,15}

CONCLUSIONS

The natural history of RAA is likely more benign than historic reports have suggested, with a low risk of rupture, slow to null rate of growth, and improved survival following rupture. While short-term follow-up at 1 year remains prudent for a newly diagnosed RAA, longer intervals

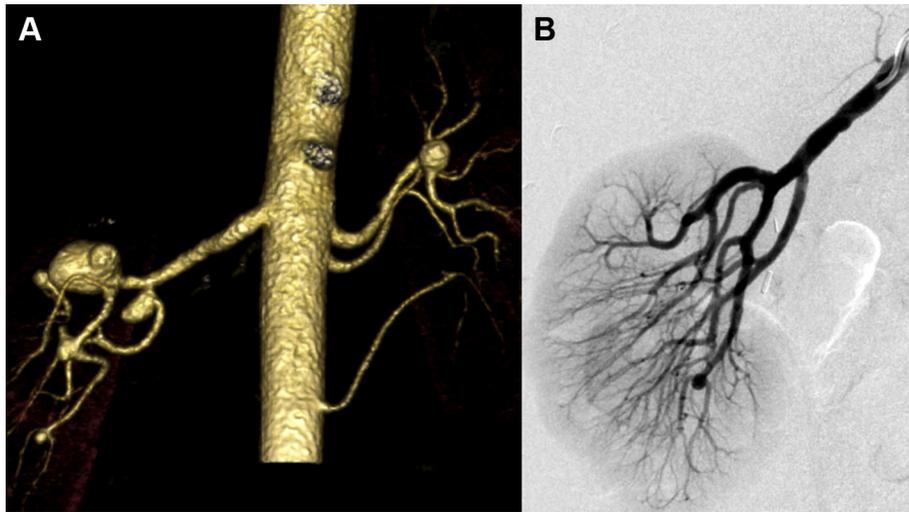


Fig 5. **A**, Computed tomography angiogram documenting multiple bilateral renal artery aneurysms (RAAs) in a young woman with fibromuscular dysplasia and hypertension (HTN); **B**, Completion arteriography following excision of two large first-order segmental right RAAs with angioplastic closure and closed aneurysmorrhaphy of two second-order segmental arterial aneurysms.

Table III. Summary of evidence on surgical and endovascular reconstructive options for renal artery aneurysms (RAAs)

Series	No.	Mean F/U, months	Approach	Morbidity	Secondary nephrectomy	Mortality	Primary patency	Vascular or renal reintervention
Hupp ¹⁷	19/18	NR	IS	'2 early occlusions' described	6%	0	89.5%	22%
Henke ⁷	168/121	91	IS N	11.5%	21%	0	NR	5.8%
Pfeiffer ¹³	94	46	IS	17%	0	0	75%-97%	6%
English ¹⁶	84/62	47	IS N	12%	0	1.6%	93%-100%	0
Chandra ⁸	10	10-12	IS EV	20%	0	0	NR	0
Robinson ¹²	26/24	99	EV	11.5%	4%	0	94%	4%
Tsilimparis ¹⁵	20	25	IS EV N	15%	5	0		10%
Morita ²²	29	NR	IS EV	56% 'long-term'	0	0	NR	14%
Klausner ⁹	16/15	36	IS EV	20%	0	0	NR	0
Klausner ¹⁰	168/149	NR	IS EV N	19% 'minor'/ 8% 'major'	3%	1.3%	NR	NR
Etezadi ²³	17	45	S	NR	0	0		6%
Sédat ²⁴	15	38	Embo S	13%	0	0		13%
Zhang ²⁵	15	25	Embo (±balloon assist) S	60%	0	0		0
Tsilimparis	24	27	Embo (±balloon assist) S Embo	17%	0	0		8%

Assist, Assistance; EV, ex vivo reconstruction; Embo, embolization; F/U, follow-up; IS, in situ reconstruction; N, planned nephrectomy; No., number of patients/number of aneurysms if known and stated; NR, not reported; S, stent.

between surveillance imaging may be appropriate, provided patient compliance with follow-up can be ensured (ie, 3 years). The antiquated size threshold for repair (2 cm) may, in fact, be too small. Given the poor prognosis of both mother and fetus with rupture, the potential for gestational rupture remains a valid indication for repair in women of child-bearing age. Medically refractory HTN also remains a compelling indication for repair in appropriately selected patients. Open reconstructions (in situ or ex vivo) remain a safe and durable therapy, while options for endovascular repair offer benefits to select patients.

AUTHOR CONTRIBUTIONS

Conception and design: DC
Analysis and interpretation: DC
Data collection: DC
Writing the article: DC
Critical revision of the article: DC, JS
Final approval of the article: DC, JS
Statistical analysis: Not applicable
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