Natural history of renal artery aneurysms

Jason Zhang, MD,[a] Keerthi Harish, BS,[a] Giancarlo Speranza, BS,[a] Charlotte A. Hartwell, MD,[b] Karan Garg, MD,[a] Glenn R. Jacobowitz, MD,[a] Mikel Sadek, MD,[a] Thomas Maldonado, MD,[a] Danny Kim, MD,[b] *and* Caron B. Rockman, MD,[a] *New York, NY*

ABSTRACT

Objective: The existing renal artery aneurysm (RAA) literature is largely composed of reports of patients who underwent intervention. The objective of this study was to review the natural history of RAA.

Methods: This single-institution retrospective analysis studied all patients with RAA diagnosed by computed tomography imaging between 2015 and 2019, identified by our institutional radiology database. Imaging, demographic, and clinical data were obtained via the electronic medical record. He growth rate was calculated for all patients with radiological follow-up.

Results: The cohort consists of 331 patients with 338 RAAs. Most patients were female (61.3%), with 11 (3.3%) of childbearing age. The mean age at diagnosis was 71.5 years (range, 24-99 years). Medical comorbidities included hypertension (73.7%), prior smoking (34.4%), and connective tissue disease (3.9%). Imaging indications included abdominal pain (33.5%), unrelated follow-up (29.6%), and follow-up of an RAA initially diagnosed before the study period (10.7%). Right RAA (61.9%) was more common than left (35.1%); 3% of patients had bilateral RAA. The mean diameter at diagnosis was 12.9 ± 5.9 mm. Size distribution included lesions measuring less than <15 mm (69.9%), 15 to 25 mm (27.1%), and more than 25 mm (3.0%). Anatomic locations included the distal RA (26.7%), renal hilum (42.4%), and mid-RA (13.1%). The majority were true aneurysms (98%); of these, 72.3% were fusiform and 27.7% were saccular. Additional characteristics included calcification (82.2%), thrombus (15.9%), and dissection (0.9%). Associated findings included aortic atherosclerosis (65.6%), additional visceral aneurysms (7.3%), and abdominal aortic aneurysm (5.7%). The mean clinical follow-up among 281 patients was 41.0 ± 24.0 months. The mean radiological follow-up among 137 patients was 26.0 ± 21.4 months. Of these, 43 patients (31.4%) experienced growth, with mean growth rate of 0.23 ± 4.7 mm/year; the remainder remained stable in size. Eight patients eventually underwent intervention (5 endovascular), with the most common indications including size criteria (4/8) and symptom development (3/8). No patient developed rupture. On multivariate analysis, obesity (P = .04) was significantly associated with growth. An initial diameter of more than 25 mm was significantly associated with subsequent intervention (P = .006), but was not significantly associated with growth. Four of five RAAs with an initial diameter 30 mm or greater did not undergo intervention. The mean clinical follow-up for these patients was 24 months; none developed rupture and two remained stable in size.

Conclusions: This large institutional cohort found that the majority of RAAs remained stable in size, with few patients meeting indications for repair based on societal guidelines. Current guidelines recommending intervention for asymptomatic aneurysms more than 30 mm seem to be appropriate given their slow progression. (J Vasc Surg 2023;77:1199-205.)

Keywords: Renal artery; Renal artery aneurysm; Natural history; Growth rate; Asymptomatic

Renal artery aneurysms (RAA) are not infrequently encountered, although with limited reports in the literature. The reported incidence of RAAs varies: 0.7% on computed tomography (CT) scans[1] and between 0.3% and 2.5% on arteriograms.[2-4] With the increasing use and availability of CT scans,[5] however, it is possible that the incidence of RAAs will increase because they are commonly detected as incidental findings. Traditionally, indications for repair of RAAs included size criteria of greater than 2 cm,[6] females of childbearing age, symptoms including pain and hematuria, medically refractory hypertension, and rupture.[2,7]

From the Division of Vascular and Endovascular Surgery, Department of Surgery,[a] and Department of Radiology,[b] New York University Langone Medical Center.

Author conflict of interest: none.

Presented at the 2022 Society for Vascular Surgery Vascular Annual Meeting, Boston, MA, June 14-17, 2022.

Additional material for this article may be found online at www.jvascsurg.org.

Correspondence: Caron Rockman, MD, Division Vascular and Endovascular Surgery Department of Surgery New York University Langone Medical Center, NYU Grossman School of Medicine, 550 1st Ave, New York, NY, 10016 (e-mail: caron.rockman@nyulangone.org).

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

https://doi.org/10.1016/j.jvs.2022.11.010

However, multiple retrospective studies have demonstrated a low growth rate of RAAs ranging from 0.06 to 0.86 mm/year,[8-11] and most contemporary authors have found minimal evidence of rupture for those undergoing nonoperative surveillance.[2,4,11,12] Given the low growth and rupture rate, the Society for Vascular Surgery clinical practice guidelines now advocate repair only for those with symptomatic RAAs, RAAs greater than 3 cm, all RAAs in women of childbearing age, and in patients with refractory hypertension and renal artery stenosis.[13]

Although the threshold for intervention for RAAs has increased in contemporary times, the overall rupture rate is still estimated to be 3% to 5%, with nongestational mortality of greater than 10%.[2] The largest study to date demonstrated no ruptures for RAAs measuring 2 to 3 cm, but found an 18% rate of rupture for those greater than 3 cm, demonstrating the continued importance of surveillance for these aneurysms.[10] Given the still limited evidence of the natural history of RAAs, particularly regarding growth, our study sought to further investigate outcomes of RAAs treated nonoperatively at a single institution.

ARTICLE HIGHLIGHTS

- **Type of Research:** Single-center retrospective cohort study
- **Key Findings:** Clinical follow-up of 281 patients at mean of 41 months with renal artery aneurysms (RAAs) demonstrated no evidence of rupture. For 137 patients with radiological follow-up, the mean growth rate was 0.23 mm/year. Obesity was significantly associated with growth of RAA on multivariate analysis ($P = .04$).
- **Take Home Message:** The natural history of RAAs is benign with indolent growth. Routine surveillance is appropriate for asymptomatic, small aneurysms.

METHODS

Patient cohort. This study is a single-institution retrospective analysis. The New York University Langone Hospital Radiology Database was searched with specific terms such as "renal artery aneurysm" and "visceral artery aneurysm" in radiology diagnostic reports of abdominal imaging studies. The study included reports from January 2015 to December 2019. Inclusion criteria included patients age 18 years and older diagnosed with RAAs noted on an abdominal CT scan. Patients without follow-up data were excluded from follow-up analysis, but initial aneurysm characteristics were analyzed and included in the study cohort. Data collected included patient demographics, medical history, comorbidities, management, and follow-up data. Childbearing age for women was defined as 18 to 49 years of age. Consent of individual patients for study inclusion was not obtained because there was no identifiable information. The present study received approval for exemption from the New York University Institutional Review Board as a retrospective chart review.

Imaging data collection and follow-up. All CT scans were reviewed by the authors to confirm the findings noted on the radiology report. Three separate authors independently reviewed each individual scan and made measurements of the aneurysm sizes to help account for variations in measurement technique. All aneurysms, defined as focal dilations of more than 50% of the normal renal artery diameter, were analyzed. Data collected included laterality, mean diameter at diagnosis (measured from outer wall to outer wall), characteristics (true vs pseudoaneurysm, presence of calcification or thrombus), associated findings on scan (presence of abdominal aortic aneurysm, evidence of atherosclerosis in the aorta, presence of additional visceral aneurysms), whether intervention was performed, and presence of symptoms. Indications for intervention were extrapolated from chart review during clinic visits with the vascular surgeon, and size criteria refer to the previous 2-cm benchmark. The anatomic location of the aneurysm was also divided into the proximal renal artery, mid renal artery, distal renal artery, renal bifurcation (division of anterior and posterior divisions), and at the renal hilum. Symptomatic aneurysms were defined as flank pain, hematuria, and medically refractory hypertension.[14] Follow-up was categorized as either clinical follow-up or radiological follow-up. Clinical follow-up was recorded by searching the electronic medical record and measuring the time from initial imaging scan to the most recent clinic appointment irrespective of specialty. Radiological follow-up was calculated for patients with at least two CT scans in the electronic health record within the aforementioned time period. Radiological follow-up was recorded by calculating the time from the initial scan documenting RAA to the most recent scan within the study time period. Our primary objective was to determine the growth rate for patients with multiple CT scans. Secondary outcomes included freedom from growth, freedom from rupture or intervention, and factors associated with growth.

Statistical analyses. Data are reported using means and standard deviations for continuous variables or frequencies for categorical variables. Growth rate was calculated by dividing growth (in millimeters) by follow-up from first to last scan (in years) for each individual patient. A multivariate analysis was performed to evaluate the effect of comorbidities and aneurysm characteristics on growth. Kaplan-Meier curves were used to estimate freedom from growth, and freedom from rupture or intervention. Statistical analysis was performed using SPSS 25.0 (IBM Inc, Armonk, NY).

Table I. Demographics of patients with renal artery aneurysm (RAA)

Characteristic	No. of patients (%)
Demographics	
Total patients	331 (100)
Age at initial presentation, years	71.5 ± 12.8 (range, 24-99)
Female	203 (61.9)
Indication for imaging	
Abdominal pain	110 (33.5)
Hypotension or tachycardia	3 (0.9)
Follow-up known aneurysm	35 (10.7)
Follow-up other pathology	97 (29.6)
Other/unknown	93 (27.7)
Comorbidities	
Hypertension	244 (73.7)
Active tobacco use	19 (5.7)
Prior tobacco use	114 (34.4)
Diabetes	69 (20.8)
Coronary artery disease	72 (21.8)
Connective tissue disease	13 (3.9)
Prior stroke	22 (6.6)
Peripheral artery disease	22 (6.6)
Hypercholesterolemia	179 (54.1)
Prednisone or immunosuppressive medication use	22 (6.6)
Obesity	47 (14.2)
No comorbidities	28 (8.5)

RESULTS

Patient demographics. There were a total of 331 patients with 338 RAAs in our cohort. The approximate yearly incidence of RAA was 0.36%; our institution performs an average of 18,300 abdominal CT scans per year. Patient demographics are provided in Table I. The mean age at diagnosis was 71.5 ± 12.8 years (range, 24-99 years). The majority of patients were female (61.9%), with 11 (3.3%) of childbearing age. Medical comorbidities included hypertension (73.7%), prior tobacco use (34.4%), diabetes (20.8%), hypercholesterolemia (54.1%), coronary artery disease (21.8%), and the presence of connective tissue disease (3.9%). The indications for imaging included abdominal pain (33.5%), hypotension or tachycardia (0.9%), follow-up of a previously documented RAA (10.7%), and follow-up of a pathology not related to RAA (29.6%). The study included three patients with confirmed fibromuscular dysplasia (FMD) and one patient with suspected FMD based on imaging.

RAA characteristics. Aneurysm characteristics are presented in Table II. The majority of patients had right RAA (61.9%); 3.0% of patients had bilateral RAAs. The mean diameter at diagnosis was 12.9 ± 5.9 mm. Size distribution of aneurysms included RAAs measuring less than 15 mm (69.9%), 15 to 25 mm (27.1%), and more than 25 mm (3.0%). RAAs were most commonly found at the renal hilum (42.4%), with RAAs also commonly found in the distal renal artery (26.7%), mid renal artery (13.1%), and the renal artery bifurcation (12.8%). The majority of aneurysms were radiologically classified as true aneurysms (98%); of these, 72.3% were fusiform and 27.7% were saccular. Additional morphological characteristics included calcification (82.2%), thrombus (15.9%), and dissection (0.9%). Associated findings on imaging included the presence of abdominal aortic aneurysm (5.8%), aortic atherosclerosis (66.2%), and additional visceral aneurysms (7.3%).

Clinical and radiological follow-up. A total of 281 patients had a mean clinical follow-up of 41.0 ± 24.0 months. Of the 50 patients who did not have follow-up in the health record, there were 32 deaths not attributable to the aneurysm and 18 patients who were lost to follow-up. Follow-up data are depicted in Fig 1. No patients developed rupture. Eight patients (2.8%) eventually underwent intervention, with the most common indications being size criteria (>20 mm; 4/8) and symptom development (two hematuria, one abdominal pain). The indication for repair for the last patient was unable to be ascertained during chart review. Of these patients, five underwent endovascular intervention (three covered stents and two coil embolizations), and three underwent open repair (two aneurysmorrhaphy and one aortorenal bypass with a prosthetic graft). There was no reported perioperative mortality, and symptomatic patients had resolution of symptoms after intervention. One patient with initial stent graft placement required extension of the stent for incomplete occlusion of RAA, and subsequently developed a small type II endoleak at 2 weeks postoperatively with plan for observation. There were no other instances of perioperative morbidity. All eight patients were alive at a mean clinical follow-up of 42.1 ± 27.5 months.

The mean radiological follow-up among 137 patients was 26.0 ± 21.4 months. Of the original 331 patients, 194 did not have radiological follow-up. There was no difference in the mean size at diagnosis between those with radiological follow-up and those without radiological follow-up (13.395 ± 5.551 mm vs 12.677 ± 6.082 mm; $P = .280$). Of the 137 patients with two or more scans, 43 (31.4%) experienced growth of RAA, with a mean growth rate of 0.23 ± 4.7 mm/year and a median growth rate of 0 mm/year. For 92 RAAs with an initial size of less than 15 mm, the mean follow-up was 28.2 ± 22.7 months, with a mean growth rate of 0.19 ± 4.43 mm/year. For 41 RAAs with an initial size 15 mm or more and 25 mm or less, the mean radiological follow-up was 22.8 ± 18.1 months, with a mean growth rate of 0.38 ± 1.34 mm/year. Four patients with imaging follow-up had an initial RAA measuring more than 25 mm with mean

Table II. Renal artery aneurysm (*RAA*) characteristics and associated imaging findings

Aneurysm characteristic (n = 338)	Percent of total
Laterality	
Right	61.9
Left	35.1
Bilateral	3.0
Size	
Mean diameter at diagnosis, mm	12.9 ± 5.9
RAA <15 mm	69.9
RAA 15-25 mm	27.1
RAA >25 mm	3.0
Location	
Proximal renal artery	5.2
Mid renal artery	13.1
Distal renal artery	26.7
Renal bifurcation	12.8
Renal hilum	42.4
Characteristics	
Fusiform	72.3
Saccular	27.7
True aneurysm	98.0
Pseudoaneurysm	2.0
Calcification	82.2
Thrombosis	15.9
Dissection	0.9
Rupture	0
Associated imaging findings (n = 331)	
Presence of AAA	19 (5.7)
Atherosclerosis of abdominal aorta/branches	217 (65.6)
Additional visceral aneurysms	24 (7.3)
Additional visceral anomalies other than aneurysm	16 (4.8)

AAA, Abdominal aortic aneurysm.

follow-up of 7.1 ± 4.8 months and a mean growth rate of −2.0 ± 4.9 mm/year. Three FMD patients with imaging follow-up did not experience growth.

Five total patients in the dataset had an initial diameter of more than 30 mm. All five patients had clinical follow-up, with a mean clinical follow-up of 23.9 ± 24.0 months. One patient underwent stent placement for an aneurysm diameter of 37 mm. Two patients had radiological follow-up at 3.6 and 12.3 months, respectively, with stable RAAs. The remaining two patients did not have follow-up scans.

On Kaplan-Meier analysis, there was estimated radiological freedom from growth of 40.8% at 51 months (Fig 2). Clinically, freedom from rupture or intervention was estimated as 86.0% at 130 months (Fig 3).

On univariate analysis (Supplementary Table, online only), hypertension was associated with a decreased risk of aneurysm growth (odds ratio [OR], 0.35; 95% confidence interval [CI]. 0.33-0.91; $P = .03$), whereas obesity was associated with an increased risk of growth (OR, 3.34; 95% CI, 1.13-9.92; $P = .03$). On multivariate analysis shown in Table III, only obesity was found to be associated with increased risk of growth (OR, 7.53; 95% CI, 1.11-51.2; $P = .04$). Other factors including age, hypertension, laterality, location of aneurysm, diabetes, thrombosis, calcification, and gender were not found to be predictive or protective from growth of aneurysm. An initial diameter of more than 25 mm was significantly associated with subsequent intervention ($P = .006$), but was not significantly associated with growth.

DISCUSSION

Despite recent Society for Vascular Surgery guidelines advocating for more a more conservative diameter intervention threshold of RAAs,[13] there remains a paucity of literature of the natural history of RAAs. Our single-institution study aimed to further delineate the behavior of RAAs over time with a focus on growth and the rupture rate. The findings of the current study were similar to contemporary reports of minimal to slow growth over time with a low risk of rupture.[4,8-12,15] No patients developed rupture at a clinical follow-up of 41 months, and only eight patients underwent intervention. For patients with radiological follow-up, a minority of patients had growth (31.4%). Overall, the mean growth rate was a modest 0.23 mm/year increase in diameter over a mean follow-up of 26 months. These findings suggest that most asymptomatic RAAs tend to be benign with indolent growth patterns.

Several similar retrospective studies observing RAAs exist in the literature. Brownstein et al[11] observed 171 RAAs for a mean follow-up of 41 months and found an overall growth rate of 0.017 cm/year, with only 24% of aneurysms increasing in size and no ruptures. Wayne et al[9] found a similarly slow growth in their cohort of 68 RAAs observed for a mean of 19.4 months with median annualized growth rate of 0.06 mm/year and no ruptures. The largest multicenter study of RAAs to date by Klausner et al[10] (n = 865 RAAs) included imaging follow-up of 454 RAAs with a growth rate of 0.086 cm/year and 64.5% of RAAs demonstrating no growth, with no ruptures for 88 conservatively managed patients at 29 months. Only three ruptures occurred in the overall cohort of 865 RAAs, and all occurred at presentation in patients with aneurysms measuring more than 3 cm, for an overall rupture rate of 18% for RAAs measuring more than 3 cm. Our study did not have any ruptures at presentation or during follow-up. Among five patients with aneurysms measuring more than 3 cm, four were observed with no evidence of rupture, and one patient underwent endovascular stent exclusion of a 3.7-cm RAA.

Additional studies that did not specifically observe growth rates found similarly low rates of rupture. Henke

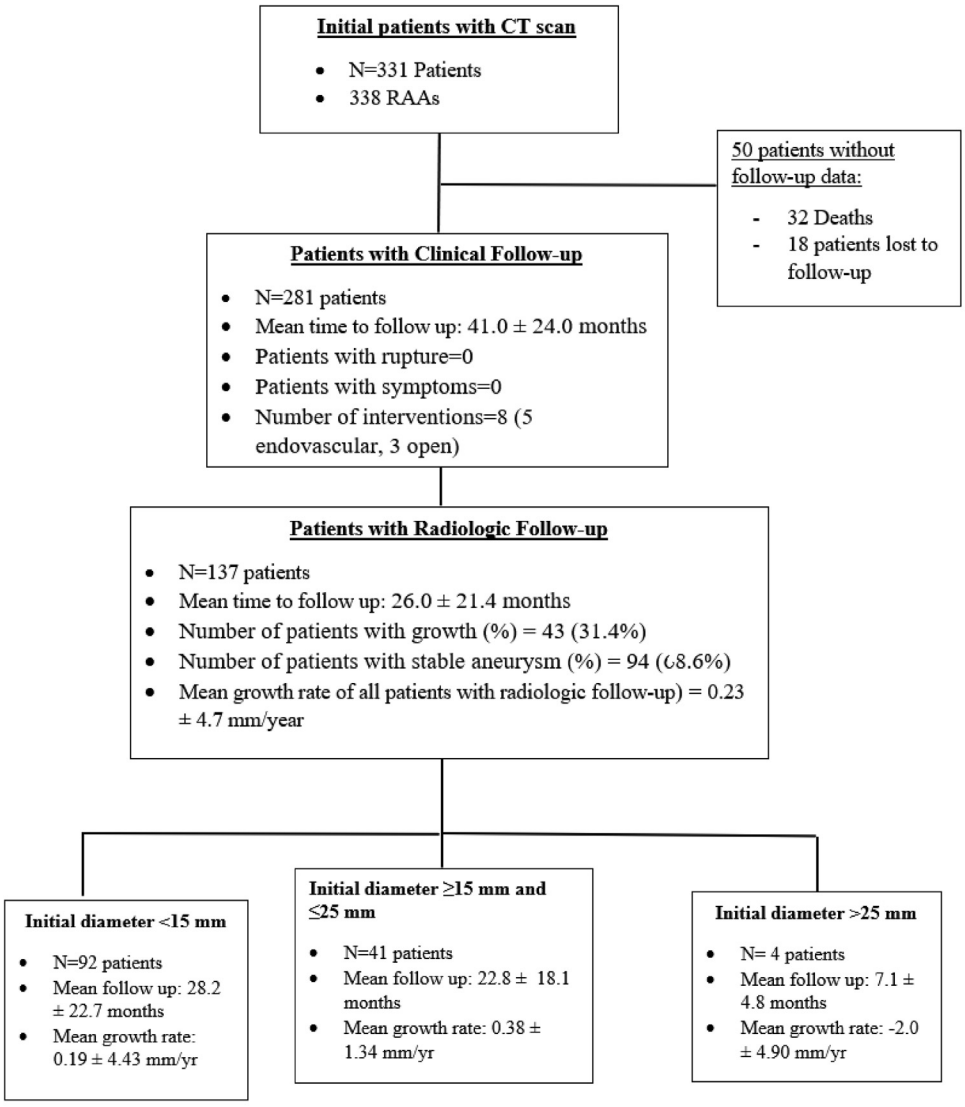

Fig 1. Flow chart depicting clinical and radiological follow-up for patients with renal artery aneurysms (*RAA*). *CT*, Computed tomography.

et al[12] observed 86 RAAs over 72 months with no ruptures. Tham et al[4] followed 69 patients with RAA treated conservatively over 4.3 years with no evidence of rupture, and none of 9 deaths in the cohort were attributable to the RAA. Finally, Morita et al[15] observed 30 RAAs over 69 months, with no ruptures and two patients developing an increase in aneurysm size during follow-up. These historical studies and the present one demonstrate that although the contemporary rate of rupture for RAA has been cited to be 3% to 5%,[2] the true rate may be lower. Although the low rate of rupture likely reflects the benign nature of RAAs, it is possible that the rate was also decreased by timely interventions in patients with large or symptomatic aneurysms.

Regarding demographics and aneurysm characteristics, our data are consistent with literature that patients with RAA are predominantly female and most have hypertension. The rate of hypertension has consistently been cited to be approximately 70%,[2,11,12] which is comparable with the 74.4% rate of hypertensive patients in our study. Female predominance has been cited up to 72%,[2] which has been thought secondary to the greater prevalence of FMD and other connective tissue diseases among females.[16] Although our study also had majority female patients (61.9%), only 3.9% of our cohort had documented connective tissue disease. Although this discrepancy may be partly attributable to the difficulty in diagnosing connective tissue disease, the clinical importance is unclear; being of female sex was not predictive of increased growth. There was, however, a 5.8% rate of associated abdominal aortic aneurysm and 7.3% rate of additional visceral artery aneurysm for patients with RAA; these rates support the possibility of systemic vascular degenerative disease either from connective tissue disease or classical risk factors such as smoking and atherosclerosis. Interestingly, although the high rates of calcification (82.2%) in

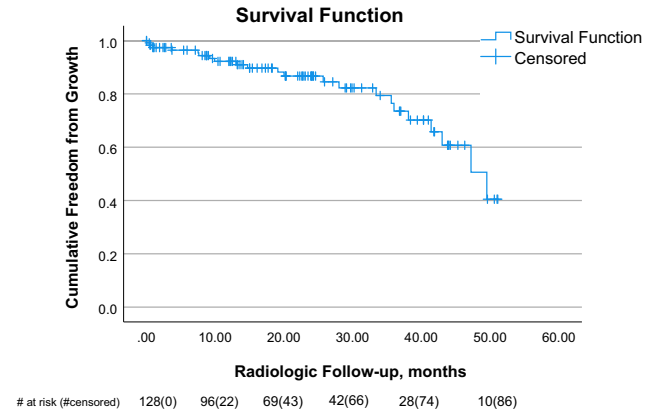

Fig 2. Kaplan-Meier curve showing freedom from growth on radiologic follow-up.

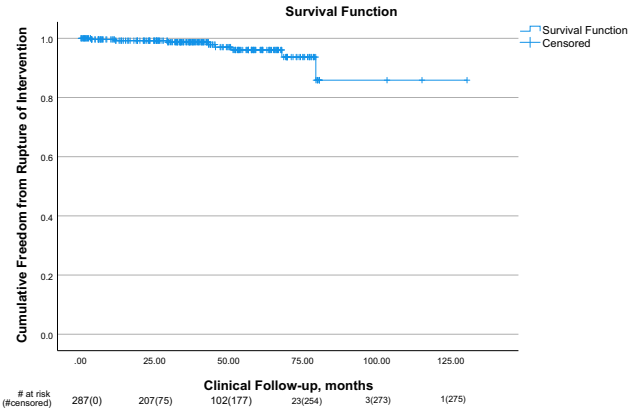

Fig 3. Kaplan-Meier curve showing freedom from intervention or rupture on clinical follow-up.

our study are similar with previous literature,[2,9,11] the location and type of aneurysm differs. The majority of RAAs presented in the literature typically present as saccular aneurysms at the renal bifurcation[2,10-12,16]; however, our study found primarily fusiform aneurysms (72.3%) at the renal hilum (42.4%) distal to the bifurcation. The etiology of these differences is unclear; other aneurysms characteristics are comparable, although the overall small sample size of RAAs studied may account for variations in presentation.

Few studies have reported associations between patient and aneurysm characteristics and RAA growth. Brownstein et al[11] found a mild positive association between hypertension and growth (OR, 1.011; $P = .01$), and mild decreased risk of growth for those with smoking history (OR, 0.987; $P < .001$), female sex (OR, 0.987; $P < .001$), mid renal artery location (OR, 0.983; $P = .001$), distal renal artery location (OR, 0.990; $P = .003$), renal artery bifurcation location (OR, 0.992; $P = .03$), and left laterality (OR, 0.993; $P = .03$). Interestingly, our study did not find a significantly positive or negative association with any of these factors on multivariate analysis, indicating that these factors likely have little effect on RAA growth clinically. We did find an increased risk of growth with obesity (OR, 7.53; $P = .04$), which is consistent with the literature demonstrating obesity as a risk factor for increased aneurysm development.[17] Calcification had previously been thought to be protective from RAA rupture[16]; however, more recent studies have not found any association with growth,[10,11] as was reflected in this study (OR, 0.2; 95% CI, 0.02-1.83; $P = .15$).

The surgical management of RAAs has evolved over time. Historically, all RAAs were repaired in an open fashion, via a variety of techniques including primary repair with or without patch angioplasty, interposition bypass, aortorenal bypass, splanchnorenal bypass, or plication of smaller aneurysms.[2] Several retrospective studies have demonstrated that conventional open repair is associated with generally low morbidity and mortality with patency rates of 81% to 100% at the time of follow-up.[11,18-21] Our study included three open repairs with two patients undergoing aneurysmorrhaphy and one aortorenal bypass with no perioperative or long-term complications, supporting the continued feasibility of open surgery. With the advent of improved technology, endovascular treatment of RAAs has increased and has become the preferred treatment for patients with amenable anatomy; typically, this procedure entails coil embolization for distal and parenchymal aneurysms and stenting for more proximal lesions with sufficient landing zone.[22] Although the cohorts of endovascularly treated RAAs are smaller, multiple studies have demonstrated high rates of technical success between 94% and 100%.[23-25] One patient in our study required extension of an initially placed stent, with continued endoleak after, demonstrating that stenting for RAA can lead to similar complications as endovascular abdominal aortic aneurysm repair.

Our study has several limitations. This study was performed in a retrospective manner with a predetermined time frame so that patients with diagnoses closer to the end point have systematically less follow-up available. Additionally, as noted in a study with similar methodology (Brownstein et al[11]) the large number of patients in our study with RAAs found incidentally may predispose our cohort to having a more benign natural history, a form of selection bias. Our method of collecting clinical follow-up only was only able to definitively determine whether a patient was alive or deceased, but could not absolutely ascertain if a patient was symptomatic. Additionally, we did not control for differences in CT imaging modality (angiography vs noncontrast), which may have resulted in small discrepancies in measurements. Finally, because our study did not observe any ruptures, we could not determine any risks factors predictive of RAA rupture.

Table III. Multivariate regression analysis of patient and aneurysm characteristics on renal artery aneurysm (RAA) growth

Factor	OR	95% CI	*P* value
Active tobacco use	33.03	0.83-1309.89	.06
Prior tobacco use	2.45	0.38-15.84	.35
Age	1.03	0.94-1.13	.49
Hypertension	0.70	0.08-6.09	.75
Diabetes	0.07	0.00-1.58	.10
Coronary artery disease	0.32	0.02-4.12	.38
Hypercholesterolemia	0.60	0.09-3.78	.60
Obesity	7.53	1.11-51.2	**.04**
Location of aneurysm	0.99	0.44-2.23	.97
Thrombosis	1.16	0.11-12.67	.90
Calcification	0.20	0.02-1.83	.15
Immunosuppression	0.26	0.02-4.13	.34
Gender	4.24	0.75-23.87	.10

CI, Confidence interval; *OR*, odds ratio.
Boldface entries indicate statistical significance.

CONCLUSIONS

This large, single-center study is consistent with previous findings that the natural history of RAAs tends to be benign, with the majority of RAAs remaining stable in size with no ruptures and few interventions. Current guidelines recommending a RAA size threshold of more than 3 cm for intervention seem to be appropriate, given their slow progression. For patients with incidentally found, asymptomatic RAAs, routine imaging and clinical follow-up should be the primary management.

AUTHOR CONTRIBUTIONS

Conception and design: JZ, KH, GS, CH, KG, GJ, MS, TM, DK, CR
Analysis and interpretation: JZ, KH, GS, CR
Data collection: JZ, KH, GS, CH
Writing the article: JZ, KH, CR
Critical revision of the article: JZ, KH, GS, CH, KG, GJ, MS, TM, DK, CR
Final approval of the article: JZ, KH, GS, CH, KG, GJ, MS, TM, DK, CR
Statistical analysis: Not applicable
Obtained funding: Not applicable
Overall responsibility: CR

REFERENCES

1. Zhang LJ, Yang GF, Qi J, Shen W. Renal artery aneurysm: diagnosis and surveillance with multidetector-row computed tomography. Acta Radiol 2007;48:274-9.
2. Coleman DM, Stanley JC. Renal artery aneurysms. J Vasc Surg 2015;62:779-85.
3. Henriksson C, Lukes P, Nilson AE, Pettersson S. Angiographically discovered, non-operated renal artery aneurysms. Scand J Urol Nephrol 1984;18:59-62.
4. Tham G, Ekelund L, Herrlin K, Lindstedt EL, Olin T, Bergentz SE. Renal artery aneurysms. Natural history and prognosis. Ann Surg 1983;197:348-52.
5. Drescher FS, Sirovich BE. Use of computed tomography in emergency departments in the United States: a decade of coughs and colds. JAMA Intern Med 2016;176:273-5.
6. Hageman JH, Smith RF, Szilagyi E, Elliott JP. Aneurysms of the renal artery: problems of prognosis and surgical management. Surgery 1978;84:563-72.
7. McCarron JP Jr, Marshall VF, Whitsell JC 2nd. Indications for surgery on renal artery aneurysms. J Urol 1975;114:177-80.
8. Klausner JQ, Harlander-Locke MP, Plotnik AN, Lehrman E, DeRubertis BG, Lawrence PF. Current treatment of renal artery aneurysms may be too aggressive. J Vasc Surg 2014;59:1356-61.
9. Wayne EJ, Edwards MS, Stafford JM, Hansen KJ, Corriere MA. Anatomic characteristics and natural history of renal artery aneurysms during longitudinal imaging surveillance. J Vasc Surg 2014;60:448-52.
10. Klausner JQ, Lawrence PF, Harlander-Locke MP, Coleman DM, Stanley JC, Fujimura N; Vascular Low-Frequency Disease Consortium. The contemporary management of renal artery aneurysms. J Vasc Surg 2015;61:978-84.
11. Brownstein AJ, Erben Y, Rajaee S, Li Y, Rizzo JA, Mojibian H, et al. Natural history and management of renal artery aneurysms in a single tertiary referral center. J Vasc Surg 2018;68:137-44.
12. Henke PK, Cardneau JD, Welling TH 3rd, Upchurch GR Jr, Wakefield TW, Jacobs LA, et al. Renal artery aneurysms: a 35-year clinical experience with 252 aneurysms in 168 patients. Ann Surg 2001;234:454-62; discussion: 462-3.
13. Chaer RA, Abularrage CJ, Coleman DM, Eslami MH, Kashyap VS, Rockman C, et al. The Society for Vascular Surgery clinical practice guidelines on the management of visceral aneurysms. J Vasc Surg 2020;72:3S-39S.
14. Poutasse EF. Renal artery aneurysms. J Urol 1975;113:443-9.
15. Morita K, Seki T, Iwami D, Sasaki H, Fukuzawa N, Nonomura K. Long-term outcome of single institutional experience with conservative and surgical management for renal artery aneurysm. Transplant Proc 2012;44:1795-9.
16. Mercier C, Piquet P, Piligian F, Ferdani M. Aneurysms of the renal artery and its branches. Ann Vasc Surg 1986;1:321-7.
17. Golledge J, Clancy P, Jamrozik K, Norman PE. Obesity, adipokines, and abdominal aortic aneurysm: health in men study. Circulation 2007;116:2275-9.
18. English WP, Pearce JD, Craven TE, Wilson DB, Edwards MS, Ayerdi J, et al. Surgical management of renal artery aneurysms. J Vasc Surg 2004;40:53-60.
19. Pfeiffer T, Reiher L, Grabitz K, Grünhage B, Häfele S, Voiculescu A, et al. Reconstruction for renal artery aneurysm: operative techniques and long-term results. J Vasc Surg 2003;37:293-300.
20. Chandra A, O'Connell JB, Quinones-Baldrich WJ, Lawrence PF, Moore WS, Gelabert HA, et al. Aneurysmectomy with arterial reconstruction of renal artery aneurysms in the endovascular era: a safe, effective treatment for both aneurysm and associated hypertension. Ann Vasc Surg 2010;24:503-10.
21. Kreienberg PB, Darling RC, Chang BB, Paty PK, Lloyd W, Papanicolaou G, et al. Surgical management of extraparenchymal renal artery aneurysms. Vasc Surg 1998;32:587-93.
22. Xie D, Gheiler E, He X. Interventional and surgical treatments for renal artery aneurysms. J Clin Nephrol Ren Care 2017;3:019.
23. Etezadi V, Gandhi RT, Benenati JF, Rochon P, Gordon M, Benenati MJ, et al. Endovascular treatment of visceral and renal artery aneurysms. J Vasc Interv Radiol 2011;22:1246-53.
24. Sédat J, Chau Y, Baque J. Endovascular treatment of renal aneurysms: a series of 18 cases. Eur J Radiol 2012;81:3973-8.
25. Eldem G, Erdoğan E, Peynircioğlu B, Arat A, Balkancı F. Endovascular treatment of true renal artery aneurysms: a single center experience. Diagn Interv Radiol 2019;25:62-70.

Submitted Jul 15, 2022; accepted Nov 4, 2022.

Additional material for this article may be found online at www.jvascsurg.org.

Supplementary Table (online only). Univariate analysis of patient and aneurysm characteristics on renal artery aneurysm (RAA) growth

Factor	OR	95% CI	*P* value
Active tobacco use	2.23	0.51-9.66	.29
Prior tobacco use	0.87	0.34-2.27	.78
Age	1.00	0.96-1.04	.91
Hypertension	0.35	0.13-0.91	**.03**
Diabetes	0.40	0.11-1.45	.16
Coronary artery disease	0.45	0.16-2.23	.60
Connective tissue disease	0.81	0.13-5.06	.82
Peripheral artery disease	0.82	0.09-7.36	.86
Hypercholesterolemia	0.51	0.20-1.29	.16
Obesity	3.34	1.13-9.92	**.03**
Laterality	0.925	0.425-2.013	.711
Location of aneurysm	0.94	0.54-1.64	.94
Thrombosis	0.73	0.19-2.76	.64
Calcification	0.47	0.17-1.32	.15
Immunosuppression	0.35	0.04-2.84	.32
Gender	1.54	0.57-4.05	.40

CI, Confidence interval; *OR*, odds ratio.
Boldface entries indicate statistical significance.