From the Society for Vascular Surgery

The contemporary management of renal artery aneurysms

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Background: **Renal artery aneurysms (RAAs) are rare, with little known about their natural history and growth rate or their optimal management. The specific objectives of this study were to (1) define the clinical features of RAAs, including the precise growth rate and risk of rupture, (2) examine the current management and outcomes of RAA treatment using existing guidelines, and (3) examine the appropriateness of current criteria for repair of asymptomatic RAAs.**

Methods: **A standardized, multi-institutional approach was used to evaluate patients with RAAs at institutions from all regions of the United States. Patient demographics, aneurysm characteristics, aneurysm imaging, conservative and operative management, postoperative complications, and follow-up data were collected.**

Results: **A total of 865 RAAs in 760 patients were identified at 16 institutions. Of these, 75% were asymptomatic; symptomatic patients had difficult-to-control hypertension (10%), flank pain (6%), hematuria (4%), and abdominal pain (2%). The RAAs had a mean maximum diameter of 1.5 ± 0.1 cm. Most were unilateral (96%), on the right side (61%), saccular (87%), and calcified (56%). Elective repair was performed in 213 patients with 241 RAAs, usually for symptoms or size >2 cm; the remaining 547 patients with 624 RAAs were observed. Major operative complications occurred in 10%, including multisystem organ failure, myocardial infarction, and renal failure requiring dialysis. RAA repair for difficult-to-control hypertension cured 32% of patients and improved it in 26%. Three patients had ruptured RAA; all were transferred from other hospitals and underwent emergency repair, with no deaths. Conservatively treated patients were monitored for a mean of 49 months, with no acute complications. Aneurysm growth rate was 0.086 cm/y, with no difference between calcified and noncalcified aneurysms.**

Conclusions: **This large, contemporary, multi-institutional study demonstrated that asymptomatic RAAs rarely rupture (even when >2 cm), growth rate is 0.086 ± 0.08 cm/y, and calcification does not protect against enlargement. RAA open repair is associated with significant minor morbidity, but rarely a major morbidity or mortality. Aneurysm repair cured or improved hypertension in >50% of patients whose RAA was identified during the workup for difficult-to-control hypertension. (J Vasc Surg 2015;61:978-84.)**

Renal artery aneurysms (RAAs) are rare, with an estimated incidence of 0.09% in the general population.[1] Although uncommon, clinicians are more frequently encountering RAA due to the increased use of cross-sectional magnetic resonance and computed tomography (CT) imaging to evaluate other diseases.[1-3] Currently accepted indications for RAA repair include symptoms, size >2 cm, and aneurysms in women of childbearing age. These criteria are based on studies conducted before the widespread use of cross-sectional imaging.[1-6] Thus, there remains significant controversy surrounding RAA treatment criteria because the incidence, risk of rupture, and growth rate have not been determined.[4]

A contemporary single-institution study recently addressed issues of RAA growth rate and risk of rupture, based on aneurysm size, but the conclusions of the study were limited by small numbers.[7] Consequently, this multi-institutional study was conducted to (1) define the clinical features of RAA, including the precise growth rate and risk of rupture, (2) examine the current management and outcomes of RAA treatment using the existing guidelines, and (3) examine the appropriateness of current criteria for repair of asymptomatic RAA.

METHODS

Inclusion criteria and patient identification. RAAs were defined as focal, isolated dilatation of all three layers of the arterial wall that measured >1.5 times the diameter of the disease-free proximal adjacent arterial segment.[8] Patients with pararenal or juxtarenal aortic aneurysms and proximal RAAs that originated from an aortic aneurysm were excluded.

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Author conflict of interest: none.

Presented at the Plenary Session of the 2014 Vascular Annual Meeting of the Society for Vascular Surgery, Boston, Mass, June 4-7, 2014.

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

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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.2014.10.107

Patients were identified using International Classification of Diseases, Ninth Revision codes 442.1 (aneurysm of renal artery) and 442.89 (aneurysms of other specific artery) using physician, hospital, and radiology billing records. Principal investigators at each institution were responsible for ensuring inclusion of all patients at their institution. Symptomatic RAAs were defined by using previously published criteria, including flank pain, abdominal pain, hematuria, and difficult-to-control hypertension. Symptoms were attributed to the aneurysm if no other etiology was discovered or if the symptoms resolved after surgery, or both. The principal investigator from each institution was responsible for reviewing the primary aneurysm images and the reports for each patient and confirming size and growth data.

Database management. This multi-institutional study examined all patients presenting with RAAs between 2003 and 2013 at each hospital, including patient transfers. Pseudotraumatic, mycotic, and post-traumatic aneurysms were excluded. Primary end points included (1) morbidity and mortality of conservative management, (2) morbidity and mortality of repair, (3) freedom from acute complications and emergency repair (rupture), and (4) patient survival.

After Investigational Review Board approval, data were collected, deidentified, and stored in a password-encrypted central database managed by the Vascular Low-Frequency Consortium at the University of California, Los Angeles. Patient consent was not required by the Investigational Review Board due to the study's minimal risk and retrospective nature. Patient data from each institution were examined for accuracy and completeness by the consortia coordinators, and incomplete entries were corrected. Collective data were reviewed, critiqued, and modified by all study participants.

Statistics. Data were maintained in an Excel 14 database (Microsoft Corp, Redmond, Wash). Statistical analysis was performed using SPSS 20 software (IBM Corp, Armonk, NY). Continuous variables are presented as mean ± standard deviation, unless noted otherwise. Differences between subgroups were analyzed using independent Student *t*-test, Kruskal-Wallis test, Mann-Whitney *U* test, and analysis of variance. Differences between subgroups of noncontinuous variables were analyzed using the χ^2 test or Fisher exact test. Multivariable analysis was performed using binary and multinomial logistic regression. Cochran and Mantel-Haenszel methods were used to derive hazard ratios and 95% confidence intervals. All time-dependent variables were analyzed using Kaplan-Meier life tables. The maximum diameter for each aneurysm was determined using the same imaging modality in sequential imaging studies to reduce variability, and growth rate was determined using a weighted average. A P value of $<.05$ was considered significant.

RESULTS

Patient demographics and comorbidities. We identified 760 patients with 865 RAAs at 16 institutions from hospitals in different regions of North America (Supplementary Table, online only). The mean age at diagnosis was 61 ± 13 years (range, 12-99 years), and RAAs occurred predominantly in women (M:F = 1:2). Comorbidities (Table I) included hypertension (82%), with a mean blood pressure of 157/86 mm Hg (on a mean of two antihypertensive medications). Unlike patients with degenerative aneurysms, only 21% had a history of tobacco use. Concomitant extra-RAAs occurred in 14% of patients, the most common sites being the abdominal aorta and splenic artery.

Table I. Patient comorbidities and concomitant extrarenal aneurysms

Variables	*Patients (N = 760), No. (%)*
Comorbidities	
Hypertension	623 (82)
Hypercholesterolemia	190 (25)
Smoking	160 (21)
Diabetes mellitus	122 (16)
Coronary artery disease	76 (10)
Chronic obstructive pulmonary disease	30 (4)
Connective tissue disorder[a]	15 (2)
Extrarenal aneurysms	
Abdominal aorta	37 (5)
Splenic artery	23 (3)
Thoracic aorta	13 (2)
Iliac artery	12 (2)
Celiac artery	5 (1)
Hepatic artery	4 (1)

[a]Ehlers-Danlos syndrome, Marfan syndrome.

Table II. Presenting symptoms

Symptoms	*Patients (N = 760), No. (%)*
Asymptomatic	569 (75)
Difficult-to-control hypertension	76 (10)
Flank pain	46 (6)
Hematuria	30 (4)
Abdominal pain	15 (2)
Other (back pain, etc)	24 (3)

Most patients were asymptomatic, with the aneurysm discovered incidentally, and 25% presented with symptoms (Table II). No patient presented to a participant site with rupture; however, three patients with ruptured RAAs were transferred, after rupture, to institutions involved in this study.

Imaging and diagnosis. CT angiography was the most frequently used imaging modality for the RAA diagnosis (58%), CT (without contrast) was the next most frequent (24%), followed by magnetic resonance angiography (6%), catheter angiography (5%), and ultrasound imaging (4%).

Aneurysm characteristics. The distribution and location of RAAs are shown in Fig 1, with 61% located on the right side. The aneurysm most commonly originated in the

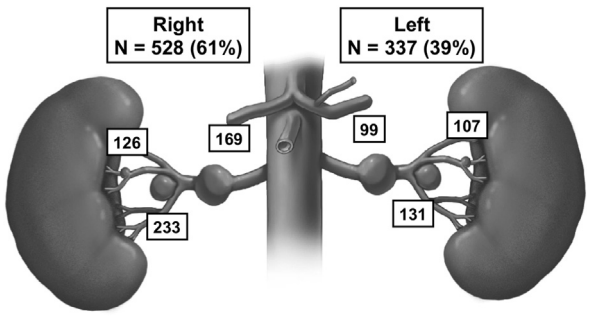

Fig 1. The distribution and location of renal artery aneurysms (RAAs).

Table III. Renal artery aneurysm (*RAA*) characteristics

Characteristics	*RAAs (N = 865), No. (%) or mean ± SD*
Laterality	
Unilateral	830 (96)
Bilateral	35 (4)
Morphology	
Saccular	753 (87)
Fusiform	95 (11)
Bilobed	17 (2)
Calcification	
Calcified	484 (56)
Non-calcified	381 (44)
Diameter, cm	1.5 ± 0.1

SD, Standard deviation.

main renal artery bifurcation (Table III), most were saccular, and 56% were calcified. Six patients had bilateral aneurysms and were observed. The remaining RAAs were located on the same kidney. The mean diameter of all RAAs was 1.5 ± 0.1 cm, the diameter of symptomatic RAAs was 1.9 ± 0.1 cm, and the diameter of asymptomatic RAAs was 1.5 ± 0.1 cm ($P < .001$). A mean of two efferent arterial branches exited from the RAA. Eleven percent of aneurysms contained mural thrombus, 4% were associated with ipsilateral fibromuscular dysplasia, and 3% were associated with renal artery stenosis.

Treatment. The management of patients with RAAs varied (Fig 2) by clinical presentation. In the 25% with symptomatic RAAs, 128 (mean diameter, 2.3 cm) were repaired and 77 (mean diameter, 1.3 cm) were observed. In asymptomatic aneurysms, 113 (mean diameter, 2.4 cm) were electively repaired and 547 (mean diameter, 1.3 cm) were observed. RAA repairs included 168 open repairs (OR) and 73 endovascular (EV) procedures. The mean diameters were 2.3 ± 0.1 cm for OR aneurysms, 2.3 ± 0.2 cm for EV aneurysms, and 1.3 ± 0.1 cm for observed aneurysms. Treated aneurysms were significantly larger than observed aneurysms ($P < .001$). Among OR aneurysms, 42% originated at the main bifurcation; among EV aneurysms, 46% originated in the main trunk. There was no difference between the location of observed, OR, and EV aneurysms, but there were significantly more efferent branches in those treated with OR, compared with those treated with EV or observed ($P < .001$). Symptomatic patients who were operatively treated underwent elective repair 3 ± 1 months after the initial diagnosis, whereas asymptomatic patients underwent elective repair 6 ± 3 months after the initial diagnosis. In the OR group, there were 15 potential childbearing women based on age <45 years.

A total of 113 asymptomatic aneurysms, which were initially managed conservatively, were ultimately repaired in 91 patients (Fig 3). The indication for repair was size >2 cm (73%), concomitant repair with abdominal aortic aneurysm repair (12%), development of symptoms (8%), rapid enlargement (4%), and patient choice (3%). Asymptomatic aneurysms that were repaired had a mean diameter of 2.4 ± 0.1 cm (range, 1.4-5.3 cm); 43 asymptomatic aneurysms <2 cm were repaired. Aneurysms repaired for size >2 cm had a mean maximum diameter of 2.6 ± 0.1 cm (range, 2.0-3.7 cm), and aneurysms repaired for rapid enlargement had a mean diameter of 2.2 ± 0.2 cm (range, 1.1-2.8 cm).

In 45 patients (37%) who underwent aneurysm repair, symptoms that led to repair did not resolve: hypertension did not resolve in 34 patients, hematuria did not resolve in 5, flank pain did not resolve in 3, and abdominal pain did not resolve in 3.

Technique. Most of those patients who were managed with OR were treated by aneurysm resection with primary closure, followed by resection with patch angioplasty, ex vivo/complex repair, aneurysmectomy with bypass, resection with primary anastomosis, unplanned nephrectomy, and planned nephrectomy (Table IV). Among the EV aneurysms, treatment included stent graft placement and coil embolization.

Complications. Hospital length of stay was significantly shorter in the EV group (2 vs 8 days; $P < .001$). Although there was no significant difference in complications, there was a trend toward higher rates of minor perioperative complication after OR. Minor perioperative complications occurred in 19% of OR patients and in 4% of EV patients ($P = .071$) and included wound infection, urinary tract infection, ileus, urinary retention, minor renal infarct, renal insufficiency, and transient renal insufficiency not requiring dialysis. Major perioperative complications occurred in 8% of OR patients and in 2% of EV patients ($P = .344$) and included multisystem organ failure, myocardial infarction, and renal failure requiring dialysis. Late postoperative complications occurred in 9% of OR patients and in 8% of EV patients and included persistent abdominal abscess, stent graft stenosis, renal bypass thrombosis, renal artery thrombosis, and incisional hernia.

Only two patients suffered 30-day mortality: one patient died of intraoperative cardiopulmonary arrest and one patient died on postoperative day 2 from multisystem organ failure. One additional patient sustained a fatal myocardial infarction 90 days postoperatively. All patient deaths occurred in those patients aged >60 with multiple comorbidities.

Effect of repair on hypertension. Among the 76 patients (10%) who underwent RAA repair with difficult-to-control hypertension as the operative indication,

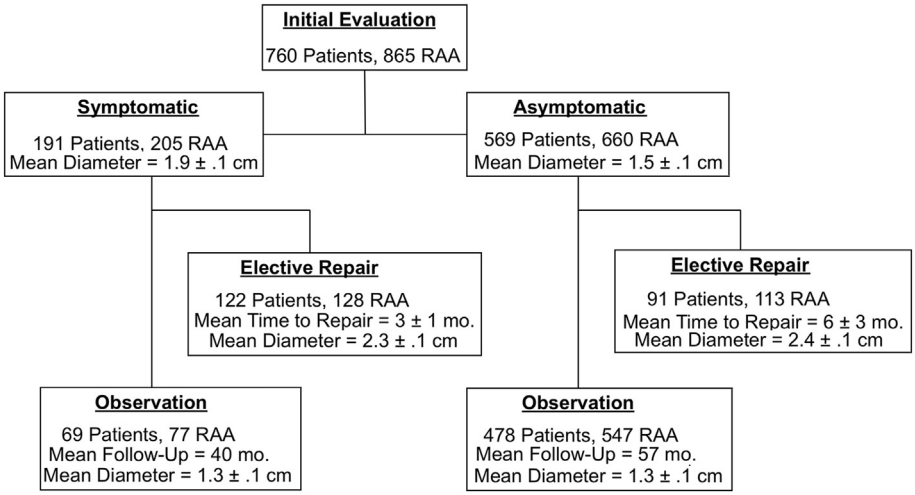

Fig 2. Management used for patients with renal artery aneurysms (*RAAs*).

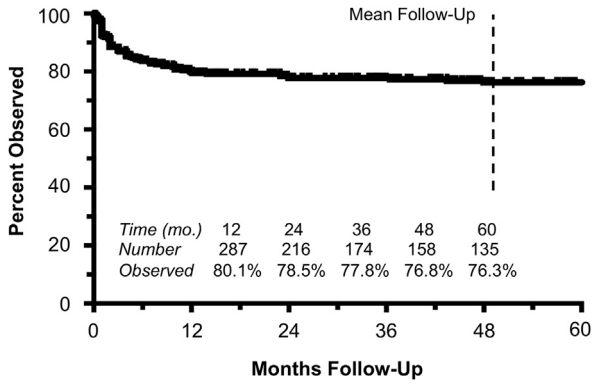

Fig 3. Freedom from repair in patients presenting with asymptomatic renal artery aneurysms (RAAs).

Table IV. Open repair (*OR*) and endovascular (*EV*) procedures performed to repair renal artery aneurysms (RAAs)

Technique	*Aneurysms (n = 241), No. (%)*	*Patients (n = 213), No. (%)*
OR		
Aneurysm resection with primary closure	60 (25)	57 (27)
Ex vivo/complex repair	29 (12)	23 (11)
Aneurysm resection with patch angioplasty	27 (11)	26 (12)
Aneurysmectomy with bypass	23 (10)	17 (8)
Resection with primary anastomosis	16 (7)	16 (8)
Unplanned nephrectomy	8 (3)	6 (3)
Planned nephrectomy	5 (2)	4 (2)
EV		
Stent graft	43 (18)	39 (18)
Coil embolization	30 (12)	25 (12)

hypertension was cured in 24 (32%; blood pressure <140/90 mm Hg off all antihypertensive medications), was improved in 20 (26%; decrease ≥15 mm Hg or decrease in the number of antihypertensive medications), and did not change in 32 (42%). The mean blood pressure of all hypertensive patients was not significantly different after surgery ($P = .604$). The average number of antihypertensive medications taken preoperatively and postoperatively (2.4 vs 2.2) was also not significantly different ($P = .433$).

Nonoperative management. Conservatively managed patients were observed for a mean of 49 ± 5 months, and no aneurysm ruptures occurred. Eighty-eight RAAs >2.0 cm were treated nonoperatively (mean diameter, 2.7 ± 0.1 cm) with a mean follow-up of 29 ± 5 months, also with no ruptures. No patient under observation developed acute renal artery complications (thrombosis or embolization). Serial imaging was performed in 83% of patients whose aneurysms were not surgically repaired, with a mean time of 9 ± 2 months between imaging studies; of these, 79% were monitored by CT, 14% by ultrasound imaging, and 7% by magnetic resonance imaging. The imaging modality used at diagnosis was the same as that used for serial imaging in 91% of patients.

There were three patients with Ehlers-Danlos syndrome (maximum aneurysm diameter, 1.1, 1.2, and 2.3 cm), two with Marfan syndrome (maximum aneurysm diameter, 1.1 and 2.4 cm), and one patient with a suspected connective tissue disorder (maximum aneurysm diameter, 2.1 cm). All of these patients were managed conservatively, and no acute complications developed. The mean aneurysm growth rate was 0.055 cm/y, which was not significantly different than the growth rate of other observed aneurysms ($P = .758$).

Growth rate. The overall growth rate, calculated from 454 aneurysms with two or more serial studies using the same imaging technique, was 0.086 ± 0.08 cm/y. There was no significant difference in growth rate between

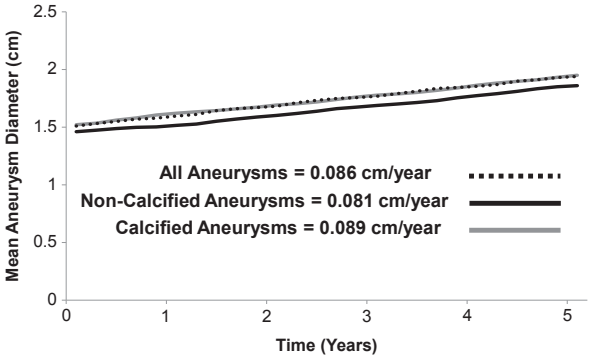

Fig 4. Growth rate of renal artery aneurysms (RAAs) managed with observation.

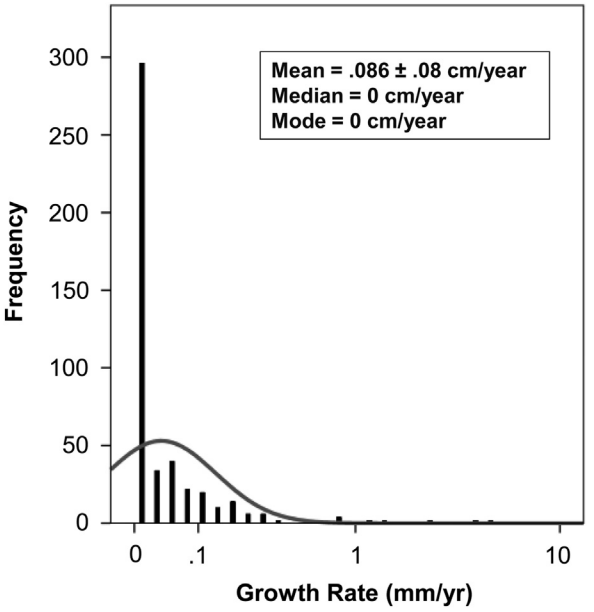

Fig 5. Distribution of renal artery aneurysm (RAA) growth rate in patients managed by observation.

calcified and noncalcified RAAs ($P = .784$; Fig 4). Most RAAs (293 aneurysms) did not grow, making the median and mode growth rate 0 cm/y (Fig 5). Aneurysms >2 cm that were observed showed a growth rate of 0.2 cm/y over a mean of 2.1 years, which was not significantly different from aneurysms ≤2 cm ($P = .083$). There was no difference in growth rate based on aneurysm morphology or calcification.

Ruptured RAAs. No database participant reported that a RAA rupture occurred at their hospital during the 10-year period. Three ruptured RAAs were identified, for an overall rupture rate of 0.3%. All three patients were referred to a participating study center from another hospital, and all survived. The first patient presented with free rupture, underwent a nephrectomy, and was transferred to the participating research center for a higher level of care. The second patient presented with a contained rupture and was transferred to the participating care center for coil embolization. The third patient had a prior contained rupture, coil embolization had failed, and presented to a tertiary care center for definitive management. All ruptured RAAs were >3 cm (mean diameter, 3.7 ± 0.2 cm). Among RAAs >3 cm, the rate of rupture was 18% (3 of 17 RAA). One of the ruptured aneurysms occurred in a patient with a suspected connective tissue disorder.

DISCUSSION

This study reports a very large series of RAAs identified in patients and was conducted over the last 10 years, during a time when cross-sectional abdominal imaging (CT angiography/magnetic resonance angiography) was routinely used for the diagnosis of many abdominal diseases and when OR and EV techniques were both available for treatment. The conclusions of this study are that rupture of asymptomatic RAAs is exceedingly rare, growth rate of RAAs is very slow, and OR is associated with significant minor morbidity but rarely with major morbidity or mortality.

The natural history of RAAs is not well defined due to their low frequency; however, that repair of RAA should be performed in symptomatic patients and in women of childbearing age is widely accepted because of the risk and high mortality of rupture during pregnancy.[9-11] Most studies, including the largest previous experience of RAA from the University of Michigan, as well as most textbooks, support the surgical treatment of all RAAs >2 cm, regardless of symptoms.[4,6] However, in the current study, and reflective of the continuing controversy about treatment criteria, 88 aneurysms between 2 and 3 cm at 13 different institutions were not surgically repaired, and no acute complications were reported, suggesting that conservative management of some asymptomatic RAAs between 2 and 3 cm may be safe.

Early studies estimated the risk for rupture of RAAs to be as high as 14%, but most authors have reported a rupture rate of ~3%.[1,3,4,10,12-16] As early as 1983, Tham et al reported an extremely low risk of rupture of RAAs, with no ruptures in 83 aneurysms monitored for a mean of 4.3 years, and many series have validated this benign natural history.[1,7,13,17,18] The results from the present study, with three ruptures in 865 aneurysms, including 88 aneurysms >2 cm and seven aneurysms >3 cm, further confirm that RAA rupture is an exceedingly rare event, with a frequency much less than 3%.

The study that has reported growth rates for RAAs used 14 aneurysms to calculate a growth rate of 0.060 cm/y.[7] The current study used 454 aneurysms from 16 different institutions and calculated a growth rate of 0.086 cm/yr. On the basis of this calculated growth rate, 46% of the asymptomatic aneurysms in this study would not require surgical repair in the next 10 years if the size threshold for asymptomatic repair were increased to 3 cm.

Some studies have suggested a beneficial effect of RAA repair on hypertension, whereas others have not.[7,19-25] The relationship between RAA and hypertension is not fully

elucidated, but the University of Michigan experience reported that surgical repair reduced blood pressure and the use of antihypertensive medications. It did not, however, establish a mechanical explanation, such as renal artery kinking, embolization, or stenosis, for the improvement.[7] Consistent with the University of Michigan's report, ~50% of the patients with difficult-to-control hypertension in the previously reported University of California, Los Angeles series and this series with difficult-to-control hypertension were cured or improved after operative repair.[7]

When performed by well-trained surgeons, OR of RAAs has been reported to be associated with a low major morbidity and mortality, and EV repair of RAAs has emerged as an alternative to OR in patients who are considered to be poor surgical candidates.[6,7,23,26-28] Most reports of EV repair of RAAs have been small series because many RAAs are not located in an optimal site for EV repair, but with careful selection, good outcomes have been obtained with stenting and coil embolization.[18,29-33] The location of the aneurysm is critical for an EV approach to be successful; stent grafting is currently limited to the main renal artery, where no branches are involved with the aneurysm.

One of the main limitations of this study is its retrospective design, which precludes capture of all RAAs, so the true natural history of RAAs remains unknown because only outcomes of patients who did undergo aneurysm repair could be analyzed. Although most patients had an imaging study to evaluate their aneurysm, the modality used was not standardized, so recommendations about optimal imaging are limited to the observation that measurement of aneurysm growth can be obtained with CT angiogram or MR angiogram and that serial growth rates and aneurysm size can be determined and may be used as criteria for repair.

Despite the large number of patients, because our study included only three patients with rupture, all referred from other hospitals, we were unable to determine specific risk factors for RAA rupture. To conclusively determine the risk of rupture, a prospective trial would be required that compares surveillance and repair for RAAs between 2 and 3 cm.

CONCLUSIONS

This large, contemporary, multi-institutional study demonstrated that asymptomatic RAAs rarely rupture, even when >2 cm and noncalcified, that the RAA growth rate is ~0.09 cm/y, that calcification does not protect against growth, that OR is associated with significant minor morbidity but rarely a major morbidity or mortality, and that aneurysm repair cured or improved hypertension in >50% of patients whose RAA was identified during the workup for difficult-to-control hypertension. This study questions current size criteria for repair of asymptomatic RAAs at 2 cm and supports the development of updated practice guidelines, because current guidelines recommending repair to prevent rupture for asymptomatic RAAs measuring >2 cm may be too aggressive.

We believe that RAA repair should be considered for asymptomatic RAAs >3 cm, those that demonstrate rapid growth, and those identified in women of childbearing age. Repair should continue to be offered to those patients with symptomatic RAAs, including those with medically refractory hypertension.

On behalf of the Vascular Low-Frequency Disease Consortium, we would like to recognize the contributions of the following additional collaborators: Nathan K. Itoga, MD, and Matthew W. Mell, MD, Stanford University, Stanford, Calif; Audra A. Duncan, MD, and Gustavo S. Oderich, MD, Mayo Clinic, Rochester, Minn; Adnan Z. Rizvi, MD, Abbott Northwestern Hospital, Minneapolis, Minn; Tazo Inui, MD, University of California San Diego, La Jolla, Calif; Robert J. Hye, MD, Kaiser Permanente, San Diego, Calif; Peter Pak, MD, Christopher Lee, BS, and Neal S. Cayne, MD, New York University, New York, NY; Jacob Loeffler, MD, and Misty D. Humphries, MD, University of California, Davis, Davis, Calif; Christopher Abularrage, MD, Johns Hopkins Hospital, Baltimore, Md; Paul G. Bove, MD, Beaumont Hospital, Royal Oak, Mich; Robert J. Feezor, MD, University of Florida, Gainesville, Fla; Amir F. Azarbal, MD, Oregon Health and Science University, Portland, Ore; Matthew R. Smeds, MD, University of Arkansas, Little Rock, Ark; Joseph M. Ladowski, BS, and Joseph S. Ladowski, MD, Indianapolis, Ind; Vivian M. Leung, MD, and York N. Hsiang, MD, University of British Columbia, Vancouver, BC, Canada; Josefina Dominguez, MD, and Fred A. Weaver, MD, University of Southern California, Los Angeles, Calif; and Mark D. Morasch, MD, St. Vincent Heart and Vascular, Billings, Mont.

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Writing the article: JK, PL, MH
Critical revision of the article: JK, PL, MH, DC, JS, NF
Final approval of the article: JK, PL, MH, DC, JS, NF
Statistical analysis: MH
Obtained funding: Not applicable
Overall responsibility: PL

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Submitted Sep 2, 2014; accepted Oct 29, 2014.

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

DISCUSSION

Dr John Blebea (*Tulsa, Okla*). I believe that your recommendation is rather aggressive, considering that this is a retrospective study and, as such, suffers by the inherent limitations. I have two questions. Firstly, how good was the follow-up of these patients? Since this is a retrospective study from many institutions, are you comfortable that the follow-up was sufficient and not many patients were lost who may have gone elsewhere either for later repair or rupture? Secondly, if you are convinced enough by these data to make the strong recommendation that in the future interventions in asymptomatic patients should wait until aneurysms are >3 cm, then why can't you make the same recommendations for symptomatic patients? You presented data for a relatively large group of untreated symptomatic patients, and none of those ruptured, so can't you make the same conclusion for symptomatic patients as well?

Dr Dawn M. Coleman. Thank you for those provocative questions. I'll freely acknowledge the limitations intrinsic to this retrospective study. I will suggest to you that I think the follow-up by institution was really quite good and that almost 80% of our patients had two to three serial imaging studies over a period of several years. And so while it is not a prospective study with a recognized limited follow-up, I think we at least have a good understanding that the growth rate is quite slow to absent, and I do feel very comfortable that the follow-up is appropriate to extrapolate recommendations from.

As far as expanding that recommendation to symptomatic patients, I am reluctant to draw the same strong conclusion. We included poorly controlled or truly medically refractory hypertension as a symptom, and I think that this symptom warrants treatment in select cases as over half of these patients with hypertension benefit from an operative intervention and that is why I think specifically these two patient cohorts should be looked at very differently.

Dr Steven Posner (*Anaheim, Calif*). Regarding improvements in blood pressure, did you break that down by endovascular vs open repair?

Dr Coleman. No, I apologize, I don't think that that was queried specifically. I don't have those data.

Dr Manish Mehta (*Albany, NY*). Nice work, great presentation. In your study, ~15% of the cohort had nonsaccular aneurysms. Did you have the opportunity to evaluate saccular vs nonsaccular aneurysm groups separately?

Dr Coleman. Aneurysm location and some of these anatomic features were queried as far as looking at indicators of progression and growth, for example, and nothing seemed to be associated at least with aneurysm growth. I hope that this answers your question.

Supplementary Table (online only). Participating institutions and the number of patients contributed

Institution	*Patients contributed (N = 760), No.*
St. Vincent Heart and Vascular, Mont	1
University of British Columbia, BC	9
University of Southern California, Calif	9
St. Joseph Hospital, Ind	13
University of Arkansas, Ark	16
Oregon Health Sciences University, Ore	21
Beaumont Hospital, Mich	24
University of Florida, Fla	26
Johns Hopkins Hospital, Md	27
University of California Davis, Calif	37
University of California Los Angeles, Calif	43
New York University, NY	69
Kaiser Permanente, Calif	90
Mayo Clinic, Minn	114
University of Michigan, Mich	127
Stanford University, Calif	134