

Fig. CAS, Carotid artery stenting; ECA, external carotid artery; ISR, in-stent restenosis.

identified from our institutional carotid database. There were a total of 312 CAS procedures (n = 299 patients) and 344 CEA procedures (n = 335) in this time period. For patients who had reoperation on the same carotid vessel (n = 5 for CAS, n = 5 for CEA), we used the last documented ultrasound prior to the date of reoperation. These ultrasounds were used to identify ECA occlusions and in-stent restenosis using consensus panel velocity criteria.

**Results:** There were 210 CAS patients with follow-up ultrasounds (67%), and there were 207 CEA patients with follow-up ultrasounds in our system (60%). The average follow-up of CAS was significantly shorter than the CEA group (0.7 vs 1.7 years;  $P < .001$ ), and CAS patients were more likely to take Plavix (97% vs 35%;  $P < .001$ ). All other variables were similar between groups. We identified significantly more occluded ECA in the CAS (14) compared with the CEA (4) group ( $P = .03$ ). Additionally, eight (57%) of 14 arteries with ECA occlusions had >50% ISR, whereas 48 (26%) of 186 arteries without ECA occlusions had >50% ISR ( $P = .02$ ) (Fig).

**Conclusions:** This is the first demonstration of increased ECA occlusion after CAS in the literature, but prior publications have identified increased external carotid stenosis. This finding was in spite of decreased follow-up time periods and increased use of Plavix in the CAS group, lending more validity to our results. A recent report identified disturbed flow in the ECA after CAS. This may be the biologic reason for these findings. The association of in-stent restenosis with ECA occlusion is also very interesting and warrants further investigation. We are currently determining whether ECA stenosis/occlusion occurs before, at the same time, or after in-stent restenosis. Establishing the timeline will be critical to identifying whether disturbed flow in the ECA has a causative role in in-stent restenosis.

**Anatomic Characteristics and Natural History of Renal Artery Aneurysms During Longitudinal Imaging Surveillance**

Erik Wayne, Matthew Edwards, Jeanette Stafford, Kimberley Hansen, Matthew Corriere. Wake Forest Baptist Medical Center, Winston-Salem, NC

**Objectives:** Renal artery aneurysms (RAA) are uncommon and rates of growth and/or rupture are unknown. Limited evidence, therefore, exists to guide clinical management of RAA, particularly those that are small and

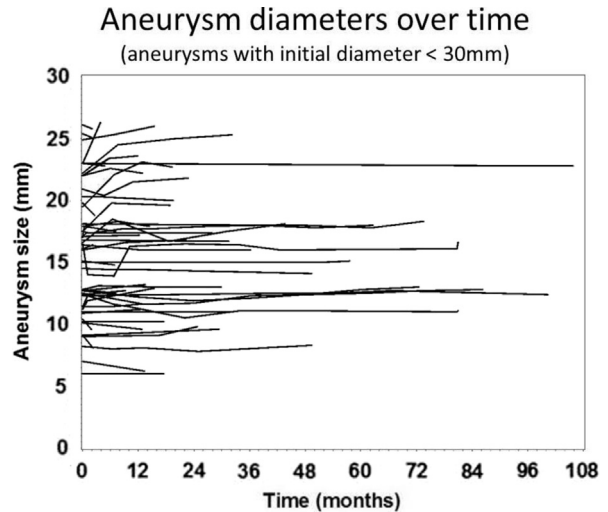


Fig.

asymptomatic. To further characterize the natural history of RAA, we studied anatomic characteristics and changes in diameter during imaging surveillance.

**Methods:** Patients evaluated for native RAA at a single institution over a three-year period (July 2010-July 2013) were identified and analyzed retrospectively. Patients with two or more cross-sectional imaging studies (computed tomography or magnetic resonance imaging) more than 1 month apart were included. Demographic and clinical data were collected from medical records and anatomic data (including aneurysm diameter, calcification, and location) were obtained from images. Aneurysm growth over time was analyzed using plots and Wilcoxon signed rank tests.

**Results:** Fifty-two aneurysms in 44 patients were analyzed. Median follow-up was 17.5 months (IQR, 8.1, 34.2 months); 70.5% of patients were female and mean age at initial presentation was  $60.4 \pm 9.5$  years; and 75.0% of patients had hypertension. Imaging studies demonstrated a 20.5% prevalence of nonrenal abdominal aneurysms and 18.2% had multiple RAA. Most RAA were located at the main renal artery bifurcation and had some degree of calcification. Mean initial aneurysm diameter was  $16.3 \pm 6.3$  mm. Median annualized growth rate was 0.03 mm (IQR, -0.16, 0.42 mm) ( $P = .34$ ). 13.5% of RAA were repaired electively and no RAA ruptures occurred.

**Conclusions:** Risk of short-term RAA growth or rupture was low. These findings suggest that annual (or less frequent) imaging surveillance is safe in the majority of patients and do not support pre-emptive repair of asymptomatic, small diameter RAA.

**EVAR Conversion for Type 1a Endoleak is not associated with Increased Morbidity or Mortality Compared with Primary Juxtarenal Aneurysm Repair**

Michael M. McNally, Salvatore T. Scali, Robert J. Feezor, Catherine K. Chang, Alyson L. Waterman, Scott A. Berceci, Thomas S. Huber, Adam W. Beck. University of Florida, Gainesville, Fla

**Objectives:** Due to concerns of increased morbidity and mortality with open surgical conversion (OSC) to manage type 1a endoleaks after endovascular aortic repair (EVAR), reports of endoluminal salvage with fenestrated and/or chimney techniques have emerged. The purpose of this analysis was to determine outcomes of elective OSC and compare with primary juxtarenal open aortic aneurysm repair (POR).

**Methods:** From 2002 to 2012, a total of 54 patients underwent EVAR conversion at median time of 26.8 months (interquartile range, 9.4, 54.6). Indications included endograft thrombosis (n = 2, 3.8%), intraoperative EVAR failure (n = 3, 5.5%), rupture (n = 5, 9.2%), graft infection (n = 6, 11.1%), and endoleak (n = 38, 70.4%). Twenty-five patients underwent elective OSC for type 1a endoleak and were compared with 25 anatomy- and comorbidity-matched patients who underwent POR during the same time period. Primary end points