General Health Physicians, Lancaster, Pa; [3]Department of Biostatistics, University of Nebraska Medical Center, Omaha, Neb; [4]Experimental Immunology Laboratory, Veterans Affairs Nebraska-Western Iowa Health Care System (VA NWIHCS), Omaha, Neb; [5]Experimental Immunology Laboratory, University of Nebraska Medical Center, Department of Internal Medicine, Division of Rheumatology, Omaha, Neb; [6]Experimental Immunology, Research in Cardiovascular Disease Laboratory at the University of Nebraska Medical Center, Department of Internal Medicine, Division of Cardiology, Omaha, Neb

Objectives: Abdominal aortic aneurysm (AAA) is a pathologic dilation of the aorta. Inflammation of the aortic wall has been shown to be involved in AAA formation. Malondialdehyde-acetaldehyde (MAA) adducts are MAA/protein hybrids with immunogenic, proinflammatory, and profibrotic properties. Levels of MAA adducts are elevated in patients with coronary artery disease; however, the role of MAA adducts in AAA is unclear. We hypothesize that levels of circulating antibodies against MAA adducts are increased in patients with AAA.

Methods: Plasma samples were collected from mice and patients with and without AAA. AAA was induced in mice by a standard $CaCl_2$ protocol, with matching sham mice. Plasma levels of anti-MAA antibodies were quantified by enzyme-linked immunosorbent assay.

Results: Patients with AAA exhibited higher levels of immunoglobulin G (IgG) and IgA anti-MAA antibody subtypes ($P = .049$ and $P = .026$, respectively) compared with control patients. Conversely, IgM anti-MAA antibodies in AAA patients were lower compared with control patients ($P = .018$). In $CaCl_2$ treated mice IgG anti-MAA antibodies were elevated after AAA formation ($P = .006$).

Conclusions: The pattern of anti-MAA antibodies is able to distinguish between patients with AAA and patients with atherosclerosis but no AAA. These results demonstrate that MAA adducts are associated with AAA and suggest they may play a role in either initiating or propagating chronic inflammation in AAA.

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S2: SVS Plenary Session II

SS6.

Natural History of Medically-Managed Acute Type B Aortic Dissections

Christopher A. Durham, Linda Wang, Emel A. Ergul, Nathan J. Aranson, Virendra I. Patel, Richard Cambria, Mark F. Conrad. Massachusetts General Hospital, Boston, Mass

Objectives: Although medical management of uncomplicated acute type B aortic dissections remains the standard of care, contemporary data regarding the natural history of medically treated patients are sparse. The goal of this study was to evaluate the ability of medical therapy to prevent long-term complications in patients with acute type B aortic dissection.

Methods: All patients with acute uncomplicated Type B aortic dissection that were initially managed medically between March 1999 and March 2011 were included. Failure of medical therapy was defined as any death or aortic-related intervention. Early failure occurred ≤15 days of presentation and late failure occurred thereafter. Predictors of long-term outcomes were determined using Cox proportional hazards models.

Results: A total of 298 patients (61.7%) with medically managed acute type B dissections were identified. The cohort was an average age of 65.9 years at presentation. There were 37 early failures (12%) including 12 deaths and 25 interventions (10 thoracic endovascular aortic repair [TEVAR]/15 open). Aneurysmal degeneration was the indication for intervention in six (24%). Mean follow-up was 4.2 years (range, 0.1-14.7 years). There were 174 failures (58.4%), including 87 deaths and 87 interventions (24 TEVAR/63 open). Fifty-seven interventions (66%) were for aneurysmal degeneration. Freedom from intervention was 77.3% ± 0.024% at 3 years and 74.2% ± 0.025% at 6 years. There were no predictors of freedom from intervention. The intervention-free survival was 55.0% ± 0.030% at 3 years and 41.0% ± 0.032% at 6 years. Age >70 years was protective against failure (hazard ratio, 0.97; confidence interval, 0.95-0.98, $P < .01$). Survival was higher in patients who required intervention at both 3 years (78% vs 73%) and 6 years (76% vs 58%; $P = .018$).

Conclusions: Medical therapy of acute uncomplicated type B dissections is successful in the short-term. However, the overall 6-year intervention-free survival is low, and survival is significantly higher in patients who undergo intervention.

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SS7.

The Contemporary Guidelines for Asymptomatic Renal Artery Aneurysms Are Too Aggressive: A North American Experience

Peter F. Lawrence[1], Jill Q. Klausner[1], Michael P. Harlander-Locke[1], Dawn M. Coleman[2], James C. Stanley[2], Gustavo S. Oderich[3], Tazo S. Inui[4], Matthew W. Mell[5], Misty Humphries[6], Paul G. Bove[7], Christopher J. Abularrage[8], Robert J. Feezor[9], Amir F. Azarbal[10], Matthew R. Smeds[11], Joseph S. Ladowski[12], York N. Hsiang[13], Josefina A. Dominguez[14], Mark D. Morasch[15], for the Vascular Low-Frequency Disease Consortium[1]. [1]Division of Vascular Surgery, University of California Los Angeles, Los Angeles, Calif; [2]Department of Surgery, Section of Vascular Surgery, University of Michigan, Ann Arbor, Mich; [3]Division of Vascular and Endovascular Surgery, Mayo Clinic, Rochester, Minn; [4]Kaiser Permanente, San Diego, Calif; [5]Division of Vascular Surgery, Stanford University School of Medicine,

Stanford, Calif; [6]Division of Vascular and Endovascular Surgery, University of California Davis Health System, Sacramento, Calif; [7]Division of Vascular Surgery, Department of Surgery, William Beaumont Hospital, Royal Oak, Mich; [8]Division of Vascular Surgery, Johns Hopkins Hospital, Baltimore, Md; [9]Division of Vascular Surgery and Endovascular Therapy, University of Florida College of Medicine, Gainesville, Fla; [10]Division of Vascular Surgery, Department of Surgery, Oregon Health and Science University, Portland, Ore; [11]Division of Vascular and Endovascular Surgery, University of Arkansas for Medical Sciences, Little Rock, Ark; [12]The Cardiothoracic and Vascular Surgeons and Nurses of Lutheran Health Network, Fort Wayne, Ind; [13]Division of Vascular Surgery, University of British Columbia, Vancouver, BC, Canada; [14]Division of Vascular Surgery and Endovascular Therapy, Keck Medical Center, University of Southern California, Los Angeles, Calif; [15]St. Vincent Heart and Vascular, Billings, Mont

Objectives: Most prior single-center series have recommended repair of asymptomatic renal artery aneurysms (RAA) >2 cm. This study evaluated the contemporary management of a large series of RAAs.

Methods: Patients with RAAs were analyzed using a standardized database by a research consortium of 15 institutions.

Results: A total of 614 RAAs were identified in 525 patients at 15 institutions (age, 61; male-to-female ratio = 1:2). Seventy-one percent of patients were asymptomatic. Symptomatic patients had severe hypertension (12%), flank pain (7%), abdominal pain (6%), and hematuria (4%). Aneurysm location included the main renal artery bifurcation (40%), main trunk (28%), primary branch (18%), secondary branch (7%), and pole artery (7%). Most RAAs were saccular (86%) and calcified (64%). Diameters were 1.8 ± 0.1 cm for symptomatic RAAs and 1.5 ± 0.1 cm for asymptomatic RAAs ($P < .001$). Aneurysms were observed (67%; diameter 1.3 ± .1 cm) or surgically treated with open repair (OR; 28%; diameter 2.1 ± .1 cm), or endovascularly (EV; 5%; diameter 2.3 ± .2 cm). For OR vs EV, minor complications were 26% and 10%, respectively ($P < .001$), and major complications were 1% and 3%, respectively ($P = .014$). Only one death occurred, in an EV patient. Conservatively managed patients were observed for 41 ± 4 months with no ruptures. Fifty-nine RAA >2 cm were treated non-OR (diameter 2.8 ± .1 cm) with a mean follow-up time of 42 ± 10 months. There were no ruptures. The growth rate for asymptomatic RAA, based on serial imaging, was 0.16 ± 0.01 cm/y (calcified = 0.16 ± 0.01 cm/y; noncalcified = 0.17 ± 0.01 cm/y; $P = .913$).

Conclusions: This largest study of RAA demonstrates that (1) asymptomatic RAA rarely rupture, even when >2 cm and not calcified, (2) open repair is associated with significant minor morbidity but rarely a major morbidity or mortality, (3) RAA growth rate is 0.16 ± 0.01 cm/y and calcification does not protect against growth, (4) the current guideline of repairing asymptomatic RAA >2 cm is too aggressive.

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SS8.

Contemporary Outcomes of Intact (iVAA) and Ruptured (rVAA) Visceral Artery Aneurysm Repair

Ankur J. Shukla, Raymond Eid, Larry Fish, Efthimios Avgerinos, Luke Marone, Michel Makaroun, Rabih Chaer. UPMC, Pittsburgh, Pa

Objectives: To review the outcomes of open and endovascular intervention for intact visceral artery aneurysm (iVAA) and ruptured VAA (rVAA).

Methods: Retrospective review of treated VAA at one institution from 2003 to 2013.

Results: We identified 261patients with VAA; of these 155 were repaired (69 ruptured, 86 intact; Table). Pseudoaneurysms were more common in rVAA (80%) vs iVAA (35%; $P < .001$). rVAA were smaller than iVAA (20.5 vs 27.5 mm; $P = .018$) at repair, and their most common presentation was abdominal pain; 18% were hemodynamically unstable. Endovascular intervention was the initial treatment for 70% (78% for rVAA, 63% for iVAA). Perioperative complication rate was higher for rVAA (19% vs iVAA 4%; $P = .003$), as well as 30-day (12% vs 0% iVAA; $P = .001$), 1-year (26% vs 4% iVAA; $P < .001$), and 3-year (30% vs 7% iVAA; $P < .001$) mortality. Lower 30-day mortality was noted with endovascular repair for rVAA (7% vs 28% open; $P = .06$). Predictors of mortality for rVAA included age (HR, 1.098; $P = .006$) whereas endovascular repair was protective (HR, 0.231; $P = .035$). Mean follow up was 26 months, and Kaplan-Meier estimates of survival were higher for iVAA at 3 years (88% vs 62% iVAA, $P = .045$). The 30-day reintervention rate was higher for rVAA (9% vs 1% iVAA; $P = .045$), but was similar between open and endovascular repair (8.5% vs 15%, P = NS).

Table.

Ruptured VAA (N = 69)	*No. (%)*
Gender (Male)	43 (62.3)
Age (mean)±SD	5S.8 ± 15
Smoking	16 (23.2)
VAA type (pseudoaneurysm)	55 (79.7)
Mean size (mm) (range)	20.5 (3.5-75)
Splenic	19 (27.5)
Celiac	6 (8.7)
SMA	8 (11.6)
Hepatic	16 (23.2)
Renal	1 (1.5)
Pancreaticoduodenal (PDA)	18 (26.1)
Comorbidities	
DM	8 (11.6)
CRI	6 (8.7)
ESRD	1 (1.4)
Hypertension	41 (59.4)
Hyperlipidemia	13 (18.8)
CAD	4 (5.9)
COPD	7 (10.1)
CVA	5 (7.2)
Pancreatitis	29 (42)