Enrico Cieri[1], Paola De Rango[1], Giacomo Isernia[1], Gioele Simonte[1], Andrea Ciucci[1], Gianbattista Parlani[1], Fabio Verzini[1], Piergiorgio Cao[2]. [1]Vascular and Endovascular Surgery; University of Perugia, Perugia, Italy; [2]Unit of Vascular Surgery, Hospital S. Camillo Forlanini, Rome, Italy

Objectives: To analyze predictors and long-term consequence of type II endoleak in a large series of elective EVAR.

Methods: Baseline characteristics, operative and follow-up data of consecutive patients undergoing EVAR were prospectively collected. Patients who developed type II endoleak according to CT scan and those without were compared for baseline characteristic, mortality, reintervention, conversion and aneurysm growth after repair.

Results: 1412 consecutive patients (91.4% males; mean age, 72.9) underwent elective EVAR in 1997-2012 and were subsequently followed for a mean of 54 ± 42 months. 218 developed type II endoleak. Multivariate analysis failed to identify type II endoleak significant independent predictors with the exception of age ($P = .026$; OR, 1.023) and neck thrombus ($P = .011$; OR, .303). Rates of type II endoleak were comparable regardless the type of device but there was a tendency for lower rate with most recent generation devices. Freedom from aneurysm sac growth >5 mm (95.2% vs 42.4%; $P < .001$) reintervention (92.3% vs 49.6%; $P < .001$) or conversion (98.5% vs 80%; $P < .001$) at 120 months was higher in patients without type II endoleak.

Fifty-two patients with type II endoleak underwent reintervention. At 60 months, rates of persisting type II endoleak were similar among patients with and without reinterventions (49.8% vs 45.6%). There were no significant difference in persisting aneurysm growth >5 mm in type II endoleak patients after reintervention and those who remained untreated (57.1% vs 42.6%).

Cox regression identified type II endoleak as independent predictor of aneurysm growth along with age and cardiac disease.

There were four aneurysm ruptures during follow-up in patients with type II endoleak. Late aneurysm related mortality at 120 months was 3.8% vs 2.1% for patients with and without type II endoleak.

Conclusions: Type II endoleak is a common marker of EVAR failure reflecting multiple meanings. Occurrence and consequences are challenging to be predicted and treatment with reinterventions often results in failure.

Author Disclosures: P. Cao: Nothing to disclose; **E. Cieri**: Nothing to disclose; **A. Ciucci**: Nothing to disclose; **P. De Rango**: Nothing to disclose; **G. Isernia**: Nothing to disclose; **G. Parlani**: Nothing to disclose; **G. Simonte**: Nothing to disclose; **F. Verzini**: Nothing to disclose.

RR11.

Use of Cryopreserved Aortoiliac Allograft (CAA) for Aortic Reconstruction in the United States

Michael P. Harlander-Locke[1], Peter F. Lawrence[1], Gustavo S. Oderich[2], Liv Harmon[1], Robert A. McCready[3], Mark D. Morasch[4], Robert J. Feezor[5], Wei Zhou[6], Jean Bismuth[7], William C. Pevec[8], Mateus P. Correa[2], Jeffrey Jim[9], Joseph S. Ladowski[10], Panagiotis Kougias[11], Paul G. Bove[12], Catherine M. Wittgen[13], John V. White[14]. [1]Vascular Surgery, University of California Los Angeles, Los Angeles, Calif; [2]Mayo Clinic, Rochester, Minn; [3]Indiana University, Indianapolis, Ind; [4]St. Vincent Heart and Vascular, Billings, Mont; [5]University of Florida, Gainesville, Fla; [6]Stanford University, Stanford, Calif; [7]Methodist Hospital, Houston, Tex; [8]University of California Davis, Davis, Calif; [9]Washington University, St. Louis, Mo; [10]Indiana Ohio Heart, Indianapolis, Ind; [11]Baylor University, Houston, Tex; [12]William Beaumont Hospital, Royal Oak, Mich; [13]Saint Louis University, St. Louis, Mo; [14]Advocate Lutheran General Hospital, Niles, Ill

Objectives: Aortic infections, even with treatment, have a high mortality and risk of recurrent infection and limb loss. CAA has been proposed for in-line reconstruction to improve outcomes in this high-risk population.

Methods: A multi-center study using a standardized database was performed at 14 of the highest volume institutions who used CAA for aortic infection.

Results: Two hundred and twenty patients (mean age = 65; M:F = 1.6/1) were treated since 2000 with 283 CAAs for prosthetic graft infection (59%), primary aortic infection (17%), enteric fistula/erosion (16%), mycotic aneurysm (4%), and other (4%). Intra-op cultures indicated infection in 66%, most frequently polymicrobial. Distal anastomosis was to the femoral artery, iliac, then distal aorta. 30-day mortality was 9% and procedure related major complications occurred in 24%, including persistent sepsis (n = 17), graft thrombosis (n = 9), graft/stump rupture (n = 8), recurrent CAA/aortic infection (n = 8), pseudoaneurysm (n = 6), recurrence of AE fistula (n = 4), and compartment syndrome (n = 1). Hospital LOS was 24 days. Ten (5%) required allograft explant; 2 developed CAA aneurysm requiring resection at 23 and 40 mo. Primary graft patency and freedom from limb loss were 93% and 97%, respectively, at 5 yr. Patient survival was 75% at 1 yr and 51% at 5 yr.

Conclusions: This largest study indicates that CAA allows in-line reconstruction of aortic infection with lower

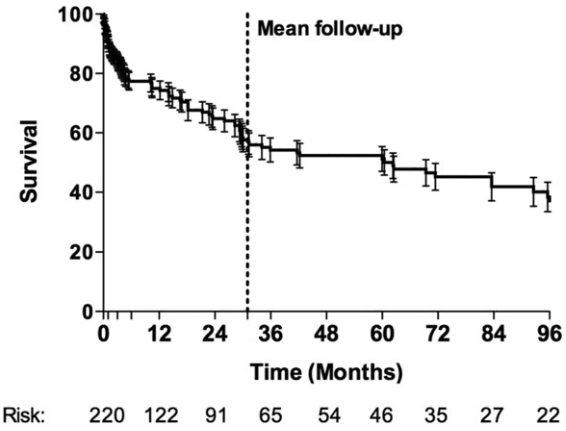

Fig.

early and long-term morbidity and mortality than other previously reported treatment options. Repair with CAA is associated with low rates of aneurysm formation, recurrent infection, and limb loss.

Author Disclosures: J. Bismuth: Nothing to disclose; **P. G. Bove**: Nothing to disclose; **M. P. Correa**: Nothing to disclose; **R. J. Feezor**: Nothing to disclose; **M. P. Harlander-Locke**: CryoLife, Research Grants; **L. Harmon**: Nothing to disclose; **J. Jim**: CryoLife, Research Grants; **P. Kougias**: Nothing to disclose; **J. S. Ladowski**: Nothing to disclose; **P. F. Lawrence**: CryoLife, Research Grants; **R. A. McCready**: Nothing to disclose; **M. D. Morasch**: Nothing to disclose; **G. S. Oderich**: Nothing to disclose; **W. C. Pevec**: Nothing to disclose; **J. V. White**: Nothing to disclose; **C. M. Wittgen**: Nothing to disclose; **W. Zhou**: Nothing to disclose.

RR12.

Familial AAA Is Associated With Increased Postoperative Adverse Events After EVAR

Koen M. van de Luijtgaarden[1], Frederico Bastos Goncalves[1], Sanne E. Hoeks[2], Ellen V. Rouwet[1], Danielle Majoor-Krakauer[3], Robert J. Stolker[2], Hence J. Verhagen[1]. [1]Erasmus MC-Department of Vascular Surgery, Rotterdam, The Netherlands; [2]Erasmus MC-Department of Anesthesiology, Rotterdam, The Netherlands; [3]Erasmus MC-Department of Clinical Genetics, Rotterdam, The Netherlands

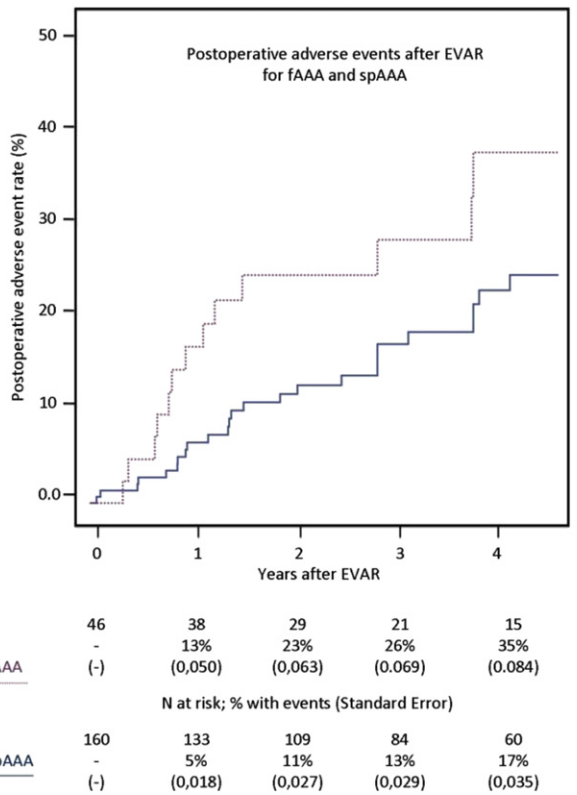

Fig.

Objectives: To investigate the risk of postoperative adverse events (PAE) in patients with familial AAA (fAAA) vs sporadic AAA (spAAA).

Methods: Patients were derived from a prospective database for EVAR. Family history was obtained by written questionnaire (93% response rate). fAAA patients were defined as having ≥1 affected 1st-degree relative, and excluding connective tissue disorders. Cardiovascular risk factors, AAA morphology (neck, sac and iliac), and follow-up (FU) information were scored. PAE was defined as a composite of secondary intervention, sac growth (>5 mm), and type I/III endoleak. PAE estimates were obtained from Kaplan-Meier plots and multivariable Cox-regression was used to explore the risk associated with fAAA.

Results: 207 patients were included (90% men; age 71 ± 8; FU 4.5 ± 3 yrs), with 46 (22%) classified as fAAA. Patients with fAAA were younger (68 vs 72 yrs; $P = .003$) and less likely smokers ($P = .056$). No difference was observed in AAA morphology. After EVAR, fAAA patients had significantly more PAE (Fig), with a 2-fold increase in risk (adjusted HR, 2.0; 95% CI, 1.1-3.8). Sac growth was observed in 20% of fAAA vs 9% of spAAA ($P = .005$), unrelated to presence of endoleak. There were no further differences in individual components of PAE, nor in overall survival.

Conclusions: Despite similar morphology, patients with fAAA had more PAE, mainly due to sac growth. Until the underlying cause is identified, patients with fAAA may need closer surveillance.

Author Disclosures: F. Bastos Goncalves: Nothing to disclose; **S. E. Hoeks**: Nothing to disclose; **D. Majoor-Krakauer**: Nothing to disclose; **E. V. Rouwet**: Nothing to disclose; **R. J. Stolker**: Nothing to disclose; **K. M. van de Luijtgaarden**: 'Lijf & Leven' foundation, Rotterdam, The Netherlands, Research Grants; **H. J. Verhagen**: Nothing to disclose.

RR13.

A Review of the Contemporary and Historical Management of 134 Patients With Splenic Artery Aneurysms

Dawn M. Coleman, Huiting Chen, Anna Eliassen, Shipra Arya, Enrique Criado, Jonathan L. Eliason, John Rectenwald, James C. Stanley. Vascular Surgery, University of Michigan, Ann Arbor, Mich

Objectives: This study aims to examine the shifting trend in the management of true splenic artery aneurysms (SAA) in an endovascular (EV) era.

Methods: A retrospective review of a single institution experience with SAA was performed. Medical records and imaging of 74 patients diagnosed with SAA between 1997 and 2012 were reviewed. This data was compared to a historical cohort of 60 patients managed before 1974.

Results: A female predominance of 80% was noted, of which 65% of women were multiparous and 13% reported grand-multiparity (≥6 pregnancies). Mean age at diagnosis was 56 years (range, 32-80). Mean aneurysm size at diagnosis was 2.0 cm (range, 0.8-3.5). 31 patients (41%) were followed conservatively and demonstrated no growth by surveillance imaging. 43 patients underwent surgical