

as the first Qa value obtained in a well functioning AVA by a two needle dilution technique, and a 1 000 mL/minute Qa cut off was used, as suggested by earlier studies. Actual Qa was defined as Qa at a random point of time. Changes in actual Qa were expressed per three month periods. CVM was assessed according to the European Renal Association and European Dialysis and Transplantation Association (ERA-EDTA) classification. Longitudinal associations between Qa characteristics and four year freedom from CVM were calculated using the joint modelling approach.

Results: A total of 5 208 Qa measurements in 165 patients (103 men; mean ± SD age 70 ± 12 years) were analysed. During follow up (December 2010 – January 2018, median 36 months), 79 patients (48%) died. An initial Qa < 1 000 mL/minute was associated with an increased four year CVM risk (hazard ratio [HR] 3.79, 95% confidence interval [CI] 1.78 – 8.10; *p* = .001). After four years, freedom from CVM was 30% lower in patients with a Qa < 1 000 (Qa < 1000 mL/minute 57% ± 7% vs. Qa ≥ 1 000 mL/minute 87% ± 4% [*p* < .001]). An association between increases in actual Qa over three month periods and mortality was found (HR 4.48 per 100 mL/minute, 95% CI 1.44 – 13.97; *p* = .010), indicating that patients demonstrating increasing Qa values were more likely to die. By contrast, actual Qa per se was not related to survival.

Conclusion: Studying novel vascular access flow characteristics may contribute to understanding excess cardiovascular mortality in patients on HD.

Figure 1. Initial access flow (>1000 mL/min (n=90), or <1000 mL/min (n=75) in relation to Freedom from CVM

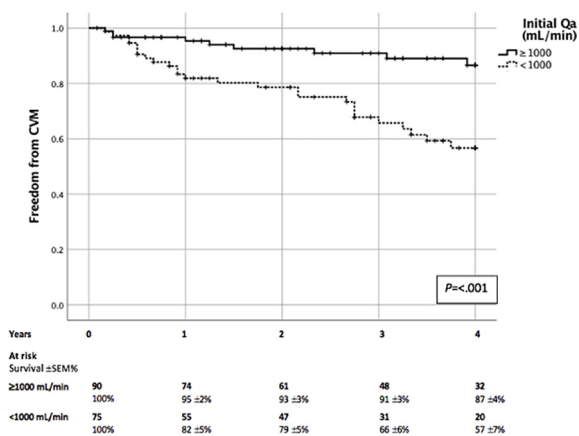
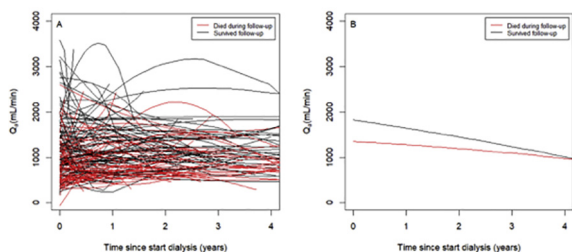


Figure 2. Access flow over time of individual patients (left) and entire study cohort (n=165, right). Curves of patients who survived (upper black line, n=86) and who died (lower red line, n=79) are significantly different (*P*=.005).



P-020

Long Term Endoleak Outcomes After Thoracic Endovascular Aortic Repair: A 25 Year Single Centre Experience

Nicola Leone*, Stefano Gennai, Luigi Alberto Maria Bartolotti, Mattia Migliari, Tea Covic, Francesca Rossi, Francesco Andreoli, Roberto Silingardi

Ospedale Civile Di Baggiovara, Azienda Ospedaliero-universitaria Di Modena, University of Modena and Reggio Emilia, Modena, Italy

Introduction: Since its introduction in 1994, thoracic endovascular aneurysm repair (TEVAR) has become standard practice in the management of a broad spectrum of descending thoracic aorta diseases. Despite several publications focusing on the short and mid-term outcomes of TEVAR, few analyses encompassing a multidecade time span are available. While the efficacy and peri-operative morbidity and mortality of this technique have been extensively investigated, data regarding long term survival and the endoleak rate are still limited.

Methods: This was a retrospective, observational, single centre cohort study of TEVAR patients between November 1995 and December 2020 (local ethics committee approval no. 507/2018). Missing mortality information (dates and causes) and eventual re-interventions performed at other centres were retrieved by the Department of Public Health. The primary endpoint of this study was freedom from endoleak in the four stent graft generations during the follow up period. No re-interventions for type II endoleak were registered; they were therefore excluded from the cumulative endoleak rate. The first generation (GEN1) included: Vanguard (Boston Scientific); AneuRx and Talent (Medtronic); Stentor (Mm Tec); Excluder (WL Gore); Endologix (Endologix); and EndoFit (LeMaitre). The second-generation (GEN2) included TAG (WL Gore) and TX (Cook Medical). The third (GEN3) included Relay Plus (Bolton Medical); Valiant Captivia (Medtronic); and Zenith Alpha and Custom-made (Cook Medical). The fourth (GEN4) included Relay Pro and custom made (Bolton Medical); Conformable C-TAG (WL Gore); Navion (Medtronic); Standard and custom made thoraco-abdominal devices (Cook Medical); Nexus (Endospan); E-Vita, Colt, and custom made thoraco-abdominal devices (Jotec); and Najuta (Kawasumi). Aorta related survival was considered whenever the cause of death was ascribable to the treated aortic segment.

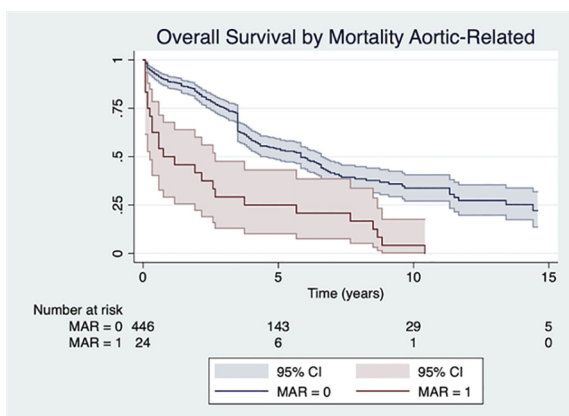
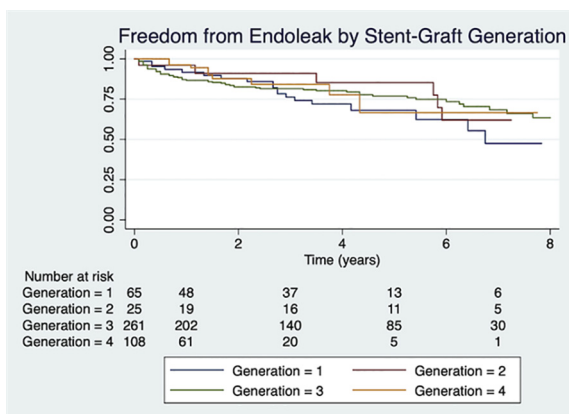
Results: A total of 509 stent grafts deployed into the thoracic aorta were included with a mean ± SD follow up of 44.3 ± 42.5 months. Early and late results are depicted in Table 2. Freedom from endoleak was 84.6%, 70.4%, 56.0%, and 51.5% at one, five, 10, and 15 years, respectively (Fig. 1). Freedom from endoleak at one year was 90.4%, 96.2%, 86.4%, and 96.6% for GEN1, GEN2, GEN3, and GEN4, respectively. Freedom from endoleak at five years was 65.6%, 61.4%, 76.2%, and 69.1% for GEN1, GEN2, GEN3, and GEN4, respectively (*p* = .37). The first two generations demonstrated a higher endoleak rate, compared to the two most recent generations (27.2 vs. 18.2%; *p* = .043). The proximal landing in zone 1 (odd ratio [OR] 2.2; *p* = .050) and GEN1 (OR 2.0; *p* = .014) was an independent risk factor for endoleak. Proximal landing in zone 5 (OR 0.2; *p* = .055) and GEN4 (OR 0.3; *p* = .001) was an independent protective factor for endoleak. GEN1 was found to be a risk factor for type Ia endoleak (OR 2.1; *p* = .059). Type Ib endoleak demonstrated a negative association with distal landing in zone 5 (OR 4.9; *p* = .001). Conversely, a

positive association was highlighted with distal landing in zone 4 (OR 0.2; $p = .036$) and GEN4 (OR 0.1; $p = .012$). Overall survival was 75.3%, 44.4%, 27.2%, and 17.6% at one, five, 10, and 15 years, respectively. Survival distinguished as non-related versus aortic related was 86.7% versus 23.5%, 52.7% versus 9.8%, 32.9% versus 2.0%, and 21.2% versus 0% at one, five, 10, and 15 years, respectively ($p < .000$; Fig. 2).

Conclusion: Potentially, half of patients could develop an endoleak at 15 years, highlighting the importance of life long radiological follow up after thoracic stent grafting. Newer stent graft generation demonstrated better long term endoleak results, despite an increasing indication of more challenging aortic morphology, as well as to extensive pathology (arch and/or thoraco-abdominal), during the 25 years analysed. This real world TEVAR study over a considerable time span yielded the conclusion that the older generation should be followed up strictly. Likewise, landing in the most extreme zones (e.g., proximally in zone 1 and distally in zone 5) required special attention owing to their higher tendency to leak. The efficacy of TEVAR was confirmed in preventing long term aortic mortality, with most deaths occurring within 30 days.

	All Generations	Generation 1	Generation 2	Generation 3	Generation 4	Statistical significance
	Count (%) or mean \pm SD	Count (%) or mean \pm SD	Count (%) or mean \pm SD	Count (%) or mean \pm SD	Count (%) or mean \pm SD	P-value
Total	N= 509	n= 71	n= 32	n= 293	n= 113	
Mean Follow-up, months	44.3 \pm 42.5	54.9 \pm 59.2	49.0 \pm 51.4	50.2 \pm 39.8	21.2 \pm 21.2	0.000
Age, years	69.3 \pm 12.4	68.8 \pm 9.3	71.6 \pm 7.8	68.1 \pm 14.1	72.3 \pm 9.7	0.012
Male	381 (74.9)	62 (87.3)	21 (65.6)	215 (73.4)	83 (73.5)	0.051
Smoker						0.000
No	239 (47.0)	33 (46.5)	14 (43.8)	165 (56.3)	27 (23.9)	
Active	113 (22.2)	11 (15.5)	9 (28.1)	59 (20.1)	34 (30.1)	
Former	157 (30.8)	27 (38.0)	9 (28.1)	69 (23.5)	52 (46.0)	
Hypertension	362 (71.1)	40 (56.3)	20 (62.5)	201 (68.6)	101 (89.4)	0.000
Hyperlipidemia	220 (43.3)	25 (35.2)	14 (45.2)	114 (38.9)	67 (59.3)	0.001
Cardiac disease	195 (38.3)	18 (25.4)	7 (21.9)	107 (36.5)	63 (55.8)	0.000
COPD	108 (21.2)	17 (23.9)	6 (18.8)	52 (17.8)	33 (29.2)	0.077
Diabetes	53 (10.4)	11 (15.5)	4 (12.5)	21 (7.2)	17 (15.0)	0.046
CKD	99 (19.5)	5 (7.0)	8 (25.0)	50 (17.1)	36 (31.9)	0.000
CVD	31 (6.1)	0 (0)	2 (6.3)	16 (5.5)	13 (11.5)	0.014
PAD	19 (3.7)	2 (2.8)	2 (6.3)	11 (3.8)	4 (3.5)	0.863
Etiology	N= 509	n= 71	n= 32	n= 293	n= 113	
Aneurysm	355 (69.7)	55 (77.5)	25 (78.1)	176 (60.1)	99 (87.6)	0.000
DTAA	247/355 (69.6)	50 (70.4)	20 (62.5)	154 (52.6)	23 (20.4)	0.005
TAAA	92/355 (25.9)	3 (4.2)	4 (12.5)	4 (1.4)	70 (61.9)	0.003
Anastomotic aneurysm	8/355 (2.2)	2 (2.8)	0 (0)	1 (0.3)	5 (4.4)	0.018
AEF	5/355 (1.4)	0 (0)	1 (3.1)	3 (1.0)	1 (0.9)	0.527
ABF	3/355 (0.8)	0 (0)	0 (0)	3 (1.0)	0 (0)	0.527
TBAD	124 (24.4)	13 (18.3)	5 (15.6)	92 (31.4)	14 (12.4)	0.000
BTAI	30 (5.9)	3 (4.2)	2 (6.3)	25 (8.5)	0 (0)	0.011
Aortic rupture (nontraumatic)	56 (11.7)	9 (12.7)	5 (15.6)	34 (11.6)	8 (7.1)	0.020

COPD, chronic obstructive pulmonary disease; CKD, chronic kidney disease; CVD, cerebrovascular disease; PAD, peripheral arterial disease; DTAA, descending thoracic aortic aneurysm; TAAA, thoraco-abdominal aneurysm disease; AEF, aorto-esophageal fistula; ABF, aorto-bronchial fistula; TBAD, type-B aortic dissection; BTAI, blunt traumatic aortic injury.



	All Generations	Generation 1	Generation 2	Generation 3	Generation 4	Statistical significance
	Count (%) or mean \pm SD	Count (%) or mean \pm SD	Count (%) or mean \pm SD	Count (%) or mean \pm SD	Count (%) or mean \pm SD	P-value
Early Results (<30-day)	N= 509	n= 71	n= 32	n= 293	n= 113	
Technical success	498 (97.8)	70 (98.6)	31 (96.9)	287 (98.0)	110 (97.3)	0.923
Technical failure	11 (2.2)	1 (1.4)	1 (3.1)	6 (2.0)	3 (2.3)	0.923
Intraoperative death	2 (18.2)	1 (100)	1 (100)	0 (0)	0 (0)	
Retrograde induced dissection/IMH	6 (54.5)	0 (0)	0 (0)	4 (66.7)	2 (66.7)	
Antegrade induced dissection/IMH	3 (27.3)	0 (0)	0 (0)	2 (33.3)	1 (33.3)	
Endoleak	8 (1.6)	1 (1.4)	0 (0)	5 (1.7)	2 (1.8)	0.900
Ib	5/8 (62.5)	1 (100)	0 (0)	4 (80.0)	0 (0)	
R	3/8 (37.5)	0 (0)	0 (0)	1 (20.0)	2 (100)	
Reinterventions	63 (12.4)	11 (15.5)	5 (15.6)	35 (12.0)	12 (10.6)	0.706
Access-related	44/63 (69.8)	10 (90.9)	4 (80)	22 (62.9)	8 (66.7)	
Endoleak	7/63 (11.1)	1 (9.1)	0 (0)	4 (11.4)	2 (16.7)	
Induced dissection/IMH	6/63 (9.5)	0 (0)	0 (0)	4 (11.4)	2 (16.7)	
Miscellaneous	6/63 (9.5)	0 (0)	1 (20)	5 (14.3)	0 (0)	
SCI	16 (3.1)	2 (2.8)	0 (0)	10 (3.4)	4 (3.5)	0.755
Stroke	7 (1.4)	2 (2.9)	0 (0)	4 (1.4)	1 (0.9)	0.620
Mortality (30-d or in-hospital)	39 (7.7)	4 (5.6)	6 (18.7)	23 (7.8)	6 (5.3)	0.076
Late Results (>30-day)	N= 509	n= 71	n= 32	n= 293	n= 113	
Endoleak	97 (19.9)	21 (29.6)	6 (18.8)	61 (20.5)	9 (8.04)	0.002
Ia	40/97 (41.2)	10 (47.6)	3 (50.0)	21 (34.4)	6 (66.7)	
Ib	35/97 (36.1)	7 (33.3)	2 (33.3)	25 (41.0)	1 (11.1)	
Ia & Ib	2/97 (2.1)	0 (0)	0 (0)	2 (3.3)	0 (0)	
IIa	12/97 (12.4)	4 (19.1)	1 (16.7)	5 (8.2)	2 (22.2)	
R	8/97 (8.3)	0 (0)	0 (0)	8 (13.1)	0 (0)	
Reinterventions	88 (17.3)	16 (22.5)	6 (18.8)	55 (18.8)	11 (9.7)	0.095
Endovascular	82/88 (93.2)	13 (81.3)	6 (100)	52 (94.5)	11 (100)	
Open conversion	6/88 (6.8)	3 (18.7)	0 (0)	3 (5.5)	0 (0)	
Mortality (late)	225 (44.2)	61 (85.9)	23 (71.9)	117 (39.9)	24 (21.2)	0.000
Overall Mortality	264 (51.9)	65 (91.6)	29 (90.6)	140 (47.8)	30 (26.6)	0.000
Aortic-related	51 (19.3)	12 (18.5)	6 (20.7)	28 (20.0)	5 (16.7)	
Non aortic-related	213 (80.7)	53 (81.5)	23 (79.3)	112 (80.0)	25 (83.3)	

SCI, spinal cord ischemia.

P-172

Microcirculatory Predictors of Thrombosis in Patients After COVID-19

Yulia Andozhskaya*

FSBBE HE I.P.Pavlov SPbSMU MOH Russia., Saint Petersburg, Russia

Introduction: The lack of a history of the course of a new coronavirus infection and the lack of data from randomised trials makes it difficult to choose the right treatment tactics and prescribe adequate prophylaxis in patients who have suffered from