The Impact Of Low BMI On Perioperative And Long-term Outcomes After TEVAR

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Objective

- While several studies have evaluated the impact of elevated body mass index (BMI) on outcomes after endovascular abdominal aortic aneurysm repair (EVAR), the relationship between underweight and long-term outcomes after thoracic endovascular aortic aneurysm repair (TEVAR) remains poorly understood.
- We investigated the role of low BMI on clinical and technical outcomes in patients who underwent TEVAR.

Methods

- The Global Registry for Endovascular Aortic Treatment (GREAT) Registry is an ongoing multicenter, multi-national, observational cohort study between August 2010-September 2016.
- All patients treated with Conformable GORE® TAG® Thoracic Endoprosthesis devices were included. Patients were considered underweight with BMI <23 kg/m2 according to National Institute of Health guidelines for patients ≥65 years.
- Normal BMI was defined by BMI ≥23-25 kg/m2. Long-term patient follow-up was assessed annually, up to 7 years postintervention. Primary outcomes were technical surgical complications, including rates of endoleak, rupture, and migration.
- Secondary outcomes included postoperative complications.



Results

- Out of 884 patients undergoing TEVAR, 161 (18%) were defined as underweight. 129(15%) were considered normal-weight, and 594 (67%) had BMI>25.
- One year postoperatively, there was no statistically significant difference in rates of endoleak (5.0% vs 6.2%, p=0.3) (Table 1).
- Underweight patients developed significantly higher rates of aortic rupture than normal weight patients at 1 year (3.1% vs 1.6%, p=0.02).

Characteristic	Underweight BMI<23	Normal Weight BMI 23-25	Overweight BMI 25-30	Obese BMI 30-35	Morbid Obesity BMI>35	p-value
	N=161	N=129	N=369	N=155	N=70	
Baseline Charecteristics						
Male	95 (59.0%)	74 (57.4%)	272 (73.7%)	106 (68.4%)	41 (58.6%)	
Female	66 (41.0%)	55 (42.6%)	97 (26.3%)	49 (31.6%)	29 (41.4%)	
Age (Years)						
n Marca (Chil Dav)	161	129	369	155	70	
Weight (kg)	05.0 (10.8)	07.5 (12.5)	05.4 (15.5)	02.0 (14.5)	55.5 (12.5)	
n	161	129	369	155	70	
Mean (Std Dev)	60.7 (10.4)	69.2 (9.0)	81.5 (10.0)	93.7 (12.5)	118.6 (18.7)	
Postoperative Details						
Any Endoleak	8 (5.0%)	8 (6.2%)	31 (8.4%)	7 (4.5%)	2 (2.9%)	0.3
Type IA Endoleak	4 (2 5%)	3 (2 3%)	6 (1.6%)	2 (1 3%)	0 (0.0%)	0.8
Type IB Endoleak	1 (0.6%)	4 (3 1%)	13 (3 5%)	1 (0.6%)	0 (0.0%)	0.076
Type II Endoleak	2 (1.2%)	2 (1.6%)	8 (2.2%)	3 (1.9%)	2 (2.9%)	>0.9
Type III Endoleak	0 (0.0%)	1 (0.8%)	3 (0.8%)	0 (0.0%)	0 (0.0%)	0.7
Type IV Endoleak	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	
Migration	0 (0.0%)	0 (0.0%)	2 (0.5%)	0 (0.0%)	0 (0.0%)	>0.9
Fracture	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	
Aortic Bunture	5 (3.1%)	2 (1.6%)	1 (0.3%)	0 (0.0%)	1 (1.4%)	0.017
All reinterventions	21 (13 0%)	20 (15 5%)	66 (17.9%)	21 (13 5%)	8 (11.4%)	0.4
All cause mortality	28 (17 4%)	20 (15 5%)	44 (11 9%)	10 (6 5%)	6 (8 6%)	0.026
23 years	20 (271170)	20 (2010/0)	44 (221570)	20 (01070)	0 (0.070)	01010
	N = 132	N = 108	N = 312	N = 140	N = 63	
Number of Subjects With Imaging and/or Event	77	80	202	95	43	
Any Endoleak	6 (7.8%)	2 (2.5%)	7 (3.5%)	4 (4.2%)	3 (7.0%)	0.4
Type IA Endolesk	1 (1 3%)	1 (1 3%)	1 (0.5%)	3 (3 2%)	1 (2 3%)	0.2
Type IB Endoleak	4 (5.2%)	1 (1.3%)	0 (0.0%)	1 (1.1%)	0 (0.0%)	0.012
Type II Endoleak	0 (0.0%)	0 (0.0%)	4 (2.0%)	0 (0.0%)	0 (0.0%)	0.5
Type III Endoleak	1 (1 3%)	0 (0.0%)	1 (0.5%)	0 (0.0%)	0 (0.0%)	0.5
Type IV Endoleak	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	0.0
Migration	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	
Fracture	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	
Aortic Bupture	0 (0.0%)	1 (1.0%)	1 (0.3%)	0 (0.0%)	0 (0.0%)	0.5
All reinterventions	8 (6 3%)	5 (4.8%)	13 (4 5%)	6 (4.7%)	4 (6 9%)	0.0
All cause mortality	14 (11.1%)	21 (20.0%)	35 (12.0%)	7 (5.5%)	3 (5.2%)	0.0006
4-7 years				. (51575)	- (c.c.r.)	
,	N = 109	N = 82	N = 265	N = 127	N = 56	
Number of Subjects With Imaging and/or Event	50	50	136	70	33	
Any Endoleak	2 (4.0%)	2 (4.0%)	7 (5.1%)	4 (5.7%)	2 (6.1%)	>0.9
Type IA Endoleak	0 (0.0%)	1 (2.0%)	3 (2.2%)	0 (0.0%)	0 (0.0%)	0.7
Type IB Endoleak	2 (4.0%)	1 (2.0%)	0 (0.0%)	1 (1.4%)	1 (3.0%)	0.089
Type II Endoleak	0 (0.0%)	0 (0.0%)	1 (0.7%)	2 (2.9%)	1 (3.0%)	0.3
Type III Endoleak	0 (0.0%)	0 (0.0%)	1 (0.7%)	0 (0.0%)	0 (0.0%)	>0.9
Type IV Endoleak	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	
Migration	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	
Fracture	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	
Aortic Bunture	1 (1 1%)	1 (1 4%)	3 (1 3%)	1 (0.9%)	0 (0.0%)	>0.9
All reinterventions	4 (4.3%)	3 (4.1%)	10 (4.4%)	6 (5.7%)	3 (6.4%)	0.9
All cause mortality	24 (26.1%)	16 (21.9%)	38 (16,9%)	23 (21.7%)	14 (29.8%)	0.2

Table 1. Technical and Postoperative Complications

Results

- After 2-3 years postoperatively, underweight patients had significantly higher rates of Type 1B endoleak when compared to normal weight patients (5.2% vs 1.3%, p=0.01).
- No difference in endoleak, migration, or rupture rates existed between underweight groups after 4-7 yrs postoperatively.
- No significant difference in reintervention rates was identified throughout all follow-up intervals. Interestingly, the rate of allcause mortality was significantly higher in the normal weight patients than underweight patients at the 1-year (17.4% vs 15.5%, p=0.03) and 2-3 year (11.1% vs 20.0%, p=.0006) intervals postoperatively. No significant difference in mortality was determined between the groups after 4-7 years postoperatively (Table 1).

Conclusions

- Patients with low BMI undergoing TEVAR experience higher rates of aortic rupture and Type 1B endoleak, with no significant difference in postoperative reintervention rates.
- These findings should be considered in risk reduction and preoperative optimization of patients considered for TEVAR.

References

- Mészáros I, Mórocz J, Szlávi J, et al. Epidemiology and clinicopathology of aortic dissection. Chest. 2000;117(5):1271–1278.
- Peterss S, Mansour AM, Ross JA, et al. Changing pathology of the thoracic aorta from acute to chronic dissection: literature review and insights. J Am Coll Cardiol. 2016;68(10):1054–1065.
- Bakris GL, Beckman JA, et al. 2010 ACCF/AHA/AATS/ACR/ASA/SCA/SCA/SCA/SIR/STS/SVM Guidelines for the diagnosis and management of
 patients with thoracic aortic disease. A report of the American College of Cardiology Foundation/American Heart Association Task
 Force on Practice Guidelines, American Stocker To Thoracic Surgery, American College of Radiology, American Stocke Association,
 Society of Cardiovascular Anesthesiologists, Society for Cardiovascular Angiography and Interventions, Society of Interventional
 Radiology, Society of Thoracic Surgeons, and Society for Vascular Medicine [published correction appears in J Am Coll Cardiol.
 2013;62(11):1039–1040]. J Am Coll Cardiol. 2010;55(14):e27–e129.
- Howard DP, Banerjee A, Fairhead JF, et al. Population-based study of incidence and outcome of acute aortic dissection and premorbid risk factor control: 10-year results from the Oxford Vascular Study. Circulation. 2013;127(20):2031–2037.
- Olsson C, Thelin S, Ståhle E, et al. Thoracic aortic aneurysm and dissection: increasing prevalence and improved outcomes reported in a nationwide population-based study of more than 14,000 cases from 1987 to 2002. *Circulation*. 2006;114(24):2611–2618.