Impact of shaggy aorta on outcomes of open thoracoabdominal aortic aneurysm repair

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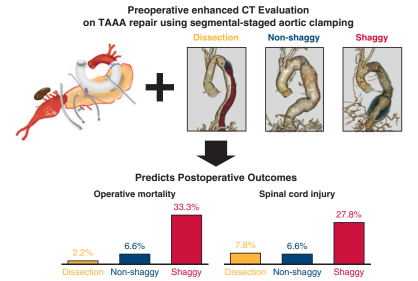
ABSTRACT

Background: The aim of this study was to evaluate the impact of diffuse aortic atherosclerosis–related thrombosis, or ‘‘shaggy aorta’’ on the outcomes of open thoracoabdominal aortic aneurysm repair (TAAA).

Methods: From October 1999 to March 2018, 251 patients underwent open TAAA repair using segmental-staged aortic clamping. Twenty-eight patients (11.2%) received emergent or urgent operations. Patients were classified into 3 groups: dissection aneurysm (139 patients, 55.4%), degenerative aneurysm without shaggy aorta (76 patients, 30.3%), and degenerative aneurysm with shaggy aorta (36 patients, 14.3%). Shaggy aorta was assessed using enhanced computed tomography and defined as patients with atheroma thickness ≥5 mm with irregular atheroma surface. Mean follow-up was 4.3 ± 4.1 years.

Results: Operative mortality was 8% (20 patients) and spinal cord injury occurred in 25 patients (10.0%), 16 of whom (6.4%) had permanent neurologic dysfunction. Operative mortality was significantly worse in patients with shaggy aorta (dissection: 2.2%, non-shaggy: 6.6%, and shaggy: 33.3%, $P < .001$) and shaggy aorta was a significant risk factor for spinal cord injury (dissection: 7.2%, non-shaggy: 6.6%, and shaggy: 27.8%, $P < .003$). Multivariable analysis demonstrated that shaggy aorta was a significant risk factor for composite outcome consisted of operative mortality, spinal cord injury, and acute renal failure (odds ratio, 4.78; 95% confidence interval, 1.91-12.3, $P < .001$).

Conclusions: Preoperative enhanced computed tomography assessment of shaggy aorta could predict high-risk patients for open TAAA repair. (J Thorac Cardiovasc Surg 2020;160:889-97)



Preoperative assessment of shaggy aorta predicted adverse outcomes.

Central Message

Shaggy aorta was a significant risk factor for spinal cord injury and early mortality after open thoracoabdominal aortic aneurysm repair.

Perspective

Preoperative enhanced computed tomography assessment of shaggy aorta could predict high-risk patients for open thoracoabdominal aortic aneurysm repair using segmental-staged aortic clamping in terms of mortality and spinal cord injury.

See Commentaries on pages 898 and 899.

Surgical repair of thoracoabdominal aortic aneurysms (TAAA) has been the most challenging operative procedure for cardiothoracic surgeons, as it continues to be associated with high morbidity and mortality.[1-4] Spinal cord injury (SCI) remains one of the most serious complications of TAAA repair. Various efforts for spinal cord protection during open TAAA repair have been employed; these include hypothermia, distal aortic perfusion, visceral perfusion, reattachment of intercostal arteries (ICAs), cerebrospinal fluid (CSF) drainage, and neuromonitoring. The incidence of permanent SCI was reported at a rate of 2.0% to 10.8% in meta-analysis of open TAAA repair.[5]

Historically, the major radicular artery (Adamkiewicz artery), arising at the level of T8-L1 in a majority of individuals, was thought to provide a significant proportion of blood flow to this region of the spinal cord in addition to the other segmental arteries (intercostal and lumbar).[6,7] Therefore, the primary strategy for the prevention of SCI was focused on preserving segmental artery flow. Griepp and Griepp[8]

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Abbreviations and Acronyms

CI = confidence interval
CPB = cardiopulmonary bypass
CSF = cerebrospinal fluid
CT = computed tomography
ICA = intercostal artery
OR = odds ratio
SCI = spinal cord injury
SCPP = spinal cord perfusion pressure
TAAA = thoracoabdominal aortic aneurysm
TEE = transesophageal echocardiography

demonstrated that the volume and density of the collateral network is far greater than that of the segmental arteries (intercostal and lumbar) directly supplying the spinal cord and that after manipulation of spinal cord blood flow during TAAA repair, spinal cord perfusion pressure (SCPP) returns to the preoperative baseline within 48 hours. This shifted the paradigm of SCI prevention toward more global strategies of maintaining SCPP while collateralization can develop.[9,10] However, besides the low SCPP, embolization was found to be a major cause of SCI following TAAA surgery.[11]

"Shaggy aorta," a term advocated by Hollier and colleagues,[12] was defined as very extensive atheromatous disease with diffuse ulcers associated with soft, loosely held debris and thrombus. The use of enhanced computed tomography (CT) imaging enables us to evaluate the severity of atheromatous disease preoperatively. Our previous report demonstrated that shaggy aorta was a significant risk factor for postoperative neurologic deficit following aortic arch surgery.[13] Ribeiro and colleagues[14] also reported that the severity of atheromatous disease predicted solid-organ infarction after endovascular treatment. In this study, we evaluated the impact of shaggy aorta evaluated by preoperative enhanced CT assessment on outcomes after open TAAA repair.

PATIENTS AND METHODS

Study Population

From October 1999 to March 2016, 276 patients underwent TAAA repair at our institution. Twenty-five patients, in whom adequate preoperative enhanced CT was not obtained, were excluded. Two-hundred fifty-one patients were included in this study.

Follow-up data were obtained by clinical visit, telephone, or written correspondence, and common closing date was used for analysis. Follow-up was available for 93.6% of the patients, and 16 patients were lost to follow-up. The mean follow-up period was 4.3 ± 4.1 years. This study protocol was reviewed and approved by the institutional review board. An informed consent waiver was granted, owing to the design of the study.

Assessment of Shaggy Aorta

Modifying the concepts formulated by Ribeiro and colleagues,[14] the diagnosis of shaggy aorta was defined by the following findings using preoperative enhanced CT (Figure 1): (1) in patients with degenerative aortic aneurysm; (2) thrombus was measured in non-aneurysmal aortic segments (<40 mm); (3) with atheroma thickness ≥5 mm; and (4) irregular atheroma surface showing finger-like projections. If the patient met all 4 findings, the diagnosis of shaggy aorta was confirmed. The assessment was performed by one observer, who was blinded to patient-identifying data, at 3 specific segments (aortic arch, descending aorta, and renal-mesenteric aorta). The most severe segment was used for diagnosis. When the aorta was replaced by prosthetic graft, atheroma thickness was considered 0 mm. All data were measured in the axial planes CT scan using Ziosotation2 (Ziosoft, Belmont, Calif).

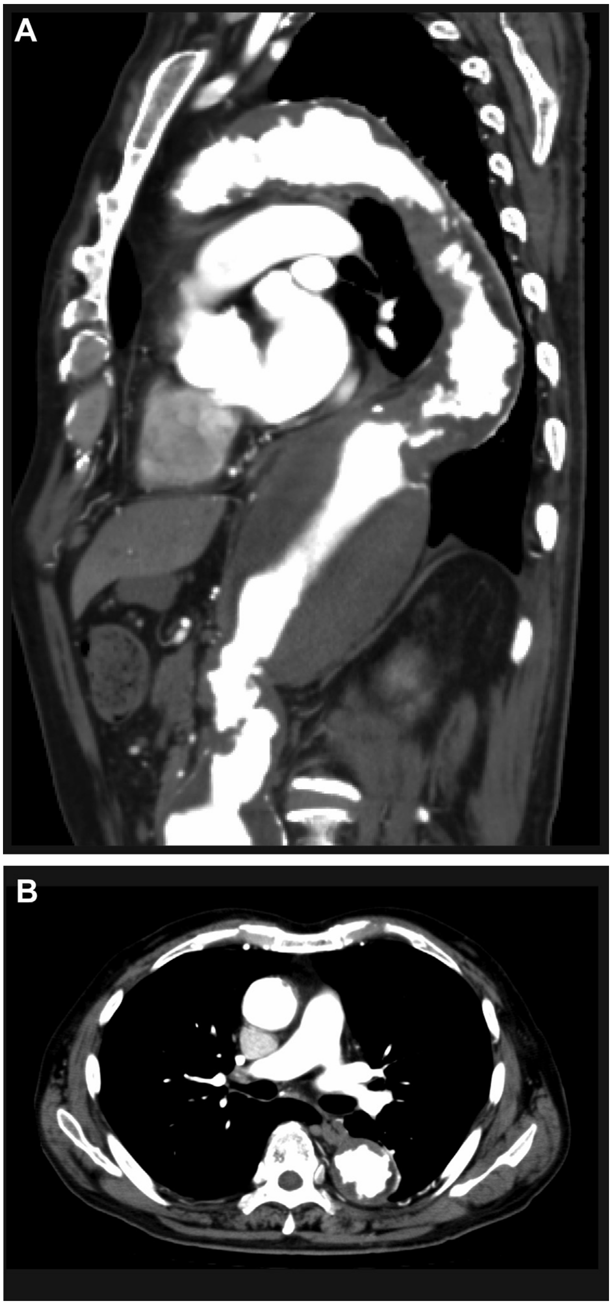


FIGURE 1. Enhanced computed tomography imaging for shaggy aorta: sagittal image (A) and axial image (B). Patients with shaggy aorta showed significant atherothrombosis at the non-aneurysmal segment of the aorta.

Surgical Approach

Our operative technique was reported in our previous study and is detailed in. Video 1[15,16] To summarize, an Adamkiewicz artery was confirmed using preoperative enhanced CT when possible. A CSF drainage catheter was inserted in the lumbar region 1 day before the operation, and CSF drainage was maintained for 72 hours postoperatively. Transcranial motor-evoked potentials were recorded throughout the operation.

Cardiopulmonary bypass (CPB) was exclusively established via arterial cannulation of the left femoral artery for inflow and the right femoral vein and the pulmonary artery for venous drainage. There were 11 patients (4.4%) who had severe atherosclerosis of the abdominal aorta, which was replaced with an interposition graft before establishing CPB. In these cases, arterial inflow was achieved via side-arm of the graft. Axillary arterial (n = 1) or descending aortic cannulation (n = 2) was performed when the femoral artery was occluded or too small to cannulate. The mean distal perfusion pressure was maintained at >70 mm Hg. We routinely use mild-to-moderate permissive hypothermia (30°C-34°C, rectal). When proximal aortic clamping was unfeasible, deep hypothermic circulatory arrest was used. Segmental-staged aortic clamping was employed to maximize segmental artery blood flow and prevent arterial ‘‘steal’’ from the back-bleeding of exposed segmental arteries.

Patent intercostal and lumbar arteries at the T8 to L2 levels were reattached. Each visceral artery was perfused by selective cannulation using a single roller pump at arterial flows of 150 to 200 mL/min. The visceral and renal arteries were individually dissected to create a button and were anastomosed using a 4-branch graft.

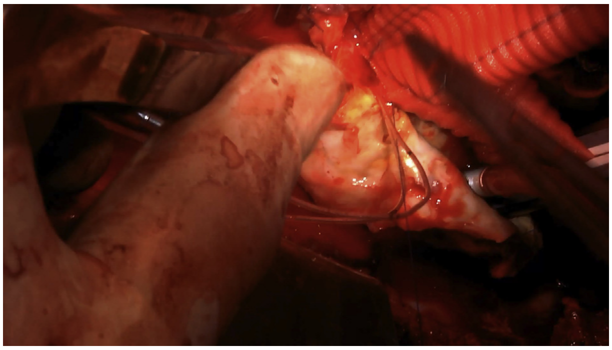
Statistical Analysis

All continuous variables are expressed as mean ± standard deviation or median with interquartile range, as appropriate. Categorical variables are expressed as the number (%) of patients. Data were analyzed by the χ^2 test for categoric variables. Assumption of normality of continuous data was tested with the Shapiro–Wilk test. If the assumption of normality was met, continuous variables were compared using the analysis of variance, whereas the Kruskal–Wallis test was used for nonparametric variables. The overall survival was calculated using the Kaplan–Meier methods with the rate ± standard error or the Cox hazard analysis model including shaggy aorta, age, and sex as variables.

P values < .05 were considered statistically significant differences. P values < .05 in the univariable analysis were used to identify variables for the multivariable regression analysis to evaluate independent predictors of the composite outcome (n = 52) using odds ratio (OR) with 95% confidence intervals (CIs). The composite outcome consisted of operative mortality, spinal cord injury including both permanent and temporary, and acute renal failure that required temporary or permanent renal-replacement therapy. For the univariable analysis we selected clinically significant risk factors based on recent literature.[1,6] These included shaggy aorta, age, male sex, diabetes mellitus, chronic kidney disease defined by estimated glomerular filtration rate <60 mL/min/1.73m^2, chronic obstructive pulmonary disease defined by preoperative forced expiratory volume in 1 second or previous diagnosis, coronary artery disease, defined by coronary artery stenosis >75%, previous aortic surgery, extent II surgery, nonelective surgery, CPB time, minimum rectal temperature, circulatory arrest, ICA reconstruction, and CSF drainage. All data analyses were performed with JMP 11.0 software (SAS Institute, Cary, NC).

RESULTS

The 251 patients were subdivided in 3 groups; dissection aneurysm (dissection: 139 patients, 55.4%), degenerative aneurysm without shaggy aorta (non-shaggy: 76 patients, 30.3%), and degenerative aneurysm with shaggy aorta (shaggy: 36 patients, 14.3%).



VIDEO 1. Extent II thoracoabdominal aortic aneurysm repair in a 74-year-old patient with shaggy aorta. The patient’s head is located at the right side of the video. The patient previously underwent total arch replacement with elephant trunk (ET) and endovascular abdominal aortic aneurysm repair. Preoperative enhanced computed tomography identified the left 11th intercostal artery (ICA) as the artery of Adamkiewicz. Cerebrospinal fluid drainage catheter was inserted in the lumbar region one day before the operation. Transcranial motor-evoked potentials were recorded throughout the operation. The patient was placed in the right lateral decubitus position. Endotracheal intubation was performed with a double-lumen endotracheal tube, enabling collapse of the left lung. A straight long incision was made from the inferior border of the left scapular to the left pararectal line. The aorta was then exposed through the 6th intercostal space with division of the costal arch. The abdominal aorta was exposed via a retroperitoneal approach. The arterial cannula was inserted in the left femoral artery. The venous drainage cannula was placed in the right femoral vein. The whole procedure was performed under mild hypothermia (29.7°, nadir rectal temperature). After full heparinization, the first segmental aortic clamps were placed at the level of the ET and the middle descending aorta (T6). The ET was identified by epi-aortic ultrasound. We used a 4-branch Dacron graft (Vascutek; Terumo, Tokyo, Japan). After proximal anastomosis, the second segmental aortic clamp was placed above the 11th ICA (artery of Adamkiewicz). We inserted balloon-tipped catheter (3 Fr, Pruitt; LeMaitre Vascular, Inc, Burlington, Mass) into the orifice of the 6th and 8th ICAs after aortic opening to prevent arterial ‘‘steal’’ from the spinal cord. The left 8th ICA was reattached to the graft using the single-cuff technique. The left 6th ICA was sacrificed. The third segmental aortic clamp was then placed above celiac artery. Bilateral 11th ICAs were reattached in a similar fashion. The last segmental aortic clamp was placed on the proximal endovascular stent. Visceral branches were reconstructed individually. Distally, the Dacron graft was anastomosed to the aorta and previously placed endovascular stent. Video available at: https://www.jtcvs.org/article/S0022-5223(19)31704-0/fulltext.

Patient Characteristics

Preoperative patient characteristics are shown in Table 1. Mean age was 62.6 ± 14.0 years (dissection: 55.2 ± 12.9 years, non-shaggy: 75.6 ± 8.9 years, and shaggy: 72.3 ± 9.5 years; $P < .001$). Patients with shaggy aorta had more comorbidities, such as chronic kidney disease and coronary artery disease, whereas patients with dissection had a greater incidence of connective tissue disease and history of aortic surgery. Emergent or urgent surgery was performed in significantly more patients with degenerative aneurysm with or without shaggy aorta. In

TABLE 1. Patient characteristics

			Degenerative aneurysm		
Variables	**Whole (n = 251)**	**Dissection aneurysm (n = 139)**	**Non-shaggy aorta (n = 76)**	**Shaggy aorta (n = 36)**	***P* value**
Age, y	62.6 ± 14.0	55.2 ± 12.9	75.6 ± 8.9	72.3 ± 9.5	<.001
Male sex	181 (72.1)	102 (73.4)	50 (65.8)	29 (80.6)	.231
BSA, m^2	1.69 ± 2.3	1.76 ± 0.23	1.58 ± 0.19	1.60 ± 0.18	<.001
Hypertension	207 (82.5)	112 (80.6)	62 (81.6)	33 (91.7)	.235
Dyslipidemia	67 (26.7)	29 (20.9)	27 (35.5)	11 (30.6)	.059
Diabetes mellitus	24 (9.6)	9 (6.5)	11 (14.5)	4 (11.1)	.160
CKD	136 (54.6)	61 (43.9)	44 (58.7)	31 (88.6)	<.001
COPD	45 (18.2)	22 (15.8)	13 (17.3)	10 (29.4)	.212
Coronary artery disease	49 (19.5)	12 (8.6)	22 (29.0)	15 (41.7)	<.001
Peripheral artery disease	15 (6.0)	5 (3.6)	8 (10.5)	2 (5.6)	.139
Connective tissue disease	38 (15.1)	38 (27.3)	0 (0)	0 (0)	<.001
Previous aortic surgery	113 (45.0)	80 (57.6)	20 (26.3)	13 (36.1)	<.001
Aortic arch replacement	58 (23.1)	39 (28.1)	12 (15.8)	7 (19.4)	
Descending aortic replacement	38 (15.1)	34 (24.5)	2 (2.6)	2 (5.6)	
TEVAR	13 (5.2)	10 (7.2)	3 (4.0)	0 (0)	
AAA repair	29 (11.6)	12 (8.6)	10 (13.2)	7 (19.4)	
EVAR	3 (1.2)	0 (0)	3 (4.0)	0 (0)	
Emergent/urgent	28 (11.2)	8 (5.8)	14 (18.4)	6 (16.7)	.010
Rupture	14 (5.6)	0 (0)	9 (12)	5 (14.3)	<.001
AKA identification	203 (80.9)	114 (82.0)	59 (77.6)	30 (83.3)	.380

BSA, Body surface area; *CKD*, chronic kidney disease; *COPD*, chronic obstructive pulmonary disease; *TEVAR*, thoracic endovascular aortic repair; *AAA*, abdominal aortic aneurysm; *EVAR*, endovascular aortic repair; *AKA*, Adamkiewicz artery.

the dissection group, 2 patients required urgent surgery in their acute phase due to lower body malperfusion.

Operative Details

Operative data are shown in Table 2. In patients with dissection, significantly more patients underwent the extent II TAAA repair, whereas significantly more patients with degenerative aneurysm received extent III or IV TAAA repair. Also, significantly more patients with dissection underwent aortic arch replacement. The median CPB time was 186 (119-234) minutes, and minimal rectal temperature was 32.9 (30.2-33.8)°C. Reflecting the extent of TAAA repair, significantly more patients with dissection received ICA reattachment.

TABLE 2. Operative data

			Degenerative aneurysm		
Variables	**Whole (n = 251)**	**Dissection aneurysm (n = 139)**	**Non-shaggy aorta (n = 76)**	**Shaggy aorta (n = 36)**	***P* value**
Extent of aortic replacement					<.001
Extent I	25 (10.0)	12 (9.4)	12 (15.8)	1 (2.9)	
Extent II	96 (38.3)	84 (60.4)	6 (7.9)	6 (16.7)	
Extent III	100 (39.8)	40 (28.8)	40 (52.6)	20 (55.6)	
Extent IV	30 (12.0)	4 (2.9)	17 (22.4)	9 (25.0)	
Concomitant arch replacement	10 (4.0)	9 (6.5)	0 (0)	1 (2.8)	.026
CPB time, min	186 (119-234)	215 (174-269)	132 (106-176)	134 (102-210)	<.001
Minimum rectal temperature, °C	32.9 (30.2-33.8)	32.2 (25.0-33.5)	33.0 (32.0-34.0)	33.1 (32.0-34.0)	<.001
Circulatory arrest	32 (12.7)	29 (20.9)	2 (2.6)	1 (2.8)	<.001
ICA reattachment	192 (76.5)	128 (92.1)	45 (59.2)	19 (52.8)	<.001
CSF drainage	205 (81.5)	128 (92.1)	50 (64.9)	27 (77.1)	<.001

CPB, Cardiopulmonary bypass; *ICA*, intercostal artery; *CSF*, cerebral spinal fluid.

TABLE 3. Early outcomes

	Whole	Dissection aneurysm	Degenerative aneurysm		
			Non-shaggy aorta	Shaggy aorta	
Variables	(n = 251)	(n = 139)	(n = 76)	(n = 36)	*P* value
Operative mortality	20 (8.0)	3 (2.2)	5 (6.6)	12 (33.3)	<.001
Spinal cord injury	25 (10.0)	10 (7.2)	5 (6.6)	10 (27.8)	.003
Paraplegia	14 (5.6)	4 (2.9)	3 (4.0)	7 (19.4)	
Paraparesis	11 (4.4)	6 (4.3)	2 (26)	3 (8.3)	
RBC transfusion, U	18 (12-28)	22 (14-28)	16 (10-26)	20 (10-34)	.053
Re-exploration	14 (5.6)	8 (5.8)	3 (4.0)	3 (8.3)	.598
Stroke	9 (3.6)	4 (2.9)	4 (5.3)	1 (2.8)	.664
Acute renal failure	34 (13.5)	8 (5.8)	11 (13.9)	15 (41.7)	<.001
Tracheostomy	27 (10.8)	8 (5.8)	9 (11.8)	10 (27.8)	.002
Hospital stay	30 (21.0-45.3)	29.0 (22.0-40.0)	27.0 (17.8-50.5)	38 (19.5-60.3)	.241
Composite outcome	52 (20.7)	17 (12.2)	15 (19.7)	20 (55.6)	<.001

RBC, Red blood cell.

Early Outcomes

Mortality. The early outcomes are summarized in Table 3. The operative mortality was 8.0% (20/251). Operative mortality was significantly worse in patients with shaggy aorta (dissection: 2.2%, non-shaggy: 6.6%, and shaggy: 33.3%, $P < .001$) (Figure 2). In the shaggy aorta group, cause of death included hemorrhage (n = 3), sepsis (n = 2), bowel ischemia (n = 1), cardiac failure (n = 1), hemorrhagic stroke (n = 1), drug-induced agranulocytosis (n = 1), disseminated intravascular coagulation due to multiple emboli (n = 1), and multiorgan failure (n = 1). In the subgroup analysis for patients with degenerative aneurysm, shaggy aorta was also a significant risk factor for operative mortality (OR, 5.83; 95% CI, 1.97-17.2, $P < .001$). In elective cases, operative mortality was 5.8% (13/223), and it was also significantly greater in patients with shaggy aorta (dissection: 1.4%, non-shaggy: 4.8%, and shaggy: 26.7%, $P < .001$).

Complications. The overall incidence of SCI was 10.0% (25/251). Paraplegia (permanent SCI) and paraparesis (temporary SCI) developed in 14 patients (5.6%) and 11 patients (4.4%), respectively. The incidence of SCI was significantly greater in patients with shaggy aorta (dissection: 7.2%, non-shaggy: 6.6%, and shaggy: 27.8%, $P < .001$) (Figure 2). In the subgroup analysis for patients with degenerative aneurysm, shaggy aorta was also a significant risk factor for SCI (OR, 4.49; 95% CI, 1.48-13.6, $P = .007$). In elective cases, SCI developed in 22 patients (9.9%), and a similar trend was observed in patients with shaggy aorta, although it was not statistically significant (dissection: 7.6%, non-shaggy: 8.1%, and shaggy: 23.3%, $P = .060$). The incidence of composite outcome was significantly greater in patients with shaggy aorta (dissection: 12.2%, non-shaggy: 19.7%, and shaggy: 55.6%, $P < .001$). In the subgroup analysis for patients with degenerative aneurysm, shaggy aorta was also a significant risk factor for composite outcome (OR, 5.08; 95% CI, 2.14-12.1, $P < .001$).

Univariable analysis revealed that shaggy aorta, age, male sex, chronic kidney disease, chronic coronary artery disease, and CPB time were significant risk factors for the composite outcome, which consisted of operative mortality, SCI, and acute renal failure (Table 4).

Multivariable analysis demonstrated that shaggy aorta (OR, 4.78; 95% CI, 1.91-12.3, $P < .001$), age (OR, 1.07; 95% CI, 1.02-1.12, $P = .002$), and CPB time (OR, 1.01; 95% CI, 1.01-1.02, $P < .001$) were significant risk factors for the composite outcome (Table 4).

Late Outcomes

Overall survival was 79.8 ± 2.9% at 5 years and 66.0 ± 4.7% at 10 years, respectively (Figure 3, *A*). Stratified by aortic pathology, 5-year survival was significantly lower in patients with shaggy aorta (dissection: 90.5 ± 3.0%, non-dissection: 70.2 ± 6.5%, and shaggy: 54.9 ± 8.9%, $P < .001$) (Figure 3, *B*). Cox hazard modeling using shaggy aorta, age, and sex showed that shaggy aorta was a significant risk factor for overall death compared with other groups (hazard ratio, 2.15; 95% CI, 1.14-3.91, $P = .018$).

DISCUSSION

With a growing elderly population, the Japanese Association for Thoracic Surgery has reported a steady increase in the number of patients with aortic disease.[17] Given that the progression of atherosclerosis contributes heavily to the development of shaggy aorta, we are likely to see an increasing number of patients with severe atherothrombosis presenting for TAAA repair. In this study, we noted a significant association of shaggy aorta with operative mortality. The Japanese Association for Thoracic Surgery recently

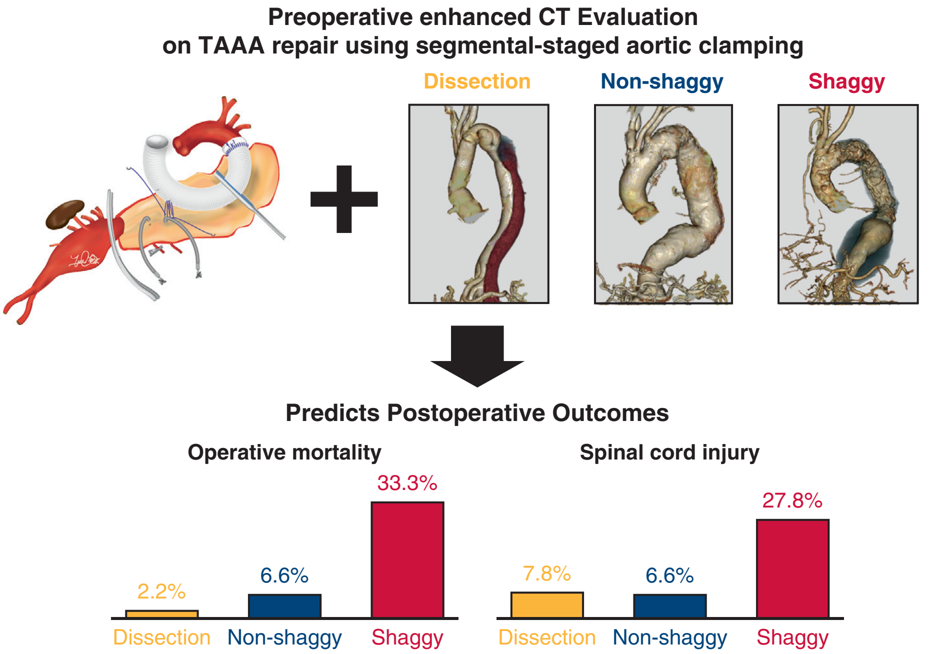


FIGURE 2. Operative mortality and the incidence of spinal cord injury were significantly greater in patients with shaggy aorta. Mortality: dissection: 2.2%, non-shaggy: 6.6%, and shaggy: 33.3%, $P < .001$. Spinal cord injury: dissection: 7.2%, non-shaggy: 6.6%, and shaggy: 27.8%, $P < .001$. *CT*, Computed tomography; *TAAA*, thoracoabdominal aortic aneurysm.

reported that the overall hospital mortality rate after open TAAA repair is 10.7%.[18] As our patient population ages, we will face more chance to encounter patients with shaggy aorta.

Permanent neurologic deficits are independently associated with increased morbidity and mortality.[19-21] This study also demonstrated that shaggy aorta diagnosed by preoperative enhanced CT assessment was a significant risk factor for operative mortality and SCI (Figure 2). Messe and colleagues[19] reported that patients with SCI had a mortality of 39% compared with 14% without SCI. Based on the concept of collateral circulation, 3 factors likely contribute to the increased incidence of SCI in patients with shaggy aorta: (1) decreased SCPP due to aortic manipulation and segmental artery sacrifice, (2) direct embolization of aortic thrombus and debris to segmental arteries, and (3) diminished preoperative microvascular and collateral circulation due to severe atherosclerosis. A recent study by Tanaka and colleagues[11] found that embolism may be a much more prevalent cause of SCI than previously thought, which suggests that the significantly greater incidence of SCI in patient with shaggy aorta is related to embolic events. Moreover, embolic events leading to organ or tissue ischemia could induce systemic hypotension, thereby precipitating or exacerbating SCI.

The optimal diagnostic method for shaggy aorta remains controversial. Ribeiro and colleagues[14] developed a quantitative method to evaluate the severity of aortic wall thrombus using a scoring system from 0 to 11. The scoring system consists of 5 factors: location, atheroma type, thickness, area, and circumference. However, the complexity of assessment proved problematic in clinical practice. To simplify the scoring process, we adopted a modified process for the assessment of shaggy aorta that uses2 factors: thrombus type (finger-like projection or not) and atheroma thickness (≥5 mm or not).

Another assessment method by Amarenco and colleagues[22] suggests that a critical threshold of 4 mm in thickness of atherosclerotic plaque measured by transesophageal echocardiography (TEE) is a significant predictor for embolic events. Izumi and colleagues[23] reported that in addition to the ≥5 mm in thickness of aortic plaque, the mobility of the plaque, as assessed by TEE, also predicts a greater incidence of embolic events. The combination of TEE and enhanced CT may enhance the accuracy of the diagnosis and determination of disease severity, although further analysis is required. Moreover, we have already adopted epi-aortic ultrasonography to determine the optimal clamp site in high-risk cases. Although enhanced CT cannot evaluate the mobility of the atherothrombotic plaque, a high association between clinical outcomes and diagnosis of shaggy aorta suggest that our methods may be more clinically applicable for cardiothoracic surgeons seeking to assess preoperative risk. Furthermore, we believe that classification of aortic pathologies based on dissection and degenerative aneurysm with or without shaggy aorta is more suitable than simply using dissection and degenerative aneurysm. This distinction will better risk-stratify patients

TABLE 4. Univariable and multivariable analysis for composite outcome

Variables	Univariate OR (95% CI)	Univariate *P* value	Multivariate OR (95% CI)	Multivariate *P* value
Shaggy aorta	7.15 (3.37-15.5)	<.001	4.78 (1.91-12.3)	<.001
Age (+1 y)	1.06 (1.03-1.09)	<.001	1.07 (1.02-1.12)	.002
Male sex	2.11 (1.01-4.89)	.048	1.48 (0.57-4.16)	.424
Diabetes mellitus	1.67 (0.61-4.12)	.301		
CKD	4.45 (2.19-9.84)	<.001	2.05 (0.83-5.39)	.123
COPD	1.16 (0.51-2.48)	.706		
Coronary artery disease	3.34 (1.67-6.64)	<.001	1.55 (0.66-3.57)	.310
Previous aortic surgery	1.42 (0.77-2.63)	.262		
Extent II	0.60 (0.30-1.15)	.127		
Non-elective	1.71 (0.67-4.05)	.236		
CPB time (+1 min)	1.01 (1.00-1.01)	.003	1.01 (1.01-1.02)	<.001
Minimum rectal temperature (+1°C)	1.03 (0.97-1.11)	.318		
Circulatory arrest	0.70 (0.23-1.79)	.482		
ICA reconstruction	0.64 (0.33-1.28)	.202		
CSF drainage	0.78 (0.38-1.76)	.555		

Composite outcome consisted of operative mortality, spinal cord injury, and acute renal failure. *OR*, Odds ratio; *CI*, confidence interval; *CKD*, chronic kidney disease; *COPD*, chronic obstructive pulmonary disease; *CPB*, cardiopulmonary bypass; *ICA*, intercostal artery; *CSF*, cerebrospinal fluid.

when performing a preoperative assessment for TAAA repair. To further elucidate these differences, we included patients with dissection to confirm the equivalent clinical outcomes between dissection and non-shaggy groups, although this might be a less relevant comparison than directly comparing aneurysm patients.

The contemporary incidence of permanent SCI following open TAAA repair stratified by aneurysmal extent was reported as 2.5% to 24% in extent I, 5.4% to 22% in extent II, 2.6% to 13% in extent III, and 1.4% to 2.2% in extent IV.[1,4,24-27] In the setting of recent literature, our study reported a low incidence of permanent SCI at our institution (4.0% in extent I, 5.3% in extent II, 7.0% in extent III, and 4.0% in extent IV) (Table E1). The results demonstrate the feasibility of our strategies in open TAAA repair using segmental-staged aortic clamping, particularly in patients with dissection or non-shaggy aorta. Although we did not have the data regarding the number of times the aorta was segmentally clamped, this varied based on the extent of patient disease. For example, patients with dissection underwent segmental clamping a greater number of times due to greater extent of repair. Similar selection bias might be in ICA reconstruction and CSF drainage, which are reputed to have positive effects on the incidence of SCI. Despite the benefit of distal perfusion with femoral arterial cannulation, there might be the potential for retrograde embolization of atheromatous debris. Therefore, this procedure may be avoided in patients with shaggy aorta.

Given that shaggy aorta was significantly associated with SCI, operative technique may need to be modified for patients with this diagnosis. Tanaka and colleagues[27] reported satisfactory outcomes of open TAAA repair using deep hypothermia (SCI, 2.0%). Although there are negative effects of using deep hypothermia, including coagulopathy, cardiac dysfunction, respiratory failure, renal failure, and stroke,[27,28] this strategy should be strongly considered in patients with shaggy aorta for spinal cord protection, as these patient may not be able to safely undergo segmental-staged aortic clamping.

Recent literature has supported the strategy of staged TAAA repair to promote collateralization. Etz and colleagues[29] performed a retrospective analysis comparing 35 staged TAAA repairs, which were performed months apart, versus 55 single-stage TAAA surgeries, and found that patients in the staged group had a significantly lower incidence of SCI (0 vs 15%). However, our results reveal that in the shaggy aorta group, more than one half of the patients (6/10) developed SCI, even in groups undergoing less severe extent III or IV TAAA repair.

Hybrid or endovascular TAAA repair has become an attractive alternative strategy, particularly in high-risk patients, as recent studies report that the incidence of SCI was 7.0% to 19% in these procedures.[24,30-32] However, shaggy aorta is considered a prominent risk factor during endovascular procedures due to high risk of diffuse embolization.[33,34] Further analysis is needed to better understand outcomes of hybrid and endovascular TAAA repair in patients with shaggy aorta.

Limitations

There are several limitations in our study. Our results come from a retrospective review using data from a single

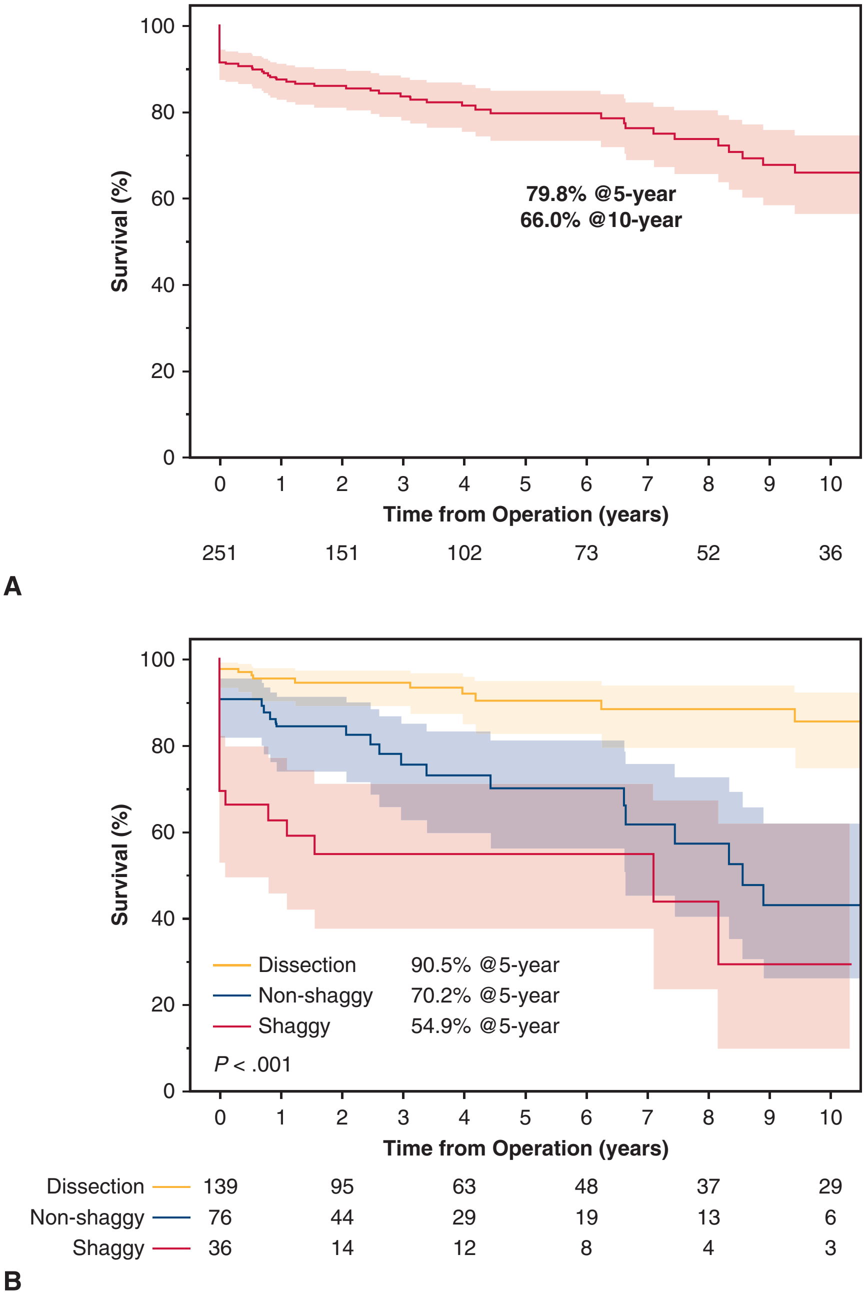


FIGURE 3. Overall survival was 79.8 ± 2.9% at 5 years and 66.0 ± 4.7% at 10 years, respectively (A). Stratified by aortic pathologies (dissection, non-shaggy aorta, and shaggy aorta), 5-year survival was significantly lower in patients with shaggy aorta (group dissection: 90.5 ± 3.0%, group nonshaggy: 70.2 ± 6.5%, and group shaggy aorta: 54.9 ± 8.9%, $P < .001$) (B).

center. Although multivariable analysis demonstrates that shaggy aorta poses a significant risk for composite outcome, there is significant selection bias between each group based on the differences in aortic pathologies. Selecting covariates for the multivariable model based on univariate significance has considerable limitations including reliance on sample size, which introduces bias. Our analysis was performed by a single analyst. As imaging was interpreted by a cardiothoracic surgeon, secondary evaluation by a radiologist would improve the accuracy of diagnosis. We did not have the data regarding the number of times the aorta was segmentally clamped, although this varied based on the extent of patient disease. Lastly, according to the definition of shaggy aorta, we did not analyze the impact of atherothrombosis inside the aneurysm (>40 mm), which may influence the outcomes. The longitudinal extent of shaggy aorta was not included in the assessment because aneurysm extent varies among patients. Further analysis is needed.

CONCLUSIONS

Open TAAA repair was performed with acceptable survival and incidence of SCI. Preoperative enhanced CT assessment of shaggy aorta could predict high-risk patients for open TAAA repair in terms of mortality and SCI. In patients who underwent segmental-staged aortic clamping for thoracoabdominal aortic repair, shaggy aorta may be a risk factor for adverse outcomes.

Conflict of Interest Statement

Authors have nothing to disclose with regard to commercial support.

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Key Words: thoracoabdominal aortic aneurysm repair, computed tomography, preoperative assessment, shaggy aorta

TABLE E1. Incidence of spinal cord injury according to aneurysm extent

			Degenerative aneurysm	
	Whole	Dissection aneurysm	Non-shaggy aorta	Shaggy aorta
Variables	(n = 251)	(n = 139)	(n = 76)	(n = 36)
Spinal cord injury	25 (10.0)	10 (7.2)	5 (6.6)	10 (27.8)
Extent I	1 (4.0)	0 (0)	0 (0)	1 (100)
Extent II	8 (8.3)	5 (5.6)	0 (0)	3 (50.0)
Extent III	14 (14.0)	4 (10.0)	5 (12.5)	5 (25.0)
Extent IV	2 (6.7)	1 (25.0)	0 (0)	1 (11.1)
Permanent spinal cord injury	14 (5.6)	4 (2.9)	3 (4.0)	7 (19.4)
Extent I	1 (4.0)	0 (0)	0 (0)	1 (100)
Extent II	5 (5.3)	2 (2.4)	0 (0)	3 (50.0)
Extent III	7 (7.0)	1 (2.5)	3 (7.5)	3 (15.0)
Extent IV	1 (4.0)	1 (25.0)	0 (0)	0 (0)