

Flood Tide Sign in CDIIIb Aneurysm: Anatomic-Hemodynamic Comprehension and Endovascular Treatment Strategies

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Abstract

Purpose: The purpose of this study was to understand the anatomic-hemodynamic characteristics of chronic dissecting DeBakey type IIIb (CDIIIb) aneurysms showing the flood tide sign (FTS), and its implication for endovascular treatment strategies.

Materials and Methods: Among 94 patients with CDIIIb aneurysm who underwent thoracic endovascular aortic repair (TEVAR) between June 2012 and June 2016, 16 patients showing the FTS in pretreatment computed tomography angiography were included. The pretreatment entry and re-entry tear sizes were measured. The growth rate of the false lumen (FL) of the descending thoracic aorta (DTA) was calculated. Wilcoxon signed-rank test was used to analyze the reduction in FL volume after treatment. Favorable aortic remodeling rate was assessed.

Results: The patients were divided into three subgroups based on the ratio of the summed area of entry and re-entry tear(s). All patients showed aneurysmal change and partial thrombosis of the FL of the DTA. The overall growth rate of the FL was 7.03 ± 6.64 mm/year. The overall FL volume reduction rate was $43.8 \pm 23.9\%$ ($p = 0.002$), and a significant FL volume reduction in each subgroup (group A, $56.3 \pm 32.4\%$, $p = 0.043$; group B, $33.3 \pm 12.7\%$, $p = 0.043$; group C, $38.7 \pm 8.8\%$, $p = N/A$) was achieved. Favorable aortic remodeling was achieved in 14 of 16 (87.5%) patients during the 19.7 ± 13.8 months of follow-up.

Conclusion: Considering the anatomic-hemodynamic status of the CDIIIb aneurysm, not only stent grafts but also adjunctive endovascular treatment is mandatory to achieve favorable aortic remodeling.

Introduction

The flood tide sign (FTS) was introduced by Umberto et al. in a case of subacute aortic dissection [1]. It was described as a delayed filling of contrast on the false lumen in arterial phase, with a blood/contrast level on the venous phase on computed tomography (CT) angiography (CTA). It was seen in a 1-month follow-up CTA of a patient who underwent graft replacement of the ascending thoracic aorta for the treatment of an acute Stanford type A (DeBakey type I) aortic dissection. The CTA revealed an entry tear distal to the left subclavian artery with a suspected lack of re-entry tear(s). The authors suspected that the FTS was due to imbalance of the entry and re-entry tear(s) that resulted in fast degeneration of the false lumen, and performed treatment by means of fenestration of the abdominal aorta to create an artificial re-entry, in order to achieve flow balance between the true and false lumen in the absence of a distal re-entry tear. In the current study, we were able to find the FTS in patients with chronic dissecting DeBakey type IIIb (CDIIIb) aneurysm treated with thoracic endovascular aortic repair (TEVAR) and adjunctive endovascular procedures.

The results of conventional TEVAR for CDIIIb aneurysms (i.e., aortic true lumen stent grafting) showed limited favorable aortic remodeling [2 - 8]. For the clinical success of the endovascular treatment for CDIIIb aneurysms, understanding the anatomic-hemodynamic situation of each patient is crucial. We hypothesized that the FTS represents flow stasis and increased diastolic pressure in the false lumen, which can contribute to aneurysmal degeneration, expansion, and partial thrombosis of the false lumen; therefore, it can be a predictor of adverse prognosis in patients with aortic dissection. Furthermore, different anatomic-hemodynamic mechanisms may be responsible for the appearance of the FTS, and therefore different endovascular strategies might be needed. The purpose of this study was to understand the anatomic-hemodynamic characteristics of CDIIIb aneurysms showing the FTS, and the implication of the FTS to endovascular treatment strategies.

Materials and Methods

Patient selection

From June 2012 to June 2016, a total of 94 patients with a CDIIIb aneurysm [including surgically treated DeBakey type I (Stanford type A) aortic dissection with residual descending thoracic and abdominal aortic dissecting aneurysm] who underwent TEVAR at our hospital were reviewed retrospectively. Among them, 16 patients (17%) who showed FTS in the pre-treatment CTA were included in this study. The institutional review board approved this retrospective clinical study.

The risk factors, surgical history, and CTA findings of patients are summarized in Table 1. The chronological age of aortic dissection of the included patients was 37.3 ± 24.3 months. The mean patient age was 59.6 ± 11.9 years (59 ± 12.9 years for men and 61.5 ± 9.5 years for women), and most of the patients (75%) were men. Many patients had a history of hypertension (81.3%) or atherosclerosis (62.5%). Concerning prior aortic surgeries, eight patients (50%) underwent graft replacement of the ascending thoracic aorta and two patients (12.5%) underwent total arch replacement.

TEVAR and adjunctive endovascular procedures

All interventions were performed under general anesthesia in a sterile hybrid aortic operating room. The procedural details are shown in Table 2. The entry tears were treated with stent grafts (Zenith TX2 Pro-Form™; COOK, Bjæverskov, Denmark/Valiant Captivia; Medtronic, Minneapolis, MN, USA) or AMPLATZER Vascular Plug (AVP; St. Jude Medical, Plymouth, MN, USA). Seven of the 16 patients underwent hybrid TEVAR (one zone 0, three zone 1, and three zone 2) after bypass surgery of aortic arch branch vessel(s); 5 of the 16 patients underwent simple TEVAR (four zone 3 and one zone 4); and the remaining 4 patients underwent closure of the entry tear with AVP. Additional aortic arch and DTA false lumen obliteration by using AVPs and/or coils were performed if necessary. Visceral artery re-entry tear(s) and iliac re-entry tear(s) were treated with a VIABAHN endoprosthesis (Gore, Flagstaff, AZ, USA). The communicating channels were also treated with AVPs and/or coils. Additional false lumen procedures were performed with AVPs, coils, or *n*-butyl cyanoacrylate (NBCA) to block retrograde flow and pressurization into the DTA false lumen. Access to the DTA false lumen was possible through the iliac tree re-entry in most tears, and through an abdominal aorta communicating channel or visceral artery re-entry tear site in cases without iliac tree re-entry.

Preprocedural drainage of cerebrospinal fluid (CSF) was performed routinely in nine patients with TEVAR extending below the T10 level to the supra-celiac or supra-SMA region, those with multiple intercostal arteries deriving from the DTA false lumen, and those with simultaneous obliteration of the DTA and the abdominal aorta false lumen.

Imaging analysis

Three-phase (precontrast, arterial phase, and 3-min delayed phase) contrast-enhanced CTA was performed by using two multidetector CT scanners. The scan parameters were as follows. For the first CT (Somatom definition AS+; Siemens): detector width, 0.6 mm; gantry rotation time, 0.5 s; pitch, 1.0; tube voltage, 100 kV; and tube current, CARE Dose4D. For the second CT (Sensation 64; Siemens): detector width, 0.6 mm; gantry rotation time, 0.5 s; pitch, 1.5; tube voltage, 100 kV; and tube current, CARE Dose4D. Contrast enhancement was achieved by injecting 70–100 mL of iodated contrast (Optiray 350; Guerbet, Xenetix 350; Guerbet, Ultravist 370; Bayer, Pamiray 300; Dongkook) at a flow rate of 4 mL/s, followed by a 50 mL saline bolus. The arterial phase was timed by means of bolus tracking, and the delayed phase was obtained 3 min later. Multiplanar reconstructions (axial, coronal, and oblique sagittal parallel to the aortic arch direction) of 3-mm slice thickness were obtained. Volume-rendering three-dimensional images were also obtained.

All CTAs of the selected patients were reviewed by four experienced radiologists with an experience ranging 5 to 26 years based on consensus. The FTS was defined as positive if a blood/contrast level was seen in the DTA false lumen on delayed CTA, while the arterial phase showed a relatively inhomogeneous lower enhancement of the aneurysmal false lumen compared with the true lumen (Figure 1). The sizes of entry and re-entry tear(s), and the maximal diameters of the DTA false lumen were measured in multiplanar CTA images. Volumetric analysis of the false lumen was done with three-dimensional reconstruction software (Aquarius iNtuition 4.4.6; TeraRecon, San Mateo, CA, USA). The volume of the false lumen was measured from the proximal DTA to the supra-celiac thoracoabdominal aorta. Thromboexclusion of the entire DTA was defined as the absence of contrast enhancement even on 3-min delayed images. Aortic remodeling was defined as favorable if reduction of the false lumen volume and complete thromboexclusion of the entire DTA were achieved.

Statistical analyses

The data were analysed by using SPSS statistical software (version 21.0; SPSS Inc., Chicago, IL, USA). The clinical characteristics are presented as frequencies and percentages for categorical variables and as means \pm SD for continuous variables. Continuous variables were compared by using the Wilcoxon signed-rank test. Probability values of <0.05 were considered statistically significant.

Results

All patients showed aneurysmal change of the DTA false lumen and partial thrombosis adhering to the wall of it. The overall growth rate of the DTA false lumen was 7.03 ± 6.64 mm/year.

Patients were divided into three subgroups based on the ratio of the summed area of entry and re-entry tears, and the suggested endovascular strategies differed among them (Figure 2). The aortic arch branch vessel re-entries, if present in patients who underwent prior ascending graft replacement for Stanford type A (DeBakey type III) aortic dissection, were calculated as entry tear(s) in this equation. In group A (entry tear dominant type), the dominant entry tear and poor distal re-entry tear(s) (i.e., massive inflow with poor exit outflow) resulted in false lumen aneurysmal degeneration. Group B (re-entry tear dominant type) showed poor entry tear but dominant distal re-entry tear(s), meaning more pressurization from retrograde flow resulting in false lumen aneurysmal degeneration. In group C (equivalent type), both proximal entry tear and distal re-entry tear(s) were equivalently large; that is, both ends acted as entries with equivalent flow coming from the opposite direction, creating flow turbulence and stasis in the middle of the DTA that resulted in false lumen aneurysmal degeneration. Concerning the ratio of the summed areas of entry and re-entry tears, the area of entry tear in group A was 14.9 ± 6.9 times larger than the sum of re-entries. In group B, the ratio was 0.08 ± 0.04 . In group C, the summed areas of entry tear and re-entry tears were similarly large, with a ratio of 0.83 ± 0.16 .

The results of TEVAR and adjunctive endovascular treatment according to the anatomic and hemodynamic considerations are summarized in Table 3. Complete thromboexclusion of the entire DTA was achieved in 14 of 16 patients (87.5%). The overall false lumen volume reduction rate was $43.8 \pm 23.9\%$ ($p = 0.002$), and a significant reduction of the false lumen volume in each subgroup (group A, $56.3 \pm 32.4\%$, $p = 0.043$; group B, $33.3 \pm 12.7\%$, $p = 0.043$; group C, $38.7 \pm 8.8\%$, $p = \text{N/A}$) was also achieved (Figure 3). Favorable aortic remodelling on the targeted entire DTA was achieved in 14 of 16 patients (87.5%) during the follow-up period of 19.7 ± 13.8 months.

No perioperative major morbidity and mortality occurred. Concerning complications related to CSF drainage, four of nine patients who underwent CSF drainage experienced transient postural headaches. Two patients showed stent-induced new intimal tear at the distal margin of the stent graft during the follow-up period, which was treated with additional stent grafting. There was no late mortality during the follow-up period.

Discussion and Conclusion

Most patients with acute type B/III aortic dissection treated with optimal medical therapy showed an increased false lumen diameter during the follow-up and eventually required endovascular or surgical treatment [9, 10]. Patients who underwent any aortic intervention had a significant survival advantage over those who were treated with medical management alone [10]. In a double-barreled aorta, in which flow entry through the entry tear and flow exit through the re-entry tear is balanced, the dissection would be in a chronic stabilized state. However, if either side is dominant or the force is equivalent, flow stasis and turbulence occur and result in progressive enlargement of the false lumen. A

randomized controlled study that enrolled patients with sub-acute uncomplicated type B aortic dissection treated with optimal medical therapy showed significant progression of dissecting aneurysm and increased adverse events [11]. Such a hemodynamic imbalance might be the major cause of progressive enlargement of the false lumen.

Among the patients with CDIIIb aneurysm who underwent TEVAR and endovascular treatment in this study, 17% showed the FTS. If there is flow imbalance between the proximal entry and distal re-entry tears, blood flow into the false lumen from the dominant side cannot escape through the poor side, resulting in flow stasis, which is visualized as the FTS. This explains why groups A and B show the FTS. This mechanism could be explained by an *ex vivo* model of chronic aortic dissection; that is, the diastolic pressure of the false lumen was the highest in the setting of a smaller proximal tear size, and the lack of a distal tear eventually influence the expansion of the false lumen during the follow-up period [12 - 14]. Furthermore, there were patients who showed fairly even sizes of entry and re-entry tears and were classified as group C. If the flow into the false lumen is equivalent between the proximal entry and distal re-entry tears, the opposing force would result in flow turbulence and stasis at the mid to distal DTA false lumen. This phenomenon could be expressed as a “hemodynamic blind sac” that would accelerate the aneurysmal degeneration of the false lumen. All patients showed aneurysmal degeneration and expansion of the false lumen, as well as partial thrombosis adhering to the wall of it. The mean growth rate of the DTA false lumen was 7.03 ± 6.64 mm/year, which means rapid enlargement of the false lumen in patients showing FTS. Therefore, patients with CDIIIb aneurysm showing the FTS and having a false lumen with a growth rate faster than 5 mm/year on follow-up CTA might be indicated for TEVAR and endovascular treatment.

Group A patients showed poor re-entry tears; thus, sealing-off of only the entry tear is sufficient to achieve favorable aortic remodelling in most cases. However, there were cases showing considerably sized re-entry tear(s) in group A owing to the huge size of the entry tear. These cases might require adjunctive false lumen obliteration and/or re-entry tear intervention if substantial retrograde false lumen flow persists after the treatment of the initial entry tear with TEVAR. This was applied in patient numbers 6 and 7. In patient number 6, all three aortic arch branch vessel re-entry tears functioned as entries after the graft replacement of the ascending thoracic aorta for the previous type A aortic dissection. Also two large (6 mm) re-entry tears involved the celiac trunk and infrarenal abdominal aorta with additional lumbar artery communicating channels. Therefore, zone 0 to supra-SMA hybrid TEVAR was done, and a tube stent graft was inserted into the infrarenal abdominal aorta to prevent re-entry flow. Patient number 7 had a huge entry tear (at least 24 mm) at the proximal DTA and a 6-mm re-entry tear involving the right renal artery. Zone 1 to supra-celiac hybrid TEVAR was performed initially; however, persistent DTA false lumen flow from the re-entry site of the right renal artery prevented DTA false lumen remodelling. Therefore, additional endovascular treatment with a VIABAHN endoprosthesis was necessary.

We suggest that adjunctive endovascular treatment for retrograde flow and pressurization into the DTA false lumen from the distal re-entry tears and communicating channels, in addition to sealing-off of the entry tear with stent grafts, is mandatory to achieve favorable aortic remodelling in groups B and C patients. These cases have substantially large re-entry tear(s) such that if only the entry tear is treated, having a sealed entry tear with persistent one-way retrograde flow and pressurization from the distal re-entry tear(s) would result in iatrogenic blind sac formation. In the natural history of type B/III aortic dissection cases with partial thrombosis in the false lumen, in which either side of entry or re-entry site flow stasis resulted in blind sac formation, showed a higher mortality rate than cases with a patent false lumen [15 - 17]. Thromboexclusion of the DTA false lumen might be affected by two factors: one is related to the distal extension of the stent graft [3, 5, 18, 19], and the other factor is the flow and pressure from distal re-entries and

communicating channels [6, 14]. Longer thromboexclusion of the DTA false lumen could be achieved by a more distal extension of the stent graft distal to the diaphragm level of the supra-celiac level with CSF drainage [20, 21]. However, retrograde flow and pressure coming from distal re-entry tears and communicating channels should be treated with adjunctive endovascular treatment, which is essential for the ultimate aortic remodelling of the entire DTA false lumen. VIABAHN endoprosthesis for the sealing-off of visceral artery and iliac artery re-entry tears, and AVP/coil for communicating channel re-entries were proposed in this study as adjuncts to endovascular treatment. In cases requiring direct adjunctive endovascular treatment for the visceral re-entry tear and multiple communicating channels, direct obliteration of the false lumen of the thoracoabdominal aorta could be conducted by means of modified stent-graft techniques [22 - 24], and embolotherapy with coils, glue, or occlusion balloons [25]. Studies reported to date show that the result of TEVAR in chronic aortic dissection is unsatisfactory [2 - 8]. The treatment planning for TEVAR and adjunctive endovascular procedures on the basis of anatomic-hemodynamic consideration, as emphasized in this study, would lead to a high rate of favorable aortic remodelling. A favorable aortic remodelling rate was achieved in (87.5%) in this study, which is higher than the reported rate in any other population of patients who underwent TEVAR for chronic aortic dissection, especially CDIIIb aneurysm.

Aortic remodelling failure occurred in two patients after the initial intervention in this study. Patient number 6 in group A underwent prior surgical graft replacement of the ascending thoracic aorta for a previous type A/I aortic dissection. The length of proximal landing zone 0 was short, and there was a raised circular spot at the distal anastomosis that resulted in type Ia endoleak. The other patient with aortic remodelling failure was patient number 16 in group C. The entry tear size was 10 mm, located at the corner of the angulated aortic arch periphery of the flap, and the thoracoabdominal aortic false lumen was large (30 × 25 mm). Only AVP closure of the primary entry tear was attempted as the initial treatment; however, type Ia endoleak occurred because the entry tear was at the periphery of the flap, resulting in incomplete sealing-off with AVP of the primary entry tear. These two patients were rescheduled for re-intervention of the reinforcement with coils inside the false lumen.

Our study has some limitations. Due to the small number of patients, statistical analysis of the false lumen volume reduction was not available in group C. Also the overall growth rate of the DTA false lumen was large but comparison study with CDIIIb aneurysm patients not showing the FTS should follow to know if it is statistically significant.

In conclusion, first, the FTS is a sign representing hemodynamic imbalance in the false lumen, which predicts aneurysmal degeneration and partial thrombosis of the false lumen of the DTA. Therefore, chronic aortic dissection patients showing the FTS with a growth rate faster than 5 mm/year might be indicated for endovascular treatment. Second, understanding the anatomic-hemodynamic consideration would be helpful for the planning of endovascular strategies for the treatment of CDIIIb aneurysm; that is, not only simple TEVAR (i.e., stent grafting in the aortic true lumen that seals-off only the primary entry tear) but also adjunctive endovascular treatment is mandatory to achieve favorable aortic remodelling, especially in cases with considerably large re-entry tears.

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Table 1: Patient characteristics, risk factors, prior surgical history, and computed tomography angiography findings.

Variables	All patients		Subgroup		
	(N = 16)	Entry dominant (A) (n = 7)	Re-entry dominant (B) (n = 6)	Equivalent (C) (n = 3)	
Mean age (years)	59.6 ± 11.9	65.3 ± 11.6	56.7 ± 12.1	61.0 ± 5.3	
Male sex, no. (%)	12 (75)	5 (71.4)	6 (100)	1 (33.3)	
Chronological age of dissection (months)	37.3 ± 24.3	49.8 ± 24.9	35.3 ± 25.5	30.6 ± 24.2	
Comorbidities					
Smoking, no. (%)	6 (37.5)	2 (28.6)	4 (66.7)	0 (0)	
Obesity, no. (%)	2 (12.5)	1 (14.3)	1 (16.7)	0 (0)	
Hypertension, no. (%)	14 (87.5)	6 (85.7)	5 (83.3)	3 (100)	
Diabetes mellitus, no. (%)	2 (12.5)	2 (28.6)	0 (0)	0 (0)	
Marfan's syndrome, no. (%)	0 (0)	0 (0)	0 (0)	0 (0)	
Atherosclerosis, no. (%)	10 (62.5)	3 (42.9)	5 (83.3)	2 (66.7)	
Previous cerebrovascular attack, no. (%)	2 (12.5)	1 (14.3)	1 (16.7)	0 (0)	
Coronary artery obstructive disease, no. (%)	2 (12.5)	0 (0)	1 (16.7)	1 (33.3)	
Peripheral arterial occlusive disease, no. (%)	0 (0)	0 (0)	0 (0)	0 (0)	
Pulmonary disease, no. (%)	0 (0)	0 (0)	0 (0)	0 (0)	
Renal impairment, no. (%)	0 (0)	0 (0)	0 (0)	0 (0)	
Prior aortic surgery					
Ascending aorta replacement, no. (%)	8 (50)	2 (28.6)	4 (66.7)	2 (66.7)	
Total arch replacement, no. (%)	2 (12.5)	0 (0)	2 (33.3)	0 (0)	
DTA or abdominal aorta replacement, no. (%)	0 (0)	0 (0)	0 (0)	0 (0)	
CT findings					
Ratio of the summed area of entry/re-entry tears	-	14.9 ± 6.9	0.08 ± 0.04	0.83 ± 0.16	
Growth rate of DTA false lumen (mm/year)	7.03 ± 6.64	9.56 ± 8.32	5.76 ± 6.67	5.36 ± 3.47	
False lumen aneurysmal change, no. (%)	16 (100)	7 (100)	6 (100)	3 (100)	
False lumen wall partial thrombosis, no. (%)	16 (100)	7 (100)	6 (100)	3 (100)	

Table 2: Thoracic endovascular aortic repair and adjunctive endovascular procedures

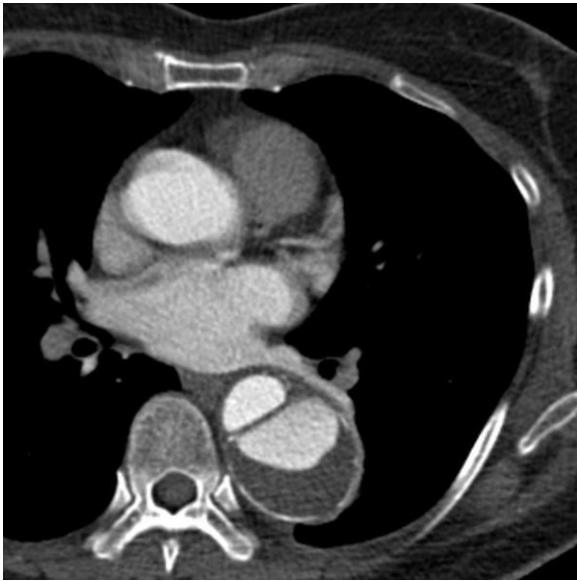
Patient no.	Subgroup	Entry tear treatment	Arch and DTA FLO	TAA FLO	Re-entry tear and communicating channel treatment
1	A	Zone 1 stent graft			
2	A	Zone 4 stent graft			
3	A	Zone 3 stent graft			
4	A	Zone 2 stent graft			
5	A	Zone 3 stent graft			
6	A	Zone 0 stent graft			Abdominal aorta stent graft
7	A	Zone 1 stent graft			RRA VIABAHN
8	B	Zone 3 stent graft			
9	B	Zone 2 stent graft		AVPs	RRA VIABAHN
10	B	AVP		AVPs	LRA VIABAHN
11	B	AVP			ICA Coil, Celiac and RRA VIABAHN, LA L2 and L4 level AVP, REIA VIABAHN
12	B	Zone 3 stent graft	NBCA	AVPs	
13	B	Zone 2 stent graft	AVPs	AVPs	
14	C	Zone 1 stent graft		NBCA	LRA VIABAHN
15	C	AVP	AVPs, Coils	AVPs	LRA VIABAHN
16	C	AVP	AVPs	AVPs	

DTA = descending thoracic aorta, FLO = FL obliteration, TAA = thoracoabdominal aorta, RRA = right renal artery, AVP = AMPLATZER vascular plug, LRA = left renal artery, ICA = intercostal artery, LA = lumbar artery, REIA = right external iliac artery, NBCA = *n*-butyl cyanoacrylate

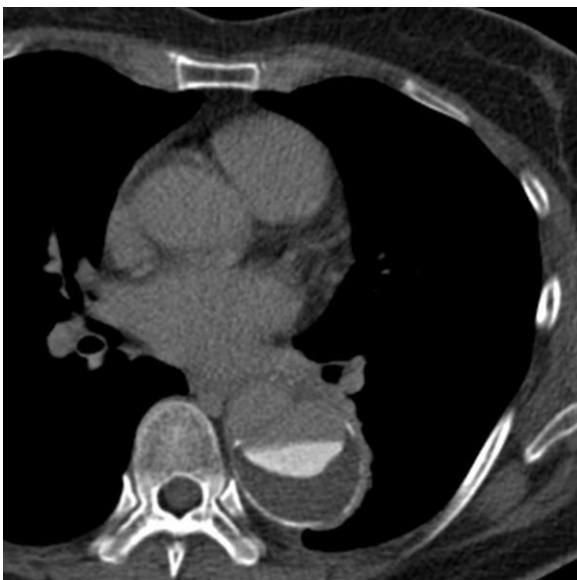
Table 3: Results of thoracic endovascular aortic repair and adjunctive endovascular treatment according to anatomic-hemodynamic considerations

Variables	All patients		Subgroup	
	(N = 16)	A (n = 7)	B (n = 6)	C (n = 3)
Thromboexclusion of entire DTA, no. (%)	14 (87.5)	6 (85.7)	6 (100)	2 (66.7)
False lumen volume reduction, (%)	43.8 ± 23.9	56.3 ± 32.4	33.3 ± 12.7	38.7 ± 8.8
Favorable aortic remodeling, no. (%)	14 (87.5)	6 (85.7)	6 (100)	2 (66.7)

Figure 1: Computed tomography angiography (CTA) axial (a, b) and oblique sagittal (c, d) images of the arterial and delayed phases showing the typical flood tide sign in CDIIIb aneurysm. The delayed phase of the CTA shows blood/contrast level in the false lumen of the descending thoracic aorta (b, d), whereas the arterial phase shows relatively inhomogeneous and weak contrast enhancement of the false lumen compared with the true lumen (a, c). Note the thrombus adhering within the wall of the aneurysmal false lumen.



(a)



(b)



(c)



(d)

Figure 2: Dissection model of the three subgroups and the suggested endovascular treatment strategies. Type A, dominant entry tear and poor re-entry tear(s). Type B, poor entry tear and dominant re-entry tear(s). Type C, entry tear and sum of re-entry tear(s) are equivalent.

TEVAR and adjunctive endovascular treatment strategies	
Group A	Groups B and C
Simple TEVAR is recommended initially in most cases. Adjunctive false lumen and re-entry management might be necessary only in selected patients during the follow-up.	Not only proximal sealing-off of the primary entry tear but also management of the distal false lumen, re-entry tears, and communicating channels are mandatory in all cases. Adjunctive interventions: FLO at the distal thoracoabdominal aorta level with AVPs/coils, closing of branch vessel re-entry with VIABAHN endoprosthesis, blocking of the communicating channel with AVP/coil

TEVAR = thoracic endovascular aortic repair by aortic true lumen stent-graft, FLO = false lumen obliteration, AVP = AMPLATZER vascular plug

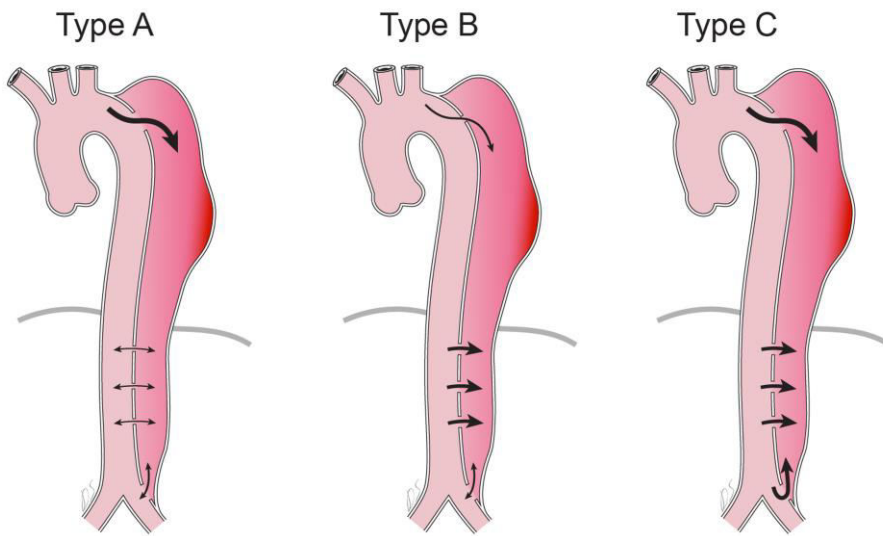
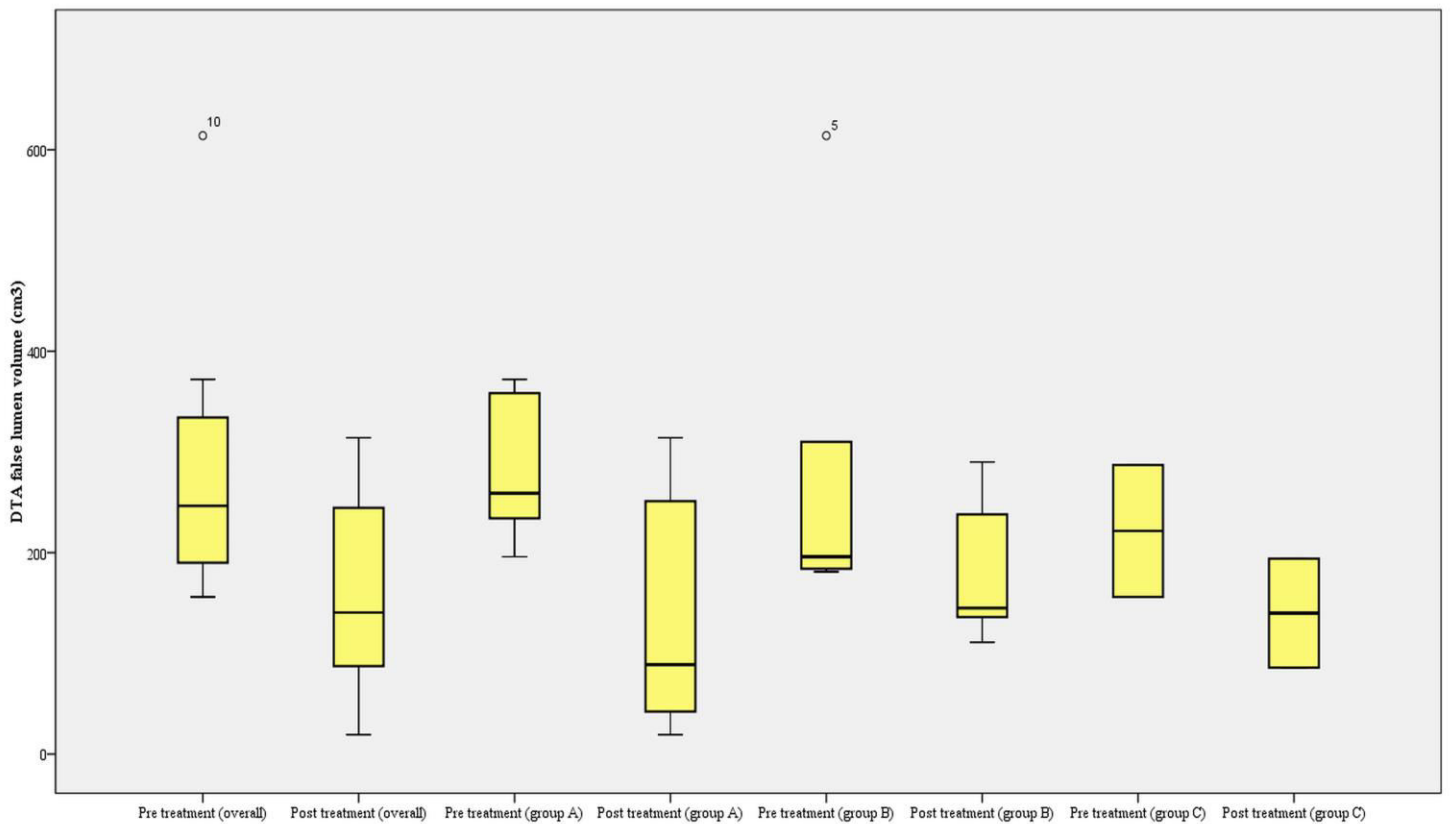


Figure 3: Posttreatment volume reduction of the entire DTA false lumen compared with pretreatment. (Overall, $43.8 \pm 23.9\%$, $p = 0.002$; group A, $56.3 \pm 32.4\%$, $p = 0.043$; group B, $33.3 \pm 12.7\%$, $p = 0.043$; group C, $38.7 \pm 8.8\%$, $p = \text{N/A}$.)



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