Clinical significance of type II endoleaks after thoracic endovascular aortic repair

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Background: **To evaluate the clinical significance of type II endoleaks (ELII) after thoracic endovascular aortic repair (TEVAR).**

Methods: **From January 1997 to June 2012, a total of 344 patients received TEVAR in our institution. ELII was diagnosed in 30 patients (8.7%; 13 males; median age: 65 years, range: 24 to 84 years), representing the study population of this retrospective, single-center analysis. Mean follow-up was 29.5 months (range, 8 months to 9.5 years).**

Results: **Primary ELII was observed in all but two cases (28/30; 93.3%). The most common sources of ELII were the left subclavian artery (LSA; 13/30; 43.3%) and intercostal/bronchial vessels (13/30; 43.3%), followed by visceral arteries (4/30; 13.4%). Overall mortality was 33.3% (10/30). ELII-related death (secondary rupture) was observed in 20% (2/10). Reintervention (RI) procedures for ELII were performed in 9 of 30 patients (30.0%); 5 of 9 (55.6%) in cases with ELII via the LSA. Indications for RI were diameter expansion in five and extensive leakage in four cases. Treatment was successful in five patients (55.6%) but failed in four cases (44.4%). In 12 of 21 (57.1%) untreated patients, ELII sealed during follow-up. In conservatively treated patients, an increase in aortic diameter has been only observed in a patient with secondary ELII.**

Conclusions: **The results presented herein suggest that the clinical impact of ELII after TEVAR must not be underestimated. Albeit a transient finding in most cases, ELII is associated with a relevant RI rate, particularly in cases involving the LSA. RI seems indicated in patients with increasing aortic diameter and/or extensive leakage. Careful surveillance of all patients with ELII is recommended.**

Thoracic endovascular aortic repair (TEVAR) has become the treatment option of choice for a wide variety of thoracic aortic pathologies.[1] Despite the proven short- and midterm safety and efficacy, there is still concern on the durability of this minimally invasive modality. Data from single-center studies show considerable reintervention (RI) rates after TEVAR, ranging from 12% to 22%, notably attributable to progression of the aortic disease and endoleak (EL) formation.[2-4]

Whereas type I and III ELs are regarded as treatment failures and warrant immediate further treatment, type II endoleaks (ELII) are usually considered as benign.[5] In contrast to endovascular repair of abdominal aortic aneurysms, where ELII has been studied in detail, specific data on clinical significance and implications of ELII in TEVAR are limited.[6-8] The aim of the present study was, therefore, to analyze clinical significance and treatment of ELII by reviewing our cumulative TEVAR experience.

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METHODS

Data source. The study design represents a retrospective single-center analysis. All patients who receive TEVAR at the authors' institution are entered into a prospectively maintained departmental database. For the underlying study investigating ELII after TEVAR, the contemporaneously collected demographic, preoperative clinical, and operative data as well as follow-up (FU) information were analyzed and supplemented from patient records and computed tomography (CT) data. An Institutional Review Board approved the study. CT data were interdisciplinary and consensually evaluated at the time of FU before being entered into the database. Computed tomographic angiography (CTA) data of patients diagnosed with ELII were additionally reviewed by an attending vascular surgeon and an attending radiologist in an independent fashion.

Study population. Between March 1997 and June 2012, a total of 344 patients underwent TEVAR for various aortic pathologies. ELII were detected in 30 of 344 cases (30/344; 8.7%; Fig 1). The indications for TEVAR in the entire cohort as well as in the 30 patients diagnosed with ELII are detailed in Table I. The comorbidities and the procedure-related data for the 30 patients with ELII (13 men, 17 women) are summarized in Table II. The median age was 65 years (range, 24 to 84 years). Median FU was 29.5 months, ranging from 8 months to 9.5 years. Operative data are summarized in Table III.

Operative details. The implantation protocol has been previously published.[3] At the authors' institution, simultaneous plug occlusion of the left subclavian artery (LSA) is performed in selected cases, in which an adequate

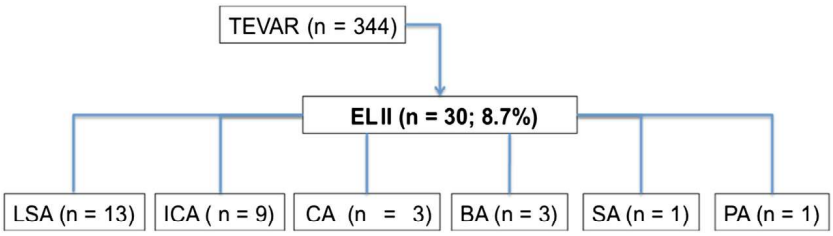

Fig 1. Overview of type II endoleaks (*ELIIs*) observed in a total of 344 thoracic endovascular aortic repair (*TEVAR*) cases. *BA*, Bronchial artery; *CA*, celiac axis; *ICA*, intercostal artery; *LSA*, left subclavian artery; *PA*, phrenic artery; *SA*, splenic artery.

Table I. Indications for TEVAR and ELII distribution

	All patients (N = 344)			*ELII patients (N = 30)*			
	Total, No. (%)	*Elective, No. (%)*	*Urgent/ emergent, No. (%)*	*Total, No. (%)*	*Elective, No. (%)*	*Urgent/ emergent, No. (%)*	*ELII source*[a]
TAA	86 (25.0)	46 (53.5)	30 (46.5)	7 (23.3)	4 (57.1)	3 (42.9)	LSA (n = 3)[2], IC (n = 3), BC (n = 1)
PAU	57 (16.6)	33 (57.9)	24 (42.1)	6 (20.0)	1 (16.7)	5 (83.3)	LSA (n = 3)[1], IC (n = 1), BC (n = 1), CA (n = 1)[1]
TAAA	54 (15.7)	36 (66.7)	18 (33.3)	10 (33.3)	6 (60.0)	4 (40.0)	IC (n = 4)[1], LSA (n = 2)[1], CA (n = 2)[1], SA (n = 1), BC (n = 1)
ADB	79 (23.0)	37 (46.8)	42 (53.2)	3 (10.0)	1 (33.3)	2 (66.7)	LSA (n = 3)[1]
Acute	39 (11.3)	9 (23.1)	30 (76,9)	2 (66.7)	-	2 (100.0)	
Chronic	40 (11.6)	28 (70.0)	12 (30.0)	1 (33.3)	1 (100.0)	-	
AT	22 (6.4)	-	22 (100.0)	-			
IMH	17 (4.9)	8 (47.1)	9 (52.9)	-			
ABF	14 (4.1)	-	14 (100.0)	1 (3.3)	-	1 (100.0)	LSA (n = 1)
ADA	5 (1.5)	-	5 (100.0)	-			
PA	7 (2.0)	4 (57.1)	3 (42.9)	2 (6.6)	2 (100.0)	-	LSA (n =1), PA (n = 1)
ICA	1 (0.3)	-	1 (100.0)	-			
LAA	1 (0.3)	1 (100.0)	-	-			
LSAA	1 (0.3)	1 (100.0)	-	1 (3.3)	1 (100.0)	-	LSA (n = 1)[1]
Totals	344 (100.0)	166 (48.3)	178 (51.7)	30 (100.0)	15 (50.0)	15 (50.0)	

ABF, Aortobronchial fistula; *ADA*, aortic type A dissection; *ADB*, aortic type B dissection; *AT*, aortic transaction; *BC*, bronchial artery; *CA*, celiac axis; *ELII*, type II endoleak; *IC*, intercostal artery; *ICA*, intercostal artery aneurysm; *IMH*, intramural hematoma; *LAA*, arteria lusoria aneurysm; *LSAA*, left subclavian artery aneurysm; *PA*, patch aneurysm; *PAU*, penetrating aortic ulcer; *RI*, reintervention; *SA*, splenic artery; *TEVAR*, thoracic endovascular aortic repair.
[a]The superscript numbers indicate the number of RIs performed for a specific type of ELII in the underlying aortic pathology.

proximal sealing of the endograft covering the LSA is not expected, namely in pathologies involving the origin of the LSA (zones 2 and 3).[9] The LSA revascularization is selectively undertaken, preferably using subclavian-carotid transposition.[10]

The preoperative and postoperative (FU) CTA scans were preferably performed following a standardized TEVAR protocol containing 1-mm slice acquisition of the entire aorta, arterial, venous, and delayed venous imaging phases as well as three-dimensional image reconstructions (Siemens Somatom; Siemens, Erlangen, Germany). FU included medical history, physical examination, and CTA before discharge, 6 months, 1 year, and annually thereafter in uneventful cases. In complicated cases, FU was adjusted accordingly.

Definitions. The definitions used in this study meet the reporting standards for TEVAR published in 2010 by the Society of Vascular Surgery.[11] ELII was defined as retrograde perfusion via branch vessels (aortic aneurysm) or perfusion of the false lumen via the overstented LSA (aortic dissection). In patients with aortic dissection, retrograde flow from distal entry tears were not considered as ELs.[5,11] Primary ELII was defined as an EL present at the implantation or initially diagnosed during the 30-day postoperative period. In contrast, every ELII detected after successful TEVAR and an uneventful perioperative interval was defined as a secondary ELII.[12] RI for ELII was defined as the need to perform additional surgical or interventional procedures to achieve EL sealing. At the authors' institution, the following conditions represent indications for RI: (1) enlargement of the aneurysmal sac (≥5 mm/6 mo) in the setting of an ELII and (2) ELII originating from large-caliber vessels (ie, LSA/visceral arteries) with CT morphologic signs of pressurizing flow (large EL cavity/

Table II. Characteristics of patients with ELIIs (n = 30)

	No.	*%*
Patient population	30	
Median age, years	65	
Age range, years	28-84	
Male sex	13	43.3
Patients ≥ ASA III	27	90.0
Previous cardioaortic surgery	9	30.0
Comorbidities		
Hypertension (arterial)	30	100.0
Smoking	17	56.7
Coronary artery disease	8	26.7
COPD	7	23.3
Diabetes	3	10.0
Renal insufficiency	4	13.3

ASA, American Society of Anesthesiologists; *COPD*, chronic obstructive pulmonary disease; *ELII*, type II endoleak.

Table III. Operative data of patients with ELIIs (n = 30)

Variable	*No. (%) or median (range)*
Treatment period (total no. TEVAR)	
1997-2001 (51)	2 (6.7)
2002-2006 (115)	13 (43.3)
2007-2011 (165)	15 (50.0)
Until 6/2012 (13)	0 (0.0)
Procedural details	
Elective status	15 (50.0)
Urgent status	8 (26.7)
Emergent status	7 (23.3)
Hybrid procedures	
Arch hybrid	6 (20.0)
TAA/A hybrid	5 (16.6)
Combined arch/TAA/A hybrid	3 (10.0)

ELII, Type II endoleak; *TAA*, thoracic aortic aneurysm; *TAAA*, thoracoabdominal aortic aneurysm; *TEVAR*, thoracic endovascular aortic repair.

opacity; Fig 2) in the arterial and delayed venous phase. Diameter expansions are detailed as increase of aortic diameter over time (mm/interval between CTA scans). Expansion was assessed by comparing the CTA before discharge to subsequent FU scans.

Statistical analysis. Data were entered in spreadsheets (Microsoft Excel for PC, 2003; Microsoft, Redmond, Wash) and transferred to PASW Statistics (v. 18.0; IBM Corporation, Somers, NY) for description and analysis. Patient and disease characteristics are described as percentages or median (range). Based on Kaplan-Meier analysis, the log-rank test was used for survival comparisons.

RESULTS

Primary and secondary ELII. The overall ELII rate was 8.7% (30/344; Fig 1 and Table I). Primary ELII was observed in 28 (28/30; 93.3%) cases. Secondary ELII was seen in two patients (2/30; 6.7%). In both cases, the EL originated from an intercostal artery. In the first patient, the EL was observed 2 years after TEVAR for thoracoabdominal aortic aneurysm (TAAA), whereas in the second case, it was detected 10 years after TEVAR for thoracic aortic aneurysms (TAAs).

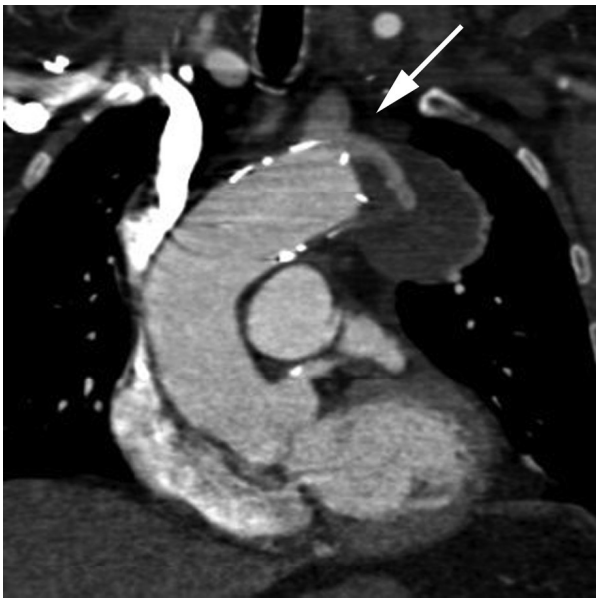

Fig 2. Double-oblique multiplanar reconstruction (MPR) of computed tomographic angiography (CTA) after thoracic endovascular aortic repair (TEVAR) shows a type II endoleak (ELII) originating from the left subclavian artery (LSA) (*arrow*).

ELII via the covered LSA. Coverage of the LSA was performed in 129 of 344 patients (37.5%; Fig 2). Primary LSA revascularization was undertaken in 46 of 129 cases (35.6%). ELII originating from the nonrevascularized LSA was seen in 13 of 83 covered, but nonrevascularized cases (15.6%) and represents the most common single source of ELII in this study (13/30; 43.3%).

The EL-related RI rate in patients with ELII via the covered LSA was 38.5% (5/13), accounting for more than the one-half of all RIs attributable to ELII (5/9; 55.6%; Table IV).

ELII via intercostal, bronchial, and phrenic arteries. Intercostal arteries (n = 9), bronchial arteries (n = 3), and phrenic arteries (n = 1) were responsible for 43.3% of all detected ELII (30.0%, 10.0%, and 3.3%, respectively). RI was required in two cases with ELII originating from intercostal arteries, leading to an RI rate of 22.2% (2/13; 15.4%; Table IV and Fig 3).

ELII via visceral arteries. In four cases (4/30; 13.4%), the celiac trunk or its branches could be identified as a source of ELII (Fig 4). In the first patient, the EL originated directly from the celiac trunk, which had not been ligated during an emergency hybrid TAAA procedure with overstenting of the celiac trunk to expand the distal landing zone. In the second case, equally a hybrid TAAA repair, there was an ELII despite intraoperative ligation of the celiac trunk, most probably because it had been ligated too distally. In the third case, performed under

Table IV. Case-by-case presentation of RIs performed due to ELIIs (n = 9)

Patient no.	*Sex/age, years*	*Diagnosis*	*ELII source*	*Indication*	*RI performed*	*Interval OP-RI, days*	*RI Success*	*FU*
1	Female/49	TAA	LSA	Suspected pressurizing EL	Surgical LSA clipping	12	N	Conversion
2	Female/73	PAU	CT	Suspected pressurizing EL	ST/coiling	3	Y	[c]
3	Female/68	TAAA	CT	Diameter expansion and rupture (12 mm/69 days)	ST/coiling	151	[a]	
4	Male/68	TAAA	LSA	Suspected pressurizing EL	LSA transposition	17	Y	[c]
5	Female/63	TAAA	ICA	Diameter expansion (10 mm/48 months)	Coiling	1461	N	Conservative; [b]
6	Male/78	TAA	LSA	Diameter expansion (5 mm/61 days)	AVP (12 mm)	29	Y	[c]
7	Male/70	LSAA	ICA	Diameter expansion (23 mm/29 months)	BP + prox. TEVAR	870	N	Conversion; [a]
8	Male/58	PAU	LSA	Suspected pressurizing EL	AVP (14 mm)	2	Y	[c]
9	Male/40	ADB	LSA	Diameter expansion (5 mm/6 months)	AVP (16 mm)	244	Y	[c] Type Ia EL

ADB, Aortic type B dissection; *AVP*, Amplatzer vascular plug; *BP + prox*, carotid, carotidal bypass and proximal TEVAR extension; *CT*, celiac trunk; *EL*, endoleak; *ELII*, type II endoleak; *FU*, follow-up; *ICA*, intercostal artery; *LSAA*, left subclavian artery aneurysm; *N*, no; *OP*, operation; *PAU*, penetrating aortic ulcer; *RI*, reintervention; *ST*, sclerotherapy; *TAA*, thoracic aortic aneurysm; *TAAA*, thoracoabdominal aortic aneurysm; *TEVAR*, thoracic endovascular aortic repair; *Y*, yes.
[a]Intraoperative death.
[b]EL still present.
[c]EL resolved.

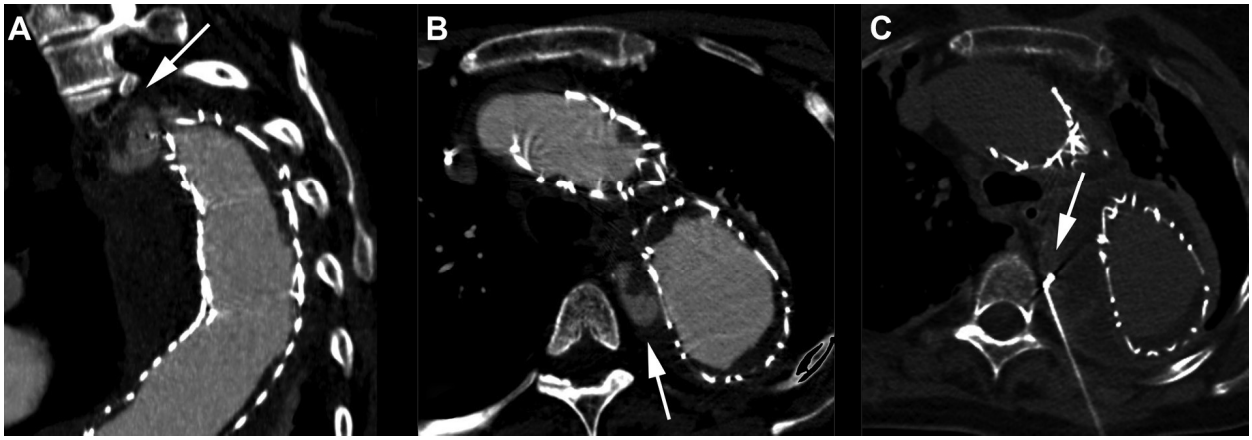

Fig 3. Axial image **(A)** and double-oblique multiplanar reconstruction (MPR) of computed tomographic angiography (CTA) **(B)** demonstrate a type II endoleak (ELII) originating from an intercostal artery (*arrow*). As shown in the axial image in **(C)** the EL was treated by computed tomography (CT)-guided embolization (patient no. 5; Table III).

emergency conditions, the celiac axis had been intentionally overstented after previous visualization of the patent pancreaticoduodenal artery to verify sufficient collateral blood flow.[13] In an additional hybrid TAAA case, ELII via the patent splenic artery was observed, most probably attributable to clip dislocation. Treatment was indicated in two of four patients with visceral ELII, yielding an RI rate of 50% in this subgroup (Table IV).

Mortality. Overall mortality of the entire cohort was 34.8% (120/344), whereas the mortality rate among patients with ELII was 33.3% (10/30). The occurrence of ELII did not influence survival (log-rank test: $P = .651$). Thirty-day mortality was 10.0% (3/30). No perioperative death was EL related. One patient died of multiorgan failure on the ninth postoperative day. Another patient died of pneumonia 26 days after endovascular exclusion of a penetrating aortic ulcer. In a patient who passed away 29 days after TEVAR of a contained ruptured TAA, the cause of death remains unclear.

Long-term mortality was 25.9% (7/27). Four of seven long-term deaths are considered to be procedure-related. At least two of these cases were mediated by ELII:

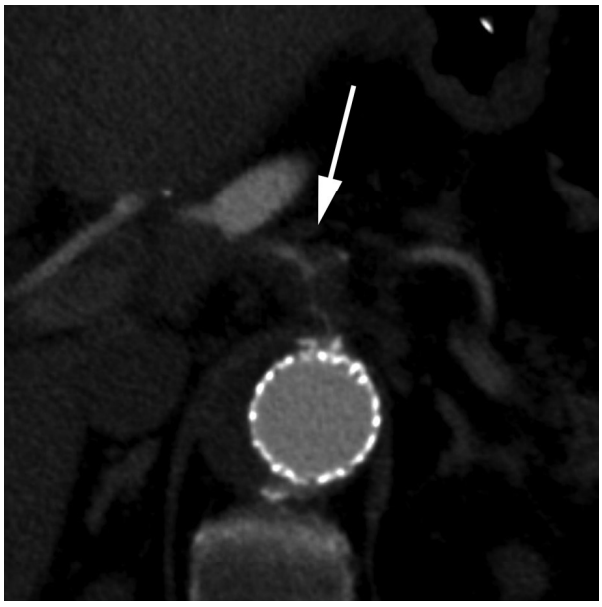

Fig 4. Double-oblique multiplanar reconstruction (MPR) of computed tomographic angiography (CTA) after thoracic endovascular aortic repair (TEVAR) reveals a type II endoleak (ELII) (*arrow*) originating from the celiac trunk.

a 68-year-old female patient (patient no. 3; Table IV) died 151 days after hybrid TAAA repair during her RI procedure attributable to rupture of the aneurysmal sac, which was continuously supplied by an ELII originating from the celiac axis. A 70-year-old patient (patient no. 7; Table IV) died of multiorgan failure after emergent conversion with open descending repair attributable to contained rupture of an aneurysmatic LSA. The pathology had been initially treated by TEVAR but was still perfused via an ELII. A third, 76-year-old female patient died of an internal bleeding 56 months after endovascular TAA exclusion. The patient was known to have an ELII via a bronchial artery; however, FU-CTA did not show sac enlargement.

In the remaining four patients, the cause of death is unrelated to ELII. Instead, causes of death were intestinal perforation, mediastinitis, pneumonia, and stroke (n = 1, each).

RIs for ELII. The RI rate was 30.0% (9/30). In total, 11 procedures (seven interventional RIs, two surgical RIs, and two late conversions) were performed in nine patients. Table IV summarizes the data in a case-by-case fashion. In five of nine patients (55.6%), the indication for RI was diameter expansion. In the four remaining cases (44.4%), RIs were performed because of large EL cavities, suggesting a pressurizing leak (Table IV). The median interval between the TEVAR procedure and the RI was 54 days (range, 3 days to 48 months). Treatment of ELII was successfully undertaken in five of nine cases (55.6%) but failed in four patients (44.4%). Two of these four patients underwent late conversion, yielding a conversion rate of 6.6% (2/30). The first conversion (open aortic arch replacement) was undertaken in 2001 in a patient (patient no. 1; Table III), who had initially undergone TEVAR for TAA repair. In the postoperative CT scan, an extensive ELII via the intentionally covered LSA was detected. Surgical clipping of the LSA was performed 12 days after TEVAR. However, subsequent imaging still demonstrated the ELII. As the patient demanded for definite repair, elective conversion was undertaken 134 days later. The second conversion was emergently performed in a patient with an LSA aneurysm, who had initially undergone TEVAR with simultaneous LSA plug occlusion (patient no. 7; Table III). FU-CTA demonstrated ongoing aneurysm growth (average expansion of 5 mm/6 mo), which was thought to be type Ia EL related. Therefore, the patient underwent carotid-carotidal bypass in conjunction with TEVAR proximalization 29 months after the index procedure. Nevertheless, further imaging studies detected ongoing sac enlargement, supplied by an intercostal artery neighboring the occluder plug. In an interdisciplinary board (vascular surgery, cardiac surgery, and radiology), open distal arch and proximal descending repair was suggested as the treatment option of choice. However, the patient initially refused surgery and did not present until contained rupture had occurred 18.5 months after his RI procedure.

Spontaneous course of ELII. A total of 21 cases (21/30; 70.0%) with ELII were conservatively treated. In 12 of 21 patients (57.1%), the EL sealed spontaneously during FU (LSA: n = 5, intercostal artery: n = 5, bronchial and phrenic artery: n = 1, each). ELII was detectable for a median of 106 days (range, 10 days to 15 months). In none of these patients, CTA revealed an increase of aortic diameter during FU. In the five cases involving the LSA, imaging suggested only marginal leakage. Thus, the patients were conservatively treated.

Six patients (6/21; 28.6%) with an ELII in their last CTA scan died during FU (LSA: n = 3; bronchial artery: n = 1; celiac trunk: n = 2). The EL had been present for a median of 24 days (range, 1 day to 40 months). In one of these patients (TEVAR for contained ruptured TAA), RI was indicated due to a large ELII cavity via the LSA. However, the patient died 29 days after discharge. One patient (1/21; 4.8%) with primary ELII (bronchial artery: n = 1) is known to be alive but lost to FU since discharge from hospital. At the time of discharge, he was not scheduled for RI.

As previously described, there are two patients with secondary ELII (2/21; 9.5%; intercostal arteries in both cases). The first patient (TEVAR for TAAA) is under surveillance with no sac enlargement detected so far. In the second, previously stable, patient (TEVAR for TAA), a 4-mm increase of aortic diameter was detected between the last regular scan and the following scan, depicting ELII (scan interval, 16 months). As the diameter remained stable within the last year of FU, the patient is currently treated conservatively.

Additional EL and RI procedures performed. In the 30 patients diagnosed with ELII, simultaneous occurrence of a type Ia EL and ELII has been observed in three patients. In the first patient (contained ruptured TAA), successful

TEVAR proximalization was performed for the type Ia EL. Regarding the ELII via the LSA, elective RI was indicated. However, the patient died 29 days after discharge. Patient no. 9 (Table IV) had a complex endoleak situation consisting of a type Ia EL in conjunction with an ELII via the LSA. The patient is currently scheduled for type Ia EL repair. Patient no. 5 (Table IV) underwent TEVAR proximalization for a type I EL 171 days after the initial TEVAR procedure (1290 days prior to her RI for ELII via an intercostal artery). Recently, FU-CTA detected a type III EL in the TAA patient with secondary ELII. The patient underwent successful endovascular relining. In a patient with TEVAR for aortobronchial fistula (ELII via the LSA, resolved spontaneously), FU-CTA detected a floating thrombus in the descending aorta, which was covered by stent graft implantation 852 days after the index procedure.

DISCUSSION

The present study shows that ELII is a frequent observation, occurring in about 9% of our TEVAR patients (30/344). Despite the fact that the majority of cases in this series were conservatively managed, we observed an ELII associated RI rate of 30%, thus, illustrating the impact of ELII on clinical practice. Furthermore, the results underline the need for careful monitoring in patients diagnosed with ELII after TEVAR.

Overall EL rates after TEVAR range between 5% and 35%.[8,14,15] ELII after TEVAR is reported to occur in around 5% to 10% of cases, which is in line with our findings.[16-19] Albeit the occurrence of ELII is regularly reported in publications on TEVAR, specific details (ie, anatomic sort, RI rate, type of RIs performed, associated aortic diameter enlargement) allowing for data interpretation are frequently missing. In contrast to the herein presented study, with secondary interventions performed in about every third patient with ELII, the RI rates reported in the literature mostly range below 5%.[4,8,14,20]

In this study, we observed ELII-mediated fatal aneurysm rupture in two cases, both illustrating potential challenges associated with ELII. In the first patient (patient no. 3; Table IV), two consecutive FU scans showed diameter expansion, caused by an ELII via the celiac trunk, which had not been ligated 151 days earlier during a hybrid TAAA repair. The patient was scheduled for elective transcatheter EL embolization but experienced contained rupture before and died of hypovolemic shock during the emergently performed RI procedure. The case underlines the fact that the dynamics of ELII must not be underestimated. Furthermore, it supports the notion that in patients with diameter expansion, a rather aggressive treatment strategy seems to be indicated.

The second patient had undergone TEVAR in conjunction with LSA plug occlusion for an aneurysmatic LSA (patient no. 7; Table III). FU-CTA detected an EL. However, imaging failed to precisely characterize the type of EL (type Ia/c vs ELII). Due to diameter expansion, the patient underwent carotid-carotidal bypass and TEVAR proximalization. As CTA still showed aneurysm growth, the patient underwent diagnostic transbrachial angiography, which ruled out type Ic EL but revealed an ELII in terms of an intercostal artery neighboring the occluder. Open conversion, in general, rarely indicated after TEVAR, was recommended by an interdisciplinary board.[15,21] Unfortunately, the patient did not present until contained rupture had occurred. The case illustrates that characterization of ELII by CTA can be challenging, especially in the aortic arch. If necessary, the use of additional imaging modalities (ie MRA, DynaCT, and/or conventional angiography) is indicated to resolve complex EL situations (eg, the combination of a type Ia and an ELII via the LSA), which we observed in patient no. 9 (Table IV).[22,23]

Coverage of the LSA is necessary to achieve proximal seal in up to 40% of patients treated with TEVAR.[24] Therefore, the risk of ELII and the potential need for RI must be taken into account if coverage of the (nonrevascularized) LSA is performed during TEVAR. Along with others, we believe that ELII via a comparably large-diameter vessel as the LSA, requires special attention.[5,22,25] Besides the two patients with increased aortic diameter, we treated three more patients with ELII via the LSA, in whom early postoperative imaging did not detect diameter expansion (Table IV). In fact, CTA showed a strong contrast enhancement in the arterial and the venous phase suggesting a pressurizing leakage (Fig 2). Nowadays, transbrachial plug occlusion can be favorably used to treat ELII via the LSA.[26] In our series, all vascular plug implantations were successful (Table IV). However, simultaneous occlusion of the LSA during TEVAR to prevent ELII formation is not performed at our institution, as ELII was only seen in around 15% (13/83) of covered, nonrevascularized, cases. In retrospect, plug occlusion would have been also the treatment of choice for the RI performed in patient no. 2 (Table IV). However, in 2001 vascular plugs were not yet available at our institution.

ELII via the celiac axis or intercostal arteries is favorably approached by EL embolization by coils and/or liquid embolic agents (ie, n-butyl cyanoacrylate or ethylene vinyl alcohol).[27-29] The procedure can be performed percutaneously through a transarterial or translumbar/transthoracic approach. In our series, a 67-year-old female with an ELII via an intercostal artery after hybrid TAAA repair was interventionally treated by CT-guided punction of the aneurysm sac and subsequent application of multiple coils (patient no. 5; Table III; Fig 3). The EL located at the distal arch had been persistent for about 4 years and had led to ongoing aneurysm growth (10 mm/48 mo) up to 8.5 cm in diameter. Postinterventional CTA showed shrinkage of the aneurysm sac, justifying a “watchful waiting” strategy.

As in endovascular repair for abdominal aortic aneurysms, there is no data-driven consensus to support any threshold for RI in ELII after TEVAR.[15,30] What we have learned from the cases reported herein is that not only treatment of ELII can be challenging but also visualization and evaluation of RI eligibility. From our experience, progressive diameter expansion in the setting of

ELII should warrant treatment to prevent rupture. Independent of diameter expansion, we advocate a prompt and interdisciplinary RI evaluation in cases of ELII involving the LSA or visceral branches. If CTA suggests a pressurizing leakage, RI should be liberally performed. Regarding small-caliber vessels, such as bronchial or intercostal arteries, we favor a "watchful waiting" strategy, as we and others frequently observed spontaneous thrombosis during FU.[25] In patients with primary ELII, the first FU scan after discharge should be performed within 3 months postoperatively.

To our knowledge, this is the first study directly addressing the issue of ELII after TEVAR. Nonetheless, the study has several limitations besides its retrospective design.

- The herein presented study focuses on clinical impact and management of ELII after TEVAR. Due to the limited number of cases available, the heterogeneity of aortic disease and sources of ELII, a statistical analysis assessing determinants and risk factors for ELII was not performed, limiting the informative value of our results.
- Assessment of EL in aortic dissection is a point of controversy.[11] Continued perfusion of the false lumen is currently considered "a special circumstance that may not be directly analogous to EL." Specific consensus reporting standards for aortic dissection are still missing. According to TEVAR criteria published by the European Association for Cardio-Thoracic Surgery (EACTS), we did not classify retrograde (cross) flow from distal entry tears but only antegrade flow from the LSA into the false lumen as ELII.[5]
- At the authors' institution, CTA is the image modality of choice to diagnose and classify EL. Acquisition of 1-mm slices and multiple phases of imaging are routinely available since the introduction of helical CT scanning in clinical practice in the middle of the last decade. Before that period, CTA might have underestimated the number of ELII after TEVAR. As described beforehand, CTA alone can fail to resolve complex EL situations. With the ongoing improvement of imaging modalities this issue might be further reduced, facilitating EL evaluation and treatment.

In conclusion, ELII is a common finding after TEVAR, which requires interdisciplinary attention. The clinical significance of ELII after TEVAR must not be underestimated, as it is associated with a relevant RI rate. A close FU is mandatory after TEVAR until EL sealing has occurred. Clearly, more data are needed to determine the risk of ELII over time.

AUTHOR CONTRIBUTIONS

Conception and design: MB, PG, DB
Analysis and interpretation: MB, PG
Data collection: MB, PG, DK
Writing the article: MB, PG
Critical revision of the article: PG, AD, DB
Final approval of the article: MB, PG, DK, MME, AD, DB
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