Early and midterm results after endovascular stent graft repair of penetrating aortic ulcers

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Purpose: **To present early and midterm results after endovascular stent graft repair of patients with penetrating aortic ulcers (PAU).**
Methods: **Between January 1997 and March 2008, a total of 202 patients received thoracic aortic endografting in our institution, 48 patients (32 men, median age 70 years, range, 48-89) with PAU. A retrospective analysis of these patients was performed. Thirty-one patients (65%) showed an acute aortic syndrome (8 contained rupture, 23 symptomatic). Follow-up scheme included postoperative computed tomography angiography prior to discharge, at 3, 6, and 12 months, and yearly thereafter. Mean follow-up was 31.3 months (1.3-112.6).**
Results: **Technical success was achieved in 93.7%. Primary clinical success rate was 81.2%. In-hospital mortality was 14.6%. Perioperative mortality was significantly (P = .036) higher in patients with acute aortic syndrome compared to asymptomatic patients (22.5% vs 0%). Postoperative complications occurred in 15 patients (31%), including 2 patients with minor strokes and 6, respectively, 5 patients with cardiac and/or respiratory complications. Early endoleaks were observed in 9 patients (19%), late endoleaks in another 2 patients. Reintervention was necessary in 4 out of 48 patients (8.4%). The actuarial survival estimates at 1, 3, and 5 years were 78% ± 6%, 74% ± 7%, and 61% ± 10%, respectively. There was no aortic-related death during follow-up. Cox regression showed age (hazard ratio [HR]; 1.08, P = .036) and a maximum aortic diameter >50 mm (HR, 4.92; P = .021) as independent predictors of death.**
Conclusion: **Endovascular treatment of penetrating aortic ulcers is associated with a relevant morbidity and mortality rate in frequently highly comorbid patients. Midterm results could prove a sustained treatment success regarding actuarial survival and aortic-related death. Emergencies show a significantly worse outcome, but treatment is still warranted in these symptomatic patients. (J Vasc Surg 2008;48:1361-8.)**

Successful open surgical repair of a ruptured penetrating aortic ulcer (PAU) of the descending aorta has first been reported by Shumacker and King in 1959.[1] Since the 1980s, due to substantial improvements in vascular imaging techniques, increasing information regarding the radiological findings and the natural history of this pathology is available.[2,3] Recently, PAUs have been defined as a distinctive entity of the acute aortic syndrome. Therefore, PAUs gain more and more attention and awareness not only in the surgical community.[4,5]

As symptomatic penetrating aortic ulcers show a poor natural history with rupture rates up to 40%, urgent surgical repair is commonly warranted.[6-8] Currently, poor data concerning the natural course of patients with asymptomatic PAUs is available. The present literature indicates a disease progression with pseudoaneurysm development in approximately 30-50% of these cases but indication for surgical treatment still remains unclear.[9-11] Over the last decade, due to its low morbidity and mortality compared to open surgery, thoracic endovascular aortic repair (TEVAR) has evolved as a first-line treatment modality for thoracic aortic pathologies in many vascular centers.[11] PAUs represent mainly short and localized lesions, frequently in elderly, often high-risk patients which makes them especially suitable and attractive for endovascular repair.[7,12,13] The aim of this study was, therefore, to evaluate early and midterm results of TEVAR and to identify risk factors for early and late death. Additionally, a subgroup analysis of symptomatic vs asymptomatic patients was performed.

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Competition of interest: none.
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METHODS

Patient population. Between January 1997 and March 2008, a total of 202 patients received thoracic aortic endografting (TEVAR) in our institution. This includes 34 patients with penetrating aortic ulcers of the thoracic aorta and 2 patients with concomitant thoracic and infrarenal PAU. In addition, 12 patients received endovascular aortic

Table I. Patient characteristics (n = 48)

Age (yrs)	70 (48-89)
Gender (male:female)	32:16
ASA classification	3 ± 0,4
Additive EuroScore	10 (3-23)
Logistic EuroScore	20.3 (2.7-94)
Hypertension	48 (100)
Smoking history	34 (71)
Diabetes mellitus	13 (27)
COPD	10 (21)
Renal insufficiency	9 (19)
Coronary artery disease	21 (44)
Previous myocardial infarction	17 (35)
Previous cardiac surgery/coronary intervention	13 (27)
NYHA	
I	7 (14)
II	17 (36)
III	5 (11)
IV	1 (2)
Initial presentation with acute aortic syndrome	31 (65)
Previous infrarenal aortic surgery	3 (6)

COPD, Chronic obstructive pulmonary disease; *ASA,* American Society of Anesthesiology; *NYHA,* New York Heart Association; *SD,* standard deviation.
Values are presented as mean ± SD, median (range) or n (%).

repair (EVAR) of an isolated infrarenal PAU in the same period. The total study population consists thus of 48 patients (32 men, median age 70 years, range, 48-89).

Baseline characteristics and cardiovascular risk factors of our patients are given in Table I. For preoperative risk stratification additive, respectively, logistic EuroScore in high-risk patients and the American Society of Anesthesiologists (ASA) classification was used.[14,15] EuroScore is a frequently used preoperative risk stratification system for cardiac and open thoracic aortic surgery. Variables include demographic, cardiac related, and surgery-related variables.[14,15]

The median logEuroScore was 20.3 (2.7-94). ASA classification was III in 79.2% and IV in 14.5% of the patients, which demonstrates a highly comorbid patient cohort. Indications for treatment were aortic rupture in 8 patients (17%), persistent or recurrent pain in 23 patients (48%), and progression of PAU size or morphological aspects (eg, diameter, pleural effusion) in the 17 asymptomatic patients (35%). Median interval between onset of symptoms and the endovascular procedure was 9 days (range, 0-224).

Additional aortic pathologies were present in 16 out of 48 (33%) patients (acute aortic dissection type Stanford B n = 3, intramural hematoma [IMH] n = 7, aortobronchial fistula [ABF] n = 2, thoracic aortic aneurysm [TAA] n = 1, thoracoabdominal aortic aneurysm [TAAA] n = 1, and infrarenal abdominal aortic aneurysm [AAA] n = 2).

Both patients with concomitant AAA received simultaneous open aneurysm repair, using a 10 mm Dacron graft as an iliac conduit for TEVAR. The patient with a TAAA (Crawford type I) and an infrarenal PAU received open surgical repair of the TAAA and staged infrarenal EVAR to minimize the risk of paraplegia. For the same reason, the patient with a TAA and an infrarenal PAU received staged TEVAR/EVAR of both pathologies. Hybrid procedures were performed in 6 out of 48 patients (visceral hybrid procedure n = 2, aortic arch hybrid procedure n = 4). Our experience with hybrid procedures has been published recently.[16,17]

Stent grafts. A total of 58 endografts were implanted. Forty-one patients received a single stent graft (median, 1; range, 1-4). Median length and diameter were 150 mm (95-200) and 34 mm (22-40), respectively. Mean covered aortic length was 154 ± 51 mm.

For TEVAR, 4 types of stent grafts were implanted: 23 Talent/Valiant (Medtronic Vascular, Santa Rosa, Calif), 28 TAG (W.L. Gore & Associates, Flagstaff, Ariz), and 3 Zenith (Cook Inc, Bloomington, Ind). In the infrarenal segment, additional 3-stent graft systems were used: 2 Excluder (Gore), 1 AneuRx (Medtronic), and 1 Lifepath device (Edwards Lifesciences, Irvine, Calif).

Pre-interventional imaging. Endograft sizing was based on centerline diameter measurements from preoperative contrast enhanced CT- or MR-angiography and three dimensional (3D) image reconstructions. For stent graft diameter selection, 15-20% oversizing was applied.

Procedure. All surgical procedures were performed in an operation theater equipped with fluoroscopic and angiographic capabilities (Series 9800; OEC Medical Systems, Inc, Salt Lake City, Utah) and a carbon fiber operating table. For exact visualization of the landing zones in thoracic endograft procedures, the patient's left shoulder was elevated to 40° with both arms fixed beside the body. Each patient received single-shot antibiotic therapy and 3000 units of heparin for anticoagulation.

Forty procedures (83%) were performed under general anesthesia, 4 under spinal/epidural and 4 under local anesthesia. Vascular access was obtained by transfemoral incision in 38 patients; in patients with small femoral arteries (n = 8), a 10-mm Dacron graft conduit was created temporarily to the left common iliac artery. In 2 patients with AAA, an infrarenal bifurcated graft was used as a conduit. The procedure protocol has been published before.[18] For exact stent graft positioning in the aortic arch, adenosine-induced cardiac arrest (AICA) was used in 14 (29%) patients. According to our protocol, AICA is indicated for any pathology located proximal to the left subclavian artery. Nevertheless, it could not be applied in all these cases due to missing application knowledge in some emergency cases.

Completion angiography was performed to assess accurate placement and exclusion of the PAU.

Follow-up. Follow-up schedule included postoperative computed tomographic angiography (CTA) before discharge, clinical examination, plain chest radiography, and CTA/magnetic resonance angiography (MRA) 6 and 12 months postoperatively and annually thereafter (Fig 1). No patient was lost to follow-up. Mean follow-up was 31.3 months (1.3-112.6).

Definitions and statistical analysis. Technical and clinical success were defined according to the reporting standards for endovascular aortic aneurysm repair.[19] En-

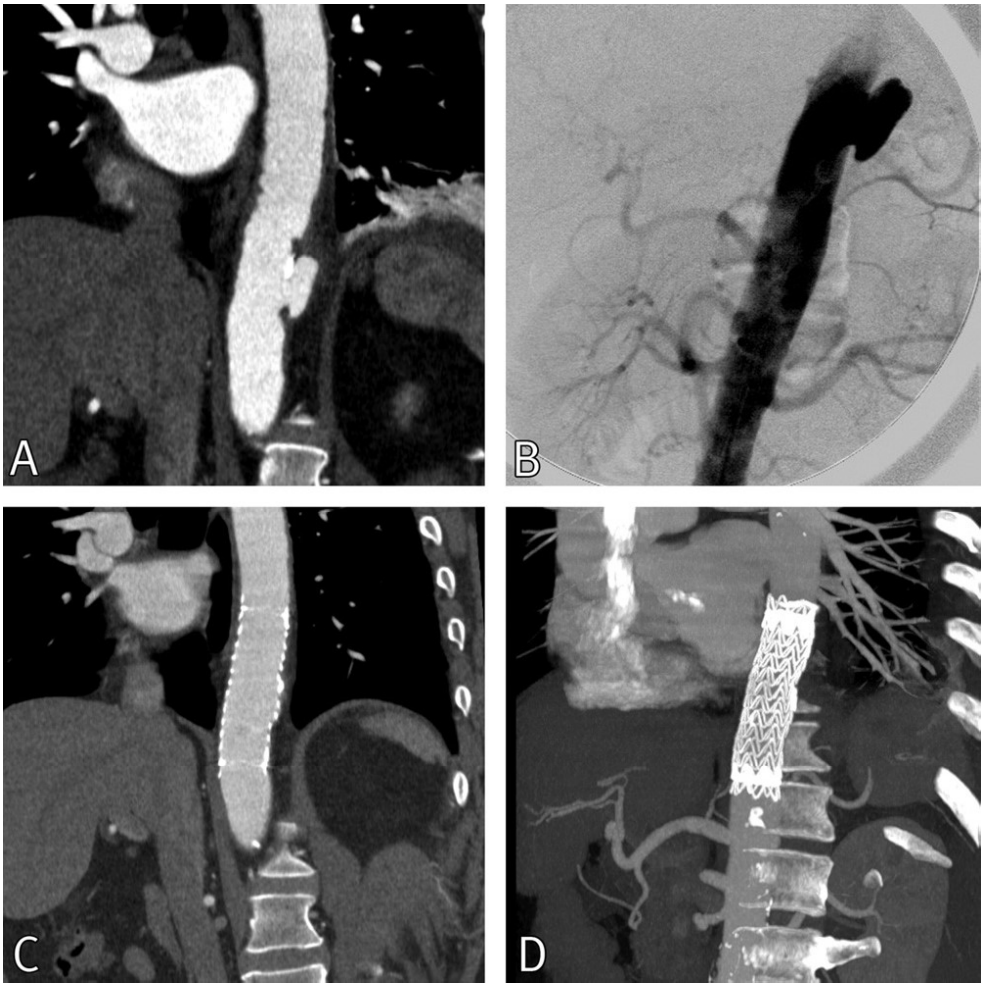


Fig 1. Pre- **(A)**/postoperative **(C** and **D)** computed tomography angiography (CTA) and intraoperative **(B)** angiography showing a successfully treated penetrating aortic ulcer with stent graft repair.

doleaks were categorized as previously described by White et al.[20] Early endoleaks were defined as apparent on intraoperative control angiography or primary postoperative CTA control. Endoleaks occurring during follow-up were defined as late endoleaks. To classify proximal landing zones, the aortic arch map with Zone 0-4 by Mitchell et al was applied.[21] Pathology location and distal landing zones were categorized using a modified (landing zone IVa-c + V) version of our previously described classification (Fig 2).

A retrospective analysis of the prospectively collected data was performed. Data are expressed as mean ± standard deviation (SD) or median (range). Actuarial survival estimate was generated using the Kaplan-Meier analysis. Log rank test was used for survival comparison. For subgroup analysis, Fisher's exact test and Mann-Whitney *U* test were used for categorical respectively continuous variables. All statistical analysis was performed using XLSTAT (Version 7.5; Addinsoft SARL, New York, NY). Cox proportional hazard model (Cox regression analysis) was used to identify independent preoperative risk factors affecting survival (MedCalc Version 9.5.2, MedCalc software, Mariakerke, Belgium). A P value $< .05$ was defined statistically significant.

RESULTS

A total of 56 PAU were found in this patient cohort with 8 patients (17%) having more than one ulcer. Distribution pattern of the pathologies show approximately 50% of the lesions in the aortic arch and proximal descending thoracic aorta (Fig 2). Lesion characteristics show a broad spectrum regarding ulcer depth (mean 23 mm ± 12; range, 5-72) and diameter (mean 22 mm ± 9; range, 9-50) and maximum aortic diameter (50 mm ± 17; range, 27-105) respectively. In 22 of 36 patients (61%) treated with TEVAR, the proximal landing zone was within the aortic arch (Zone 0-3).[21] For distal landing zone extension, covering of the celiac trunk (Zone IVa) without prior revascularization was performed in 2/36 patients (Table II).

Early outcomes. Technical success could be achieved in 45/48 patients (93.7%). Two patients showed an en-

Table II. Proximal and distal landing zone characteristics

Proximal landing zone diameter (mm)	27 ± 4
Proximal landing zone	
Zone 0	2 (4)
Zone 1	4 (8)
Zone 2	6 (12)
Zone 3	10 (20)
Zone 4	14 (28)
Infrarenal	14 (28)
Distal landing zone diameter (mm)	25 ± 5
Distal landing zone	
Zone I	0
Zone II	0
Zone IIIa	20 (40)
Zone IIIb	12 (24)
Zone IVa	2 (4)
Zone IVb	0
Zone IVc	0
Infrarenal aorta	12 (24)
Iliac artery	4 (8)

SD, Standard deviation.
Values are presented as mean ± SD (range), median (range) or n (%).

Table III. Postoperative morbidity and mortality

ICU stay (d)	1 (0-20)
Primary endoleak	9 (18.7)
Type I	2
Type II	6
Type IV	1
Secondary endoleak	2 (4.2)
Type I	1
Type II	0
Type IV	1
Early conversion	0
Late conversion	1
Postoperative complications	15/48 (31.2)
Neurological complications	
Stroke/TIA	2
Paraplegia	0
Cardiac complication	6
Respiratory complications	5
Wound infection	3
Aorto-esophageal fistula	2
Renal failure	2
Bleeding	1
Aortic rupture	1
30-day mortality	3 (6.2)
In-hospital mortality	7 (14.6)
Reintervention	4 (8.3)

Values are presented as median (range) or n (%).

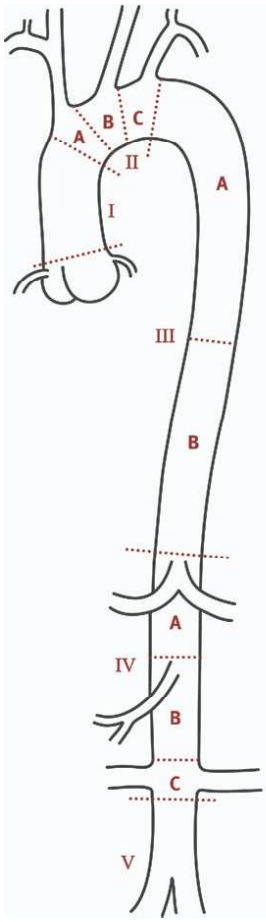
doleak type I at the proximal landing zone (Zone 2) on the first postoperative CTA control, but refused further reinterventions. One of these patients (76 years, ASA 4, logEuroScore 53.5) died 4 months postoperatively of severe pneumonia and the other is under CTA surveillance 6 months postoperatively. One patient (85 years, ASA 4, logEuroScore 94) died 2 days postoperatively after TEVAR of a ruptured PAU as a result of a secondary aortic rupture in the aortic arch proximal to the stent graft. Intraoperative completion angiography had shown no endoleak and the autopsy revealed the stent graft in the correct position with successful exclusion of the PAU. The reason for this rupture remained unclear, manipulation with stiff guidewires or the nose cone of the stent graft might be an unprovable explanation. No early conversion was necessary.

Early endoleaks were observed in 9 patients (19%) (Table III). This includes 2 patients with an endoleak type I described above. Another patient showed a persistent endoleak type II via the celiac trunk and received successful embolization. One patient needed late conversion 11 months after EVAR of an infrarenal PAU due to sac enlargement as a result of a persistent endoleak of unknown origin. Intraoperatively, material fatigue with an endoleak type IV could be verified. An additional 5 patients with an endoleak type II, which all sealed spontaneously, were noticed.

Thirty-day mortality rate was 6.2% (3/48), in-hospital mortality was 14.6% (7/48). The causes of death in these 7 patients were aortic rupture (n = 1), myocardial infarction (n = 1), respiratory complications (n = 1), multi-organ failure (n = 1), and bowel perforation (n = 1). Two patients died of fatal bleeding caused by an aorto-esophageal fistula (AEF). Both AEFs were de novo lesions and not present/symptomatic at the time of stent graft placement. We consider compression of the esophagus by the pseudoaneurysm sac to be the cause, since both patients had large (8.5 cm, respectively, 10.5 cm) concomitant pseudoaneurysms (Fig 3). Fifteen patients (31%) had postoperative complications (Table III). Two patients suffered from a nonprocedure-related postoperative minor stroke, 1 patient on the second postoperative day after an episode of atrial fibrillation, and the other in sequel of a fatal multiorgan failure. No paraplegia was observed. Two patients with preoperative-impaired renal function showed postoperatively a contrast-induced nephropathy requiring temporary dialysis. Cardiac complications included 5 patients with myocardial infarction and 1 with an episode of atrial fibrillation requiring electric cardioversion. Median intensive care unit (ICU) stay was 1 day (range, 0-20 days). Clinical success was achieved in 39/48 patients (81.25%).

Late outcomes. The actuarial survival estimates at 1, 3, and 5 years were 78% ± 6%, 74% ± 7%, and 61% ± 10%, respectively (Fig 2). Causes of late death were lung carcinoma in 2 patients, pneumonia in 2, myelodysplastic syndrome in 1, and 2 patients died for unknown reason at the age of 93 and 87, 3½ years and 2½ years postoperatively, respectively (Fig 4). There was no aortic-related death during follow-up.

Late endoleaks occurred in 2 patients. This includes 1 patient with an infrarenal proximal type I endoleak at 1 year postoperatively which could be sealed successfully by embolization. This infrarenal PAU with a contained ruptured pseudoaneurysm was just below the renal arteries (juxtarenal) with a very short, calcified landing zone and was treated in an emergency procedure with a stent graft that already covered the renal arteries with bare stents. Due to high comorbidities, conversion with aortic cross-clamping was denied and embolization was attempted. A second patient showed a wire fracture and a consecutive endoleak



Localization		n	(%)
I		0	(0)
II		10	(18)
	A	1	(2)
	B	4	(7)
	C	5	(9)
III		29	(52)
	A	14	(25)
	B	15	(27)
IV		3	(5)
	A	3	(5)
	B	0	(0)
	C	0	(0)
V		14	(25)

Fig 2. Localization and distribution pattern of all penetrating aortic ulcer (PAU) (n = 56).

type IV 2 years after initial operation. The patient received successful endorepair with a bridging maneuver. Reintervention (early and late) was necessary in 4/48 patients (8.4%).

Subgroup analysis. Subgroup analysis of patients with acute aortic syndrome vs asymptomatic patients revealed the following results: Mean ASA classification and logEuroScore were not statistically significant in both groups ($P = .331$, $P = .111$, respectively) representing equally distributed risk factors in both groups. All perioperative deaths (in-hospital mortality) occurred in patients with acute aortic syndrome ($P = .036$) but there was no difference regarding midterm actuarial survival in both groups ($P = .457$). All reinterventions were necessary in symptomatic patients, although this was not statistically significant ($P = .163$). Symptomatic patients had a significantly larger maximum aortic diameter (median 52.5 mm vs 40.5 mm; $P = .002$).

Cox proportional hazard regression showed age (HR, 1.08; 95% CI 1.00-1.18; $P = .036$) and a maximum aortic diameter >50 mm (HR, 4.92; 95% CI 1.28-18.92; $P = .021$) as independent predictors of death. Female gender (HR, 1.053; 95% CI 0.35-3.13; $P = .925$) and additional aortic pathologies (HR, 0.319; 95% CI 0.071-1.43; $P = .138$) were not predictors of overall survival.

DISCUSSION

The present study indicates that endovascular stent graft repair in patients with PAU is a minimally invasive treatment option but associated with a relevant morbidity and mortality rate. Midterm results showed a sustained primary treatment success with respect to actuarial survival and aortic-related death. Moreover, symptomatic patients showed a significantly worse perioperative outcome but no difference regarding midterm cumulative survival. In opposition to Gottardi et al, age and a maximum aortic diameter >50 mm proved to be independent predictors for death in our series.[22] Additionally, Demers et al found female gender (HR, 7.4; 95% CI 0.8-72.5; $P = .08$) as an outcome predictor which could not be approved in this series.[13]

The prevalence of PAU among patients presenting with acute aortic syndrome is about 2%-8%.[6,7] Although frequently causing the same symptoms, PAU and classic aortic dissection must be distinguished reliably as they differ regarding their natural course. Rupture rate in symptomatic PAU is reported to be as high as 40% compared to 4% in Stanford type B dissections.[6]

Despite defined potential risk factors for disease progression in symptomatic patients (recurrent/persistent pain, increase of pleural effusion, PAU morphology, and location), rupture still remains unpredictable in patients presenting with PAU.[7] Therefore, an aggressive approach towards surgical treatment, at least in symptomatic patients, is now generally accepted.[12,13,23-26] In opposition to that, Cho et al presented a retrospective series of 105 patients (75% symptomatic) with conservatively managed PAU over a 25-year period.[27] Medical treatment was associated with a significantly lower early mortality rate compared to surgical repair (4% vs 21%) and the authors, therefore, advocated a rather conservative treatment strategy. However, disease progression with development of saccular aneurysms occurred in 48% of the medically-treated patients. Limitations of their study include the large time period (25 years) with significant changes in imaging (only 54/105 patients had serial imaging) and surgical techniques (different devices being used). The median interval (9 days, range, 0-224) between onset of symptoms and the endovascular procedure in our series seems quite long, but is comparable to a series by Demers et al with a mean interval of 17 ± 17 days (range, 6 hours-60 days).[13] Reasons for a delayed treatment in our series included delays in diagnosis, referral to our department, and patient refusal for treatment in an acute setting. If possible, we would aim for stent graft placement within 48 hours after onset of symptoms.

Early mortality after TEVAR in PAU is reported between 0-11% and is thus in line with our results.[13,22,25] Regarding midterm survival, our data confirm 1, 3, and 5-year survival rates of 85%, 75%, and 60%, respectively.[12,13] PAUs frequently represent short, localized lesions, conceptually ideal targets for stent graft repair. This is supported by a 93.7%

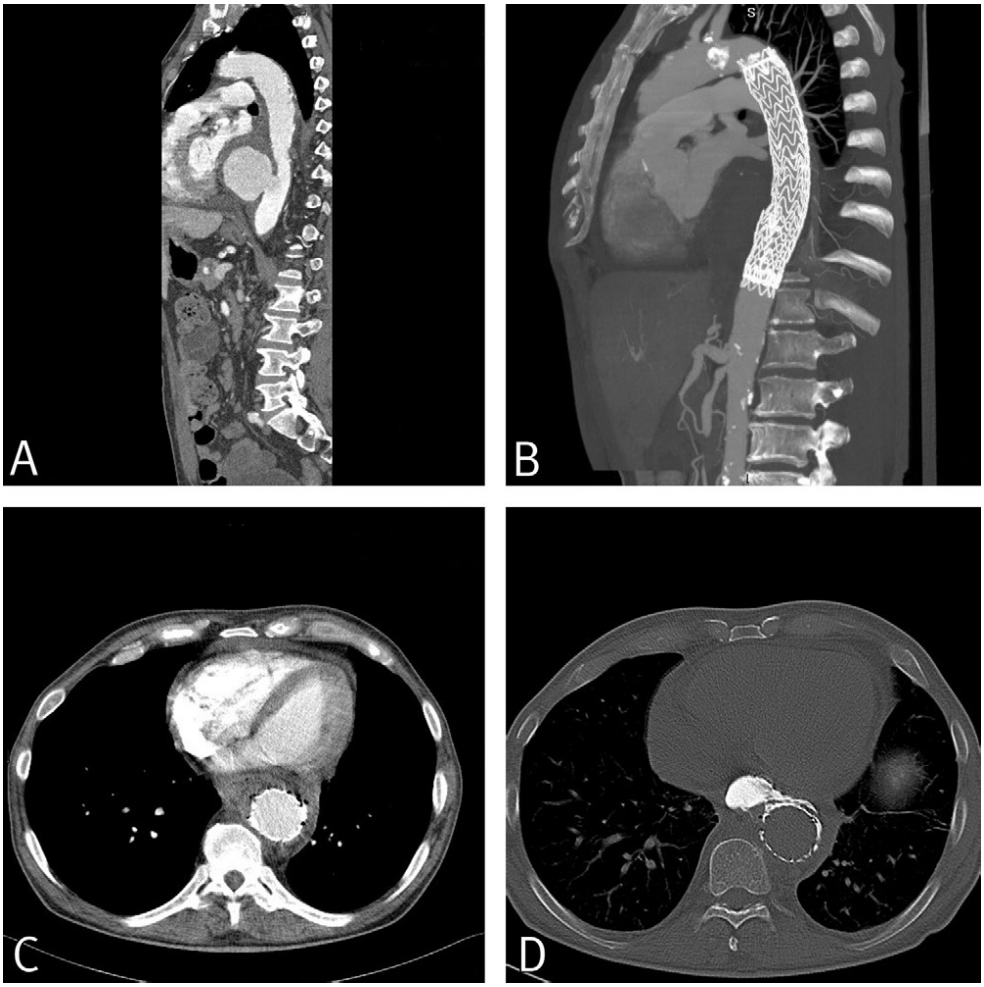


Fig 3. Pre- **(A)** and postoperative computed tomography angiography (CTA) showing a successful exclusion **(B)** of a penetrating aortic ulcer with a large pseudoaneurysm but stent graft infection **(C)** and evidence of an aorto-esophageal fistula **(D)**.

primary technical success rate combined with a relatively low reintervention rate in our and other series.[12,13] Sufficient landing zones remain the keystone of a successful exclusion. In our experience, a minimum of 15 mm seems mandatory. In the distal arch, this distance needs to be measured at the inner curve using centerline measurements. We recommend left subclavian artery debranching in order to avoid type II endoleakage and minimize paraplegia risk.

Neurological complications (minor stroke) occurred in 2 patients, all nonprocedure-related, although periprocedural stroke due to wire manipulation in the aortic arch is well described for TEVAR.[12] No paraplegia was observed in our series of patients. This is especially remarkable as 11/48 patients (23%) were at an increased risk for paraplegia (5× infrarenal AAA repair, 2× infrarenal and thoracic EVAR, 2× visceral hybrid procedures, 1× open TAAA surgery, and 1× TEVAR for TAA) but might be explicable with the short covered aortic length.

The indication for surgical treatment in asymptomatic patients remains debatable, which makes early and midterm results after TEVAR particularly interesting. In our series, asymptomatic patients had a significantly lower in-hospital mortality rate than symptomatic patients (0% vs 22.5%). No differences regarding midterm survival were observed. Our results compare favorably with a 30-day mortality rate of 12% in asymptomatic patients reported by Cho et al.[27] We are not aware of any other reports comparing surgical results of asymptomatic and symptomatic patients with PAU. Cho et al also reported a rupture rate of 4% in asymptomatic patients during follow-up.[27] Furthermore, no data regarding the natural course in exclusively asymptomatic patients with PAU is available. Thus the actual proportion of asymptomatic patients showing disease progression or rupture remains unclear. Series which combine follow-up in conservatively treated symptomatic and asymptomatic patients show aortic dilation and aneurysm formation in about 30-50% which might lead to surgery in a delayed staged.[2,9,10,27] In our series, asymptomatic patients had a significantly smaller maximum aortic diameter. This is of special interest as the development of large

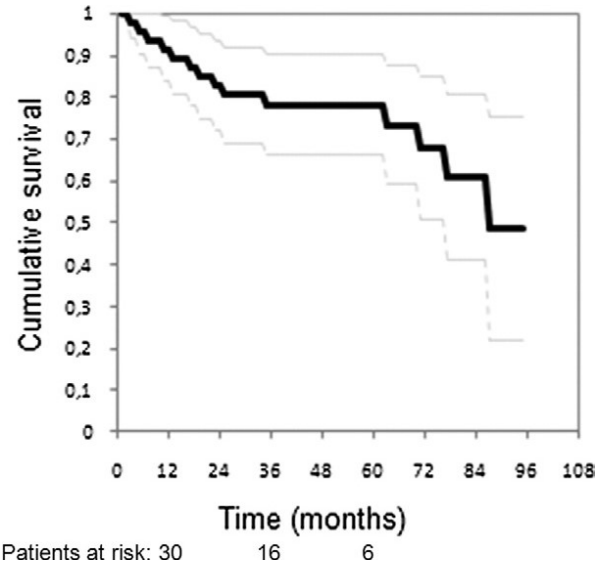


Fig 4. Kaplan-Meier estimate of actuarial survival in all patients (n = 48) treated with thoracic endovascular aortic repair (TEVAR) for penetrating aortic ulcers.

thoracic pseudoaneurysms bares the fatal risk of aorto-bronchial or -esophageal fistulas as seen in 4/48 patients (8.3%) in our series. A recently published series by Brown et al of 115 patients treated with TEVAR for a variety of thoracic pathologies (4 patients with PAU) showed a graft infection rate of 5.2% which was lethal in 4 out of 6 patients.[28]

In our opinion, stent graft repair is also advocated in asymptomatic patients until future studies define risk factors for disease progression and rupture in this subgroup of patients. At present, indication for stent grafting is based on PAU size, CT-documented progression, and morphological aspects of the lesion. Further studies focusing on the natural course of patients with asymptomatic PAUs are, therefore, needed.

Limitations. Although this series represents, to our knowledge, the largest single center study of endovascular-treated PAU, our study is still limited by the relatively small number of patients and its retrospective character. It is an inherent limitation of our approach that we cannot provide information regarding the natural disease course in this cohort. It thus remains unclear which patients benefit most from endovascular treatment. Although the presented mid-term results seem promising, the durability and effectiveness of TEVAR has to be proven during long-term follow-up.

CONCLUSION

Endovascular treatment of penetrating aortic ulcers is associated with a relevant morbidity and mortality rate in frequently high-comorbid patients. Midterm results could prove a sustained primary treatment success regarding actuarial survival and aortic-related death. Endovascular treatment is warranted in symptomatic patients, although these patients are at an increased perioperative risk. General guidelines for treatment of asymptomatic patients with PAU have yet to be defined.

AUTHOR CONTRIBUTIONS

Conception and design: PG, DB
Analysis and interpretation: PG, DB, AD
Data collection: DK, TW, AD
Writing the article: PG, DB
Critical revision of the article: DB, HK
Final approval of the article: PG, DB
Statistical analysis: PG, DB, DK
Obtained funding: Not applicable
Overall responsibility: DB, PG

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