Endovascular Repair of Infrarenal Penetrating Aortic Ulcers: A Single-Center Experience in 20 Patients

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Purpose: To present the early and midterm results of endovascular stent-graft repair in patients with infrarenal penetrating aortic ulcers (PAU).
Methods: Between January 1997 and December 2009, 20 patients (17 men; median age 72 years, range 48–85) with PAU of the infrarenal aorta underwent endovascular repair. In this cohort, 2 (10%) patients had a concomitant PAU in the descending thoracic aorta. Indications for treatment were aortic rupture in 2 patients, persistent or recurrent pain in 3 patients, and progression of PAU size or morphological aspects (e.g., thin wall) in the 15 asymptomatic patients. Follow-up included serial aortic imaging at predefined intervals.
Results: The technical success rate was 100%. In-hospital mortality was 10%, with 2 patients dying of myocardial infarction (MI). Postoperative complications were observed in 5 of 20 patients (4 MIs, 1 case of pneumonia, 1 acute on chronic renal failure, and a lymphatic complication at the access site). Primary endoleaks were observed in 4 (20%) patients and a secondary endoleak in 1 patient. The median follow-up was 22.3 months (range 0.4–104). The actuarial survival estimates at 1, 3, and 5 years were 77%, 69%, and 69%, respectively, with no aorta-related death during follow-up. The reintervention rate during follow-up was 10%, including 1 conversion at 11 months for type III endoleak.
Conclusion: Patients with PAU have a significant number of cardiovascular comorbidities. The endovascular repair of infrarenal penetrating aortic ulcers thus is accompanied by mortality and morbidity caused predominantly by cardiac complications.

Key words: penetrating aortic ulcer, aorta, endovascular repair, stent-graft

Although the first surgical repair of a penetrating aortic ulcer (PAU) was described in 1959, PAUs have only recently come into

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sharper focus for vascular specialists,[1–4] owing to substantial improvements in vascular imaging techniques that have led to the identification of PAU as a distinctive entity of the acute aortic syndrome. The number of asymptomatic ulcers found incidentally on axial aortic imaging has raised questions regarding the natural history and prognosis of PAUs, as well as the indications for and method of treatment.[5–8]

PAUs frequently present as short, localized lesions in rather aged patients with significant comorbidities, which makes them ideal targets for an endovascular approach. The predominant location of PAU is the thoracic aorta; so

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far, only a few case reports and small series have been published on infrarenal PAUs.[2,9–11]

Little data regarding the natural course of (especially asymptomatic) PAUs are currently available, and no consensus therapeutic regimen has been established.[4] Generally, urgent surgical repair is advocated in patients with symptomatic PAU, as these lesions have reported rupture rates up to 40%, especially when accompanied by an intramural hematoma.[12,13] Indications for surgical treatment of asymptomatic patients with PAUs still remain unclear, so the results of surgical or endovascular treatment are particularly interesting. We sought to examine the early and midterm results of endovascular repair of infrarenal PAUs.

METHODS

Patient Population

Between January 1997 and December 2009, 420 patients underwent infrarenal endovascular repair in our institution; among these were 20 patients (17 men; median age 72 years, range 48–85) with penetrating aortic ulcers of the infrarenal aorta (Table), who are the subjects of this retrospective study. Indications for treatment were aortic rupture in 2 patients, persistent or recurrent pain in 3 patients, and progression of PAU size or demonstrative morphological aspects (e.g., sacciform thin wall indicative of impending rupture) in 15 asymptomatic patients. Median interval between onset of symptoms and the endovascular procedure was 1 day (range 0–44) in the 5 symptomatic patients. While 16 patients had isolated abdominal aortic lesions, 4 had additional aortic pathologies [2 concomitant PAUs in the descending thoracic aorta, 1 thoracic aortic aneurysm (TAA), and 1 thoracoabdominal aortic aneurysm (TAAA) with the abdominal PAU near the aortic bifurcation]. These 4 patients received prior open (TAAA) and endovascular (TAA/PAU) repair to minimize the risk of paraplegia, with a 2-stage surgical approach of the asymptomatic abdominal PAUs. In all, 22 PAUs (20 in the abdominal aorta) were found and treated in this patient cohort.

TABLE
Characteristics of 20 Patients With Penetrating Aortic Ulcers

Age, y	72 (48–85)
Men	17 (85%)
ASA classification	3 (2–4)
Hypertension	19 (95%)
Smoking history	13 (65%)
Diabetes mellitus	8 (40%)
COPD	1 (5%)
Renal insufficiency	4 (20%)
Coronary artery disease	8 (40%)
Previous myocardial infarction	6 (30%)
Previous cardiac surgery / coronary intervention	7 (28%)
Initial presentation with acute aortic syndrome	5 (25%)

Values are presented as median (range) or count (percentage).
ASA: American Society of Anesthesiologists, COPD: chronic obstructive pulmonary disease.

Imaging

Stent-graft sizing was based on centerline diameter measurements from preoperative contrast-enhanced computed tomographic angiography (CTA) or magnetic resonance angiography (MRA) and 3-dimensional (3D) reconstructions. A 15% to 20% oversizing was applied. All elective patients received a preoperative cardiovascular workup including electrocardiogram (ECG), stress ECG/echocardiography, and coronary angiography with intervention if necessary to obtain anesthesiology approval for the aortic procedure.

Procedure

All surgical procedures were performed in an operating theatre equipped with fluoroscopic and angiographic capabilities [Series 9800 (OEC Medical Systems, Inc., Salt Lake City, UT, USA) or Axiom U (Siemens, Forchheim, Germany)] and a carbon fiber operating table.[14] Each patient received single-shot antibiotic therapy and 3000 units of heparin for anticoagulation. General anesthesia was used in 15 patients; 2 received spinal/epidural anesthesia, and 3 had local anesthesia. Vascular access was obtained by transfemoral incision in 19 patients, while 1 patient required an 8-mm Dacron iliac conduit, which was then used as an iliofemoral crossover bypass graft. Twenty stent-grafts were implanted: 16 Talent/Valiant/AneuRx (Medtronic Vascular, Santa Rosa, CA, USA), 2 Excluder

(W. L. Gore & Associates, Flagstaff, AZ, USA), 1 Zenith (Cook Medical, Bloomington, IN, USA), and 1 Lifepath (Edwards Lifesciences, Irvine, CA, USA). Aortomonoiliac stent-graft systems with extra-anatomical crossover bypass were used in 6 patients owing to rupture (n=2), distal aortic diameter <20 mm (n=3), or severely calcified, stenotic iliac vessels (n=1). The use of the Valiant prosthesis in this series was for indications outside the recommendations in the manufacturer's instructions for use. Completion angiography was performed to assess accurate placement and successful exclusion of the PAU.

Follow-up included postoperative CTA before discharge, clinical examination, plain chest radiography, and CTA/MRA at 6 and 12 months and annually thereafter.

Definitions and Statistical Analysis

Technical success was defined according to the reporting standards for endovascular aortic aneurysm repair.[15] Endoleaks were categorized as described by White et al.[16] Primary endoleaks were defined as apparent on intraoperative control angiography or the initial postoperative CTA. Endoleaks occurring during follow-up were classified as secondary endoleaks.

Data are expressed as median (range). Actuarial survival estimates were generated using the Kaplan-Meier analysis. $P<0.05$ was considered to indicate a statistically significant difference. All statistical analysis was performed using MedCalc (version 9.5.2; MedCalc Software, Mariakerke, Belgium).

RESULTS

Technical success with exclusion of the abdominal aortic lesion was achieved in all patients (100%). Two (10%) patients died in-hospital: a 74-year-old man with significant comorbidities [history of myocardial infarction (MI) with aortocoronary bypass grafts, stage III chronic renal disease) and symptomatic PAUs of the descending and infrarenal aorta died on day 42 due to a recurrent MI; the other patient (ASA IV) with an asymptomatic PAU died of an MI on day 40.

Postoperative complications were observed in 5 (25%) of 20 patients, including 4 patients with MIs. Among these, the patient who died on day 42 also had pneumonia requiring prolonged ventilation and contrast-induced acute on chronic renal failure requiring temporary dialysis. The other complication was a lymphatic fistula at the access site requiring revision.

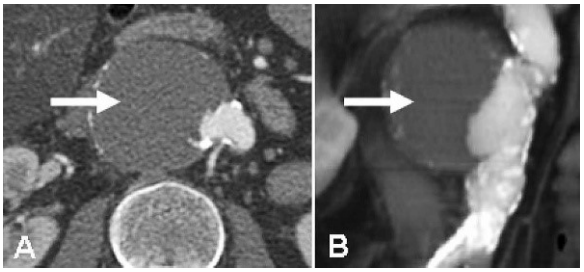

Figure ◆ Computed tomographic angiography showing an infrarenal penetrating aortic ulcer. (**A**) Axial view with extraluminal contrast (arrow). (**B**) Multiplanar reformation (coronal view) showing typical calcifications of the abdominal aorta and the PAU wall, with the contrasted lumen typically located off center, contrary to abdominal aortic aneurysms.

Primary endoleaks were observed in 4 (20%) patients, including 3 type II endoleaks that sealed spontaneously during follow-up. One persistent primary endoleak led to enlargement of the sac; the patient underwent successful open conversion at 11 months after material fatigue sustaining a type III endoleak was identified (5% conversion rate).

Median follow-up was 22.3 months (range 0.4–104), and no patient was lost of follow-up. The actuarial survival estimates at 1, 3, and 5 years were 77%, 69%, and 69%, respectively, with no aorta-related death during follow-up (Figure). Causes of late death in 4 patients were progression of a myelodysplastic syndrome, bronchial cancer, and pneumonia in a patient who required immunosuppression after kidney transplantation. The fourth patient, an 87-year-old man, died of an unknown reason 30 months after endovascular repair.

One patient developed a secondary proximal type I endoleak 1 year postoperatively. At the reintervention, there was no landing zone for a proximal aortic extension, so because of the patient's multiple severe comorbidities, embolization was performed around the proximal landing zone, which excluded the leak successfully. With the conversion noted above, the reintervention rate was 10%.

DISCUSSION

The present study indicates that endovascular repair of infrarenal PAUs has substantial rates of mortality (10%), morbidity (25%), and reintervention (10%). There was also a high rate of MIs in this small cohort. Associated with this was the high atherosclerotic burden (40% coronary artery disease, 30% history of MI), so it seems prudent to recommend that all elective patients should receive a preoperative cardiovascular workup. Very little published data are available regarding the results of endovascular treatment of infrarenal PAU. Recently, Batt and coworkers[2] published a review of 46 patients with infrarenal PAUs (8 patients from their own series, 10 case reports, and 7 series with 2 to 7 patients from 1974 to 2005). Only 9 of these patients received endovascular treatment. There was no perioperative death in these 9 patients, which compares favorably to our series, but the small numbers of patients in both series makes it difficult to interpret these data.

PAUs frequently present as short, localized lesions—conceptually ideal targets for stent-graft repair.[17] Indeed, the 100% technical success rate in this and other series would seem to support this treatment strategy.[9,18,19] Nevertheless, endovascular repair in PAU can be challenging due to heavy calcification of the access vessels and/or the aortic bifurcation, which have to be considered when planning the procedure. Batt et al.[2] described extensive calcifications of the infrarenal aorta in 56% of the reviewed cases. In our series, 4 (20%) patients needed an aortomonoiliac stent-graft system followed by crossover bypass for this reason. Reintervention due to a substantial secondary endoleak in 2 of our patients underscores the necessity for close, lifelong follow-up of these patients.

The 20% rate of concomitant aortic lesions in our cohort seems to be higher than in patients with infrarenal aortic aneurysms (12%),[20,21] probably due to the extensive atherosclerotic disease throughout the thoracoabdominal aorta. We would therefore recommend aortic imaging from the ascending aorta to the femoral arteries in these patients to exclude additional aortic pathologies.

The indication for surgical intervention in patients with PAU remains debatable, and recommendations are mainly based on experience with thoracic PAUs. No doubt exists that PAU in the setting of rupture is a life-threatening condition.[4,22] Although no general treatment regime exists so far, most authors would recommend surgical treatment in patients with symptomatic PAU, as more than a third of these patients could progress to rupture.[3,12,13,23–25] A conservative treatment approach in these patients usually involves surveillance in the intensive care unit, with strict blood pressure control and repeated CTA to exclude further progression.

There is a lack of valid data regarding the natural course of patients with asymptomatic PAU; therefore, there is no consensus regarding the indication for surgical treatment of this subgroup of patients. The question thus remains: Do we treat the large number of PAUs found incidentally on serial aortic imaging? So far, no answer can be given, and further studies to evaluate the natural course of this disease are needed. Wall stress calculations, for example, using finite element analysis may potentially help to predict rupture risk in patients with PAU. Yet, such approaches, though very promising, are still experimental.[26]

Our treatment strategy in asymptomatic patients includes follow-up with serial aortic imaging and workup/treatment of cardiovascular comorbidities. Indications for therapy are based on documented PAU progression, symptoms (e.g., PAU-associated abdominal/back pain), or morphological aspects (e.g., concomitant large pseudoaneurysm) that might signal an impending rupture. Abdominal PAUs treated by an endovascular approach should be included in a rigorous follow-up program just as abdominal aortic aneurysms are because the complications, such as endoleaks or graft migration, are the same.

Conclusion

Endovascular repair of infrarenal penetrating aortic ulcers is associated with relevant mortality and morbidity rates due to predominantly cardiac complications (e.g., MI). There is a lack of data concerning the natural course

of this disease; therefore, surgical treatment in asymptomatic patients needs careful patient selection.

REFERENCES

1. Shumacker HB, King H. Surgical management of rapidly expanding intrathoracic pulsating hematomas. *Surg Gynecol Obstet*. 1959;109: 155–164.
2. Batt M, Haudebourg P, Planchard PF, et al. Penetrating atherosclerotic ulcers of the infrarenal aorta: life-threatening lesions. *Eur J Vasc Endovasc Surg*. 2005;29:35–42.
3. Demers P, Miller DC, Mitchell RS, et al. Stent-graft repair of penetrating atherosclerotic ulcers in the descending thoracic aorta: mid-term results. *Ann Thorac Surg*. 2004;77:81–86.
4. Svensson LG, Kouchoukos NT, Miller DC, et al. Expert consensus document on the treatment of descending thoracic aortic disease using endovascular stent-grafts. *Ann Thorac Surg*. 2008;85:S1–41.
5. Eggebrecht H, Plicht B, Kahlert P, et al. Intramural hematoma and penetrating ulcers: indications to endovascular treatment. *Eur J Vasc Endovasc Surg*. 2009;38:659–665.
6. Erbel R, Alfonso F, Boileau C, et al. Diagnosis and management of aortic dissection. *Eur Heart J*. 2001;22:1642–1681.
7. Quint LE, Williams DM, Francis IR, et al. Ulcer-like lesions of the aorta: imaging features and natural history. *Radiology*. 2001;218:719–723.
8. Vilacosta I, Roman JA. Acute aortic syndrome. *Heart*. 2001;85:365–368.
9. Tsuji Y, Tanaka Y, Kitagawa A, et al. Endovascular stent-graft repair for penetrating atherosclerotic ulcer in the infrarenal abdominal aorta. *J Vasc Surg*. 2003;38:383–388.
10. Harris JA, Bis KG, Glover JL, et al. Penetrating atherosclerotic ulcers of the aorta. *J Vasc Surg*. 1994;19:90–99.
11. Ventura M, Mastromarino A, Cucciolillo L, et al. Abdominal aortic intramural hematoma related to penetrating ulcer: an inappropriate indication for endovascular repair? *J Endovasc Ther*. 2003;10:392–396.
12. Ganaha F, Miller DC, Sugimoto K, et al. Prognosis of aortic intramural hematoma with and without penetrating atherosclerotic ulcer: a clinical and radiological analysis. *Circulation*. 2002;106:342–348.
13. Coady MA, Rizzo JA, Hammond GL, et al. Penetrating ulcer of the thoracic aorta: what is it? How do we recognize it? How do we manage it? *J Vasc Surg*. 1998;27:1006–1016.
14. Weber TF, Tetzlaff R, Rengier F, et al. Respiratory displacement of the thoracic aorta: physiological phenomenon with potential implications for thoracic endovascular repair. *Cardiovasc Intervent Radiol*. 2009;32:658–665.
15. Chaikof EL, Blankensteijn JD, Harris PL, et al. Reporting standards for endovascular aortic aneurysm repair. *J Vasc Surg*. 2002;35:1048–1060.
16. White GH, Yu W, May J, et al. Endoleak as a complication of endoluminal grafting of abdominal aortic aneurysms: classification, incidence, diagnosis, and management. *J Endovasc Surg*. 1997;4:152–168.
17. Schumacher H, Eckstein HH, Kallinowski F, et al. Morphometry and classification in abdominal aortic aneurysms: patient selection for endovascular and open surgery. *J Endovasc Surg*. 1997;4:39–44.
18. Vasquez J, Poultsides GA, Lorenzo AC, et al. Endovascular stent-graft placement for non-aneurysmal infrarenal aortic rupture: a case report and review of the literature. *J Vasc Surg*. 2003;38:836–839.
19. Eggebrecht H, Baumgart D, Herold U, et al. Multiple penetrating atherosclerotic ulcers of the abdominal aorta: treatment by endovascular stent graft placement. *Heart*. 2001;85:526.
20. Crawford ES, Cohen ES. Aortic aneurysm: a multifocal disease. Presidential address. *Arch Surg*. 1982;117:1393–1400.
21. Ruigrok YM, Elias R, Wijmenga C, et al. A comparison of genetic chromosomal loci for intracranial, thoracic aortic, and abdominal aortic aneurysms in search of common genetic risk factors. *Cardiovasc Pathol*. 2008;17:40–47.
22. Melissano G, Astore D, Civilini E, et al. Endovascular treatment of ruptured penetrating aortic ulcers. *Ann Vasc Surg*. 2005;19:270–275.
23. Piffaretti G, Tozzi M, Lomazzi C, et al. Endovascular repair of abdominal infrarenal penetrating aortic ulcers: a prospective observational study. *Int J Surg*. 2007;5:172–175.
24. Dalainas I, Nano G, Medda M, et al. Endovascular treatment of penetrating aortic ulcers: mid-term results. *Eur J Vasc Endovasc Surg*. 2007;34:74–78.
25. Gottardi R, Zimpfer D, Funovics M, et al. Midterm results after endovascular stent-graft placement due to penetrating atherosclerotic ulcers of the thoracic aorta. *Eur J Cardiothorac Surg*. 2008;33:1019–1024.
26. Fillinger MF, Marra SP, Raghavan ML, et al. Prediction of rupture risk in abdominal aortic aneurysm during observation: wall stress versus diameter. *J Vasc Surg*. 2003;37:724–732.