The relevance of aortic endograft prosthetic infection

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Background: **Vascular prosthetic graft infection is a severe complication after open aortic aneurysm repair. Reports of infected endografts are scarce. General treatment consensus with infected graft material is that it should be removed completely. The objective of this study was to describe the incidence of endograft infection after endovascular repair of abdominal (EVAR) and thoracic aortic aneurysm (TEVAR) and to report treatment options and their outcome.**

Methods: **A retrospective cohort study was performed of patients endovascularly operated for abdominal and thoracic aortic aneurysm in two large hospitals (one tertiary referral center and one large community hospital) between March 1996 and June 2009. Diagnosis of infected endograft was made based on clinical findings, blood tests and cultures, imaging studies (computed tomography, fludeoxyglucose positron emission tomography), and intraoperative findings at reoperation.**

Results: **Eleven patients with an infected endograft were identified in 1431 endovascular procedures. One other patient was referred from another hospital. Patients were aged 68 ± 9 years, and all but one were male. The median time from initial TEVAR/EVAR to the diagnosis of infection was 115 days (range, 7-3748 days), with 42% of patients presenting within 3 months after TEVAR/EVAR. Seven patients were diagnosed with endograft infection after elective TEVAR/EVAR and five after emergency TEVAR/EVAR. The incidence was significantly higher in patients that were treated in an emergency setting (0.56% vs 2.79%; $P = .002$), while there was no significant difference between TEVAR and EVAR procedures (1.37% vs 0.77%). All patients were initially treated with antibiotic therapy, which was complemented with surgical intervention in six patients. In four patients, the infected graft material was completely explanted. Isolated microorganisms included *Staphylococcus species* (n = 4), *Streptococcus species* (n = 4), *Enterobacter cloacae* (n = 1), *Escherichia coli* (n = 1), *Pseudomonas aeruginosa* (n = 1), and *Listeria monocytogenes* (n = 1). Median time of follow-up was 201 days (range, 6-2023 days). During the study period, three out of 12 patients died, of which two were treated conservatively (P = ns). At their last follow-up visit, seven of nine patients still used antimicrobial therapy.**

Conclusions: **The incidence of endograft infection is below 1%, with a mortality rate of 25%. Although consensus is that infected graft material should always be removed, this study shows no significant difference in mortality between the conservatively- and the surgically-managed group, possibly related to the small sample size. There may be a role for conservative treatment in selected cases of patients with an infected endograft. (J Vasc Surg 2011;54:327-33.)**

In the last decade, endovascular aortic aneurysm repair (EVAR) has evolved into a widely accepted treatment modality for aneurysms of both the abdominal (AAA) and thoracic (TAA) aorta. Reported complications following EVAR include endoleak, endograft migration, limb occlusion, and rupture. Graft infection following EVAR has scarcely been reported and so far appears to be confined to single cases.[1-8] The incidence of graft infection following open aneurysm repair is reported to be between 0.4% and 3%.[9,10] The minimal invasive character of EVAR may have given rise to the hypothesis that endograft infection is a negligible complication that only very rarely occurs. Follow-up protocols after EVAR, therefore, do usually not include a screening strategy to detect endograft infection. On the other hand, it may be anticipated that the high morbidity and mortality rates, as described with infected grafts after previous open repair,[11] may also be applicable to patients with aortic endografts in situ. General consensus with open surgery is that infected graft material should be completely removed and replaced by autologous material or reconstructed extra-anatomically,[12] as conservative measures usually are associated with poor outcome.[13] Also with endovascular repair, treatment with graft removal has been described, as cases left untreated may even end up in rupture of the AAA.[14,15] One case previously published by our group,[3] however, suggests that there is also a place for conservative treatment with endograft preservation as the patient described is still alive and well after almost 4 years of conservative treatment with antibiotics.

The aim of the present study was twofold; first, to describe the incidence of endograft infection after thoracic endovascular aneurysm repair (TEVAR) or EVAR in a large

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series of patients treated in two hospitals, and second, to report treatment options and their outcome.

PATIENTS AND METHODS

Design of the study. A retrospective two-center cohort study was performed of all patients undergoing TEVAR/EVAR between March 1996 and May 2009 in two hospitals, the University Medical Center Groningen, Groningen, The Netherlands (UMCG) and the Rijnstate Hospital, Arnhem (RHA), The Netherlands, using institutional databases. Information about initial TEVAR/EVAR procedure, underlying disease, demographic aspects, diagnosis, treatment, and follow-up were all studied.

Definitions. Comorbidities were defined as recommended by the Ad Hoc Committee on Reporting Standards.[16] The diagnosis of graft infection was based on a combination of criteria, including clinical findings such as fever and pain, elevated infection parameters, evidence of graft infection on computed tomography (CT) scan, magnetic resonance imaging, leukocytes scan, or ^{18}fluorodeoxyglucose positron emission tomography (FDG PET) scan combined with CT-scan, operative findings (eg, necrosis, purulent fluid, and infected graft material), and isolation of microorganism from either blood, drain material, or the endograft itself. On CT scan, a graft infection was suspected if periprosthetic tissue infiltration and/or fluid- or gas-filled collections were observed. On the FDG-PET, focal pathological uptake was used as a diagnostic criterion. Time to infection was defined as the window between EVAR and the presentation of symptoms that led to the diagnosis of endograft infection.

Follow-up protocol. During follow-up, diagnostic investigations were focused on graft function and migration, endoleak, and structural abnormalities due to failing material. Additionally, serum creatinine was measured every 6 months to assess kidney function. There was, however, a difference in follow-up protocol between the treatment hospitals.

In the UMCG series of patients treated with EVAR, there have been two types of surveillance protocols. Before 1999, the Eurostar protocol was used, including computed tomography angiography (CTA) at discharge, 1 month, 6 months, 1 year, and yearly thereafter. Also, ankle-brachial index, plain abdominal x-ray and duplex ultrasound scanning (DUS) were performed at the same intervals. From 1999, patients with an uncomplicated completion angiography were discharged after plain abdominal x-ray only. DUS was only performed in selected cases (ie, type II endoleak at completion angiogram). Follow-up started with CTA at 1 month, used as a reference for future examinations. Thereafter, patients were followed with abdominal x-ray and DUS at 6 months, 1 year, and yearly thereafter.

For the RHA series, the Eurostar protocol was followed until 1999. From then on, follow-up started with CTA at 6 weeks; thereafter, follow-up consisted of abdominal x-ray and DUS every 6 months. Since 2009, the first CTA at 6 weeks has been replaced by abdominal x-ray and DUS. CTA was only performed when irregularities were found during standard follow-up in either one of the hospitals. These irregularities included the persistence of an endoleak, growth of the aneurysm sac, stenosis of a limb, and changes in the position of the endograft. For the two TEVAR cases, time intervals of follow-up were similar as described above, but thoracic x-ray was performed instead of DUS and abdominal x-ray.

Statistics. The data are presented as mean and standard deviation, unless indicated otherwise. P values $<.05$ were considered to be statistically significant. Differences between categorical variables were tested with Pearson χ^2 test. Statistical analysis was performed using the Statistical Package for the Social Sciences (SPSS 16-0; SPSS, Chicago, Ill).

RESULTS

Subjects. Between March 1996 and May 2009, a total of 1431 EVAR and TEVAR procedures were performed in the two hospitals combined. From these patients, 11 endograft infections were identified. Two of them were treated before by open AAA repair, which accounted for a true primary endograft infection rate of 0.63% of the study population. Another patient with a suspected endograft infection was referred from another hospital for further diagnosis, but was initially treated elsewhere. Patients were aged 68 ± 9 years, and all but one were male. Comorbidity included a cardiac history in seven patients, five were known to have hypertension, and six had a history of chronic obstructive pulmonary disease, of which none used corticosteroids as a standard prescription. Six patients were smokers.

Two patients had been treated for aneurysmal dilatation of the aorta prior to EVAR; in one case, this concerned open repair of a so-called mycotic (ie, infected) aneurysm of the abdominal aorta with autologous deep femoral vein, and the other patient was treated for a ruptured AAA for which open reconstruction with a Dacron prosthesis was performed.

Seven out of 12 patients were originally admitted for elective TEVAR/EVAR (0.56% of total elective cases) and five for emergency TEVAR/EVAR (2.79% of total emergency cases; $P = .002$). Ten procedures were performed in order to exclude an AAA (0.77% of all AAAs) and two for a TAA (1.37% of all TAAs; $P = .46$). Ten were primary procedures, and two were redo-operations. All TEVAR/EVAR procedures were performed in operating theaters with laminar flow. Antibiotic prophylaxis was given to all patients prior to surgery and included cefazolin 1000 mg intravenously in 10 cases and amoxicillin-clavulanic acid 1200 mg intravenously in two cases. The implanted endograft during initial TEVAR/EVAR involved a Talent device (Medtronic, Santa Rosa, Calif) in nine cases and a Cook Zenith device (Cook Inc, Bloomington, Ind) in three cases.

Four patients underwent surgery between initial EVAR and the presentation of symptoms of endograft infection. One of these patients underwent stripping of the great

Table I. Isolated microorganisms and treatment

Patient	*Microorganisms isolated*	*Origin cultured M.O.*	*Antimicrobial therapy*	*Treatment*	*Outcome*	*Therapy at last follow-up*
1	*Staphylococcus aureus*	Blood	Piperacillin/tazobactam	Surgical	Survived	Ciprofloxacin + minocydin
	Staphylococcus epidermidis	Drain/puncture				
	Pseudomonas aeruginosa	Endograft				
2	*Streptococcus milleri*	Blood	Benzylpenicillin + gentamicin	Surgical	Survived	Clindamycin + co-trimoxazole
		Periaortic material/ endograft				
3	None		Amoxicillin/clavulanic acid	Surgical	Survived	Amoxicillin/clavulanic acid + metronidazole
4	*Streptococcus viridans*	Blood	Gentamicin + amoxicillin	Surgical	Survived	None
	Streptococcus constellatus					
	Staphylococcus aureus					
5	*Escherichia coli*	Blood	Clindamycin + piperacillin/ tazobactam	Surgical	Survived	None
		Drain/puncture				
		Endograft				
6	*Enterobacter cloacae*	Blood	Metronidazole + cefuroxim	Surgical	Died	
		Endograft				
7	None		Ciprofloxacin	Conservative	Survived	Ciprofloxacin
8	*Streptococcus constellatus*	Blood	Gentamicin + penicillin	Conservative	Survived	Co-trimoxazole
9	None		Amoxicillin/clavulanic acid + gentamicin	Conservative	Died	
10	*Staphylococcus aureus*	Blood	Flucloxacillin	Conservative	Died	
11	None		Piperacillin/tazobactam	Conservative	Survived	Amoxicillin/clavulanic acid
12	*Listeria monocytogenes*	Drain/puncture	Amoxicillin/clavulanic acid + co-trimoxazole	Conservative	Survived	Co-trimoxazole

saphenous vein and phlebectomy 1 month before presentation of symptoms (Table I; patient no. 12). In another patient, coil embolization for a type II endoleak and extension of a leg of the endograft was performed 5 months before presentation of symptoms (Table I; patient no. 6). The third patient underwent sigmoidal resection because of diverticular disease 6 years before presentation of symptoms and 4 years following EVAR (Table I; patient no. 11). The fourth patient presented with signs of infection 6 months after the implantation of a femorofemoral crossover bypass for an occluded leg of the endograft (Table I; patient no. 5).

Time to infection. The median time from TEVAR/EVAR to the diagnosis of infection was 115 days (range, 7-3748 days). Infection was diagnosed within 30 days after TEVAR/EVAR in 25% of cases, within 3 months in 42%, and within 1 year in 83%.

Diagnosis. The diagnosis of an endograft infection was based on a combination of clinical symptoms and imaging studies, supported by microbial cultures whenever possible. Infection parameters were elevated in all patients at time of diagnosis. Fever was present in eight patients, malaise in three, and nine patients suffered from pain. Other symptomatology included weight loss in two patients and cough, hemoptysis, dyspnea, nausea, vomiting, and rectal blood loss all present in one patient.

Table II. Diagnostic tests performed during work-up of suspected endograft infection

Diagnostic tests	*N (%)*
Clinical findings	
Pain	9 (75)
Fever ($T > 37.5°C$)	8 (67)
Elevated C-reactive protein (>5 mg/L)	12 (100)
Elevated leukocyte count ($>10 \times 10^{\hat{}}9$/L)	9 (75)
Imaging	
Computed tomography	12 (100)
FDG-PET	9 (75)
Leukocyte scan	3 (25)
Bacterial cultures	
Blood	10 (83)
Computed tomography-guided drainage	2 (17)
Wound drainage	1 (8)

FDG-PET, Fludeoxyglucose positron emission tomography.

A CT scan was performed in all cases (100%) to aid in the diagnostic process, a FDG-PET in nine (75%), and a leukocyte scan in three cases (25%). Blood samples were cultured in 10 cases. In one case, this was complemented with cultures from drain fluid that was left in situ after

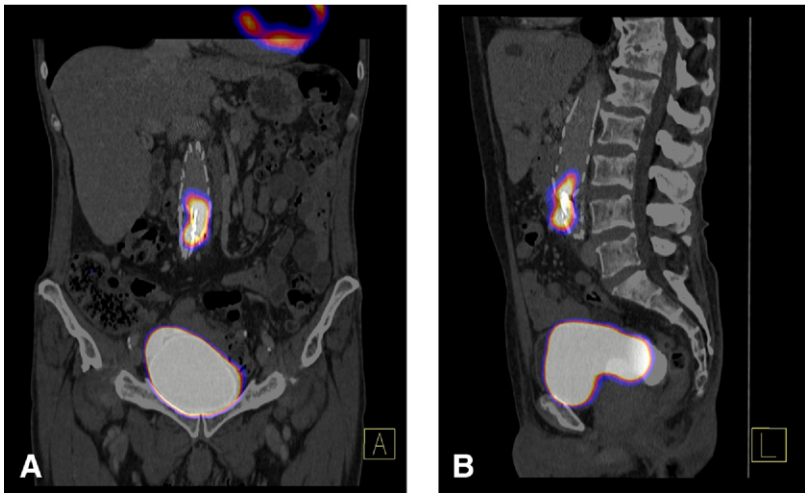

Fig 1. Coronal **(A)** and sagittal **(B)** views of fused FDG-PET-CT imaging of an infected aortic endograft. Clearly, a hot spot at the site of the overlap zone between body and endograft limbs is visible.

CT-guided puncture and, in another case, samples of drain fluid from a wound drain were cultured. In one patient, only a CT-guided puncture with subsequent drainage provided a culture, and in another patient, no cultures were drawn. Table II shows an overview of used diagnostic tests and their frequencies. Microorganisms were isolated in eight out of 12 patients during the diagnostic process and/or treatment. Table I lists the isolated microorganisms, the combinations in which they were found, and the subsequent antimicrobial therapy.

Staphylococcus aureus was the most frequently isolated bacteria (n = 3). During treatment for the infection or after surgical intervention, additional microorganisms were isolated in two of eight patients. In one of these two patients, *Staphylococcus epidermidis* and *Pseudomonas aeruginosa* were found intraoperatively, while in the other patient, *Streptococcus constellatus* and *Staphylococcus aureus* were found in blood cultures during the course of antimicrobial therapy. In the four patients in which all bacterial cultures remained negative, the diagnosis was based solely on clinical findings and imaging modalities (Table I; patient nos. 3, 7, 9, and 11). These cases presented with abnormalities on CT scans and/or FDG-PET scans (eg, periaortic gas, fluid collections, hot spots on FDG-PET; Fig 1). Combined with clinical symptoms, the diagnosis of endograft infection was most likely, and therefore they were treated as such.

Treatment. After the diagnosis of endograft infection was made, one patient, diagnosed with low-grade infection based on elevated laboratory infection parameters, periaortic gas on CT, and a hot spot at the site of the prosthesis on FDG-PET, was treated in the outpatient clinic with antibiotics. The remaining 11 patients were admitted for treatment. In total, six patients were treated with antimicrobial therapy only, guided by bacterial cultures whenever possible. Only one of these patients was unfit for surgery due to his pulmonary condition. The third column of Table I shows the antimicrobial therapies given to the various patients during admission. Patients in whom cultures turned out negative were treated with broad-spectrum antibiotics.

In the other six patients, antimicrobial therapy was followed by surgical intervention. In one of these cases, following TEVAR of a ruptured TAA for a suspected infected aneurysm, a secondary EVAR was performed with the original endograft in situ, using a Gore Excluder device (W. L. Gore, Flagstaff, Ariz) to seal the proximal rupture. Postoperatively, the patient was treated with intravenous antimicrobial therapy for 8 weeks, after which the patient was discharged with oral antibiotic therapy (amoxicillin). In another patient, the operative risk for removal of the infected endograft and open reconstruction was considered too high. It was decided to perform a laparotomy to rinse the infected area. During the procedure, duodenal erosion was found, which was oversewn and covered using an omental flap. The patient was treated postoperatively with benzylpenicillin and gentamicin intravenously.

In the remaining four patients, open reconstruction with removal of the endograft was performed. In one case, the abdominal aorta was reconstructed using an aortobi-iliac silver-coated polyester prosthesis. In another patient, who had previously undergone a femoral-femoral crossover because of an occluded right leg of the endograft, a left-sided axillofemoral bypass was performed using a 6 mm polyester prosthesis before the endograft was removed during subsequent laparotomy.[17] During this procedure, it was discovered that part of the small intestine adhered to the right common iliac artery, uncovering a small defect when detached. The defect was closed, after which the endograft was completely removed. In another case, explantation of the endograft was followed by reconstruction using autologous deep femoral vein (Fig 2). In the last case, explantation with subsequent reconstruction with com-

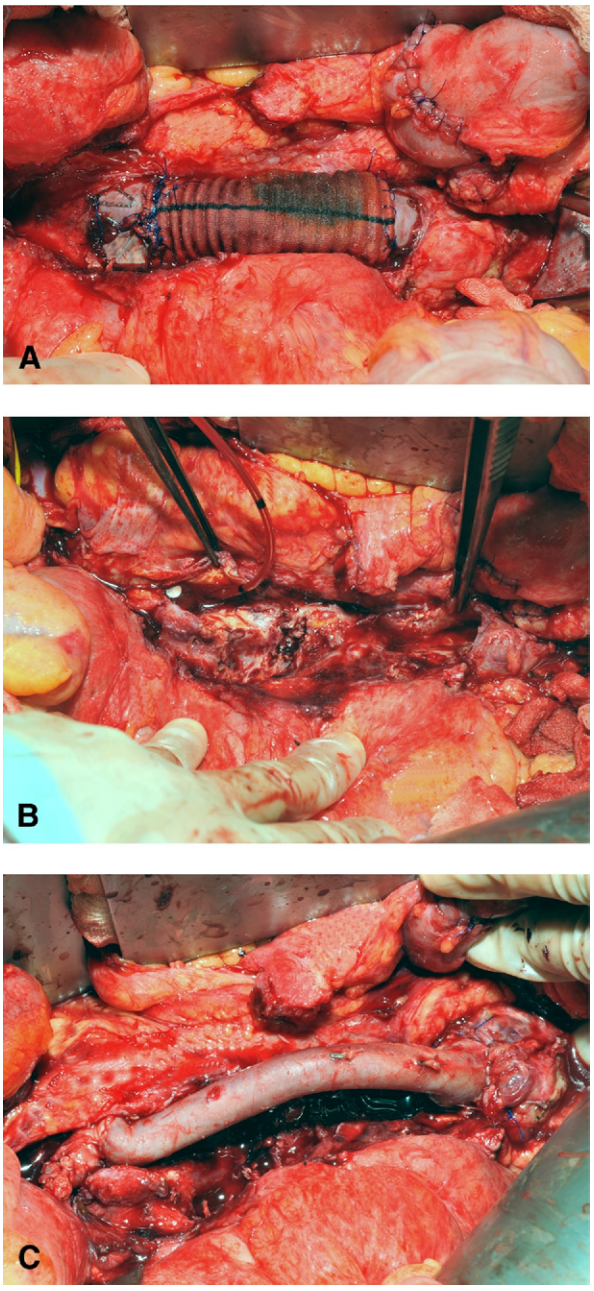

Fig 2. Intraoperative findings during reoperation after previous polyester (tube) prosthesis and right aorto mono-iliac endograft with femorofemoral crossover bypass. **A**, Completely disconnected anastomoses of the polyester prosthesis from the aortic wall both at proximal and distal ends (head of patient to the *right*). The *brownish* area at the ventral side of the prosthesis indicated the marks of an aortoduodenal fistula; blood flow was secured only by the endograft. In the *right upper corner*, the suture line of duodenal closure is depicted. **B**, Operation area after explantation of both polyester prosthesis and endograft. Proximal aortic wall (on the *right*) and wall of the right iliac artery (on the *left*) are marked by tweezers. **C**, Intraoperative view after reconstruction with autologous superficial femoral vein.

bined autologous vein and a silver-coated polyester prosthesis and a femorofemoral crossover bypass was performed.

Follow-up and outcome. All patients (n = 1431) were followed-up after discharge. At 1 year, 15% (217/1431) of patients were lost to follow-up. From the remaining cohort, 86% (1050/1214) were alive, and 14% (164/1214) had died. After 5 years, 23% (332/1431) were lost to follow-up, and 433 patients did not yet reach 5-year follow-up because they had been treated within the past 5 years. From the remaining cohort, 48% (323/666) were alive at 5-year follow-up, and 52% (343/666) had died. Due to the low incidence of infected endografts, there was no strict protocol for surveillance of infected endografts. Follow-up is mainly based on the clinical performance of the patient and preference of the treating physician. The median time of follow-up among the patients with endograft infection was 201 days (range, 6-2023 days). The patient with 6 days of follow-up only died at that time. During their last follow-up visit, seven of nine patients still used antimicrobial therapy (Table I). During the course of treatment and follow-up, three of the 12 patients died (25%). Two of these patients were treated conservatively with antimicrobial therapy only, and one was treated with open surgery (P = ns).

One of the deceased patients presented with hypotension and increased heart rate 1 day after being discharged following primary EVAR. He was readmitted for antibiotic treatment but deteriorated during the course of a week and died 6 days after readmission due to graft-related sepsis. The second patient died 130 days after being admitted for graft infection, 150 days after primary TEVAR. CT scan showed a new aneurysmal dilatation of the thoracic aorta, proximal of the endograft. A hot spot on FDG-PET scan confirmed the diagnosis of an infected endograft. The patient was treated intravenously with amoxicillin/clavulanic acid and gentamicin, seemed to recover gradually, and was discharged with oral antibiotic therapy (amoxicillin/clavulanic acid). Eventually, the patient died of aortobronchial fistula, which was found during autopsy.[18] The third patient presented with pain and malaise, and the FDG-PET scan showed illumination of periaortic tissue. CT scan was repeated 1 week later and showed a leaking aneurysm. Emergency explantation with in situ reconstruction using an aortobi-iliac silver-coated polyester prosthesis was performed, and the patient seemed to recover gradually. However, infection parameters remained elevated, and the patient remained septic. The patient died 145 days after original admission for endograft infection; no autopsy was performed.

DISCUSSION

In the present study, we have shown that the incidence of endograft infection following TEVAR/EVAR is below 1%. The observed incidence is in accordance with previous estimations. Heyer et al reported incidences of prosthetic infection of 0.3% following EVAR and 4.8% following TEVAR.[19] Sharif et al noted an incidence of infection of

0.6%.[20] Ducasse et al performed a retrospective multicenter analysis that showed 0.4% infected endografts on a total of 9739 endovascular treatments.[21] In our series, 42% of patients presented with signs of infection within 3 months after TEVAR/EVAR. In two of five patients presenting with symptoms within 3 months after EVAR, *Staphylococcus aureus* species were isolated. *Staphylococcus aureus* is commonly present on the skin and has been reported to be the most frequently isolated microorganism in endograft infections.[21] The short lag time to infection emphasizes the role of prophylactic antibiotics during surgery. Antibiotic prophylaxis in vascular surgery has been proven beneficial to reduce surgical site infections after reconstruction of the aorta, procedures on the leg that involve a groin incision, any procedure that implants a vascular prosthesis or endoluminal graft, and lower extremity amputation for ischemia.[22,23] The given prophylactics in this series were either cefazolin or amoxicillin/clavulanic acid. Microorganisms isolated in patients who presented with symptoms of infection within 3 months after TEVAR/EVAR were all sensitive to the given prophylactics. Additional surgical procedures may pose an increased risk for infection of the inserted endograft.[19] In this series, four out of 12 patients underwent additional surgery following EVAR. Three of them underwent procedures that are considered to be clean procedures, while the other underwent colonic surgery. Unfortunately, the current study group is too small to draw conclusions on the possible indication, dosage, and duration of antibiotic prophylaxis in patients following TEVAR/EVAR that need other surgical procedures.

Due to extensive variation in presenting symptoms, it may be difficult to diagnose the endograft infection. In this series, the diagnosis was made based on several factors, including clinical findings, cultures, and imaging studies. A CT scan was performed in all cases to solidify the diagnosis; FDG-PET was used in 75% of cases and proved a useful diagnostic tool, especially in cases in which clinical findings pointed toward infection, but no microorganisms were isolated from cultures. Microorganisms were isolated in eight out of 12 patients, which may raise the question whether the diagnosis of endograft infection was appropriate in the four other cases. Nevertheless, the combination of other diagnostics provided sufficient evidence to support the diagnosis of an infected endograft. All presented with elevated infection parameters, while three of these four patients had clinical complaints, including pain and fever. CT scan showed abnormalities, such as periaortic gas and fluid around the prostheses, and the FDG-PET scan showed an increased uptake at the location of the prosthesis in all four cases. The usefulness of an FDG-PET scan in the diagnosis of a graft infection has been previously described.[24,25]

Aortic endograft infection is associated with high morbidity and mortality rates. Heyer et al reported a 30% mortality in 10 cases with an infected endograft, with 75% mortality in four (40%) conservatively treated patients. Sharif et al reported a 50% mortality in six cases with an infected endograft, with 100% mortality in two (33%) conservatively treated patients. Ducasse et al described 18% mortality in 65 patients with endograft infection, with 36% mortality in 11 (17%) conservatively treated patients. In our series, overall mortality was only 25% in 12 cases, with 33% mortality in six (50%) conservatively treated patients. General consensus is that infected graft material should be completely removed and that conservative treatment should be reserved for patients who are unfit to undergo surgery. In this series, however, 50% of patients were treated conservatively.

Furthermore, in only four of 12 cases was the infected graft material completely removed. These observations raise the question whether it is always necessary to remove all infected material and suggest that there could be room for conservative treatment in selected cases, even if the patient is fit enough for surgical intervention. On the other hand, it is more than likely that patients with worse infections were treated by graft excision and that those with more minor infections were treated with graft preservation, rendering any conclusions inconclusive. Adequate patient selection and monitoring of therapy might improve results of conservatively-treated patients.

The retrospective design of our study has several drawbacks. Due to the design, the decision-making process was not always very clear. A prospective registration on infected endografts may well give additional information considering the outcome of various treatment strategies. Due to the low incidence of graft infections, the power of our data may be limited. The number of treated patients with an infected endograft was too small to draw any conclusions on actual survival rates. Moreover, the minor difference in incidence between EVAR and TEVAR did not reach statistical significance, due to the low number. However, this observation is in accord with previous findings of Heyer et al.[19]

CONCLUSIONS

In conclusion, endograft infection is a rare complication after TEVAR/EVAR associated with high mortality rate. A large percentage of cases present with symptoms within 3 months after TEVAR/EVAR (42%), and the incidence is significantly higher following emergency TEVAR/EVAR. In our series, there was no significant difference in survival between surgically and conservatively treated patients, possibly related to the small sample size.

AUTHOR CONTRIBUTIONS

Conception and design: PC, MR, CZ
Analysis and interpretation: PC, MR, CZ
Data collection: PC, MR, CZ
Writing the article: PC, MR, CZ
Critical revision of the article: IT, SS, JD
Final approval of the article: PC, MR, IT, SS, JD, CZ
Statistical analysis: PC, MR, CZ
Obtained funding: Not applicable
Overall responsibility: CZ

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