

Endarterectomy of the Aneurysm Sac in Open Abdominal Aortic Aneurysm Repair Reduces Perigraft Seroma and Improves Graft Incorporation

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Abstract

Background Fluid around the graft in the original aneurysm sac after open abdominal aortic aneurysm (AAA) repair is a poorly researched phenomenon. If large, such perigraft seroma can cause symptoms of compression, and cases of rupture have even been described. We assessed whether endarterectomy of the aneurysm sac reduces the incidence of perigraft fluid and improves graft incorporation.

Design and methods Starting in July 2005, all patients with elective open AAA repair were alternately treated either with conventional thrombectomy or thrombectomy plus endarterectomy of the aneurysm sac. All patients were treated with a polytetrafluoroethylene (PTFE) graft. The maximum axial width of the perigraft fluid collection was measured on computed tomography (CT) scans 1 year after operation.

Results The CT scans of 115 patients were available; 56 had endarterectomy of the aneurysm sac and 59 did not. Fluid collections were significantly smaller in patients with endarterectomy (median width 4.0 versus 8.0 mm; $P = 0.0001$). Eight patients with endarterectomy had a fluid collection wider than 10 mm compared to 28 patients

without endarterectomy (OR 0.18, 95% CI 0.07–0.46). After endarterectomy, 17 patients had radiological signs of complete graft incorporation in comparison to only 6 patients without endarterectomy (OR 3.85, 95% CI 1.39–10.66). No patients were symptomatic or reoperated for perigraft seroma.

Conclusions Endarterectomy of the aneurysm sac in open AAA repair appears to improve graft incorporation. The high rate of asymptomatic perigraft seroma is surprising, and its clinical significance is unknown. Ultrafiltration of PTFE grafts may be an underlying mechanism.

Introduction

Serous fluid collections around the prosthesis in the original aneurysm sac (perigraft seroma) after open AAA repair are a poorly researched phenomenon. To date there is no literature on how often fluid collections around the graft occur, what extent of fluid collection can be considered normal, what natural course perigraft seromas take, or what clinical importance they have. Symptomatic perigraft seroma after open AAA repair is generally considered to be quite rare and only individual case reports exist [1–6]. The condition generally comes to be diagnosed because of symptoms of compression, but even cases of ruptured perigraft seroma have been described [7].

Our clinical experience suggested that asymptomatic serous fluid collections may be quite common. Asymptomatic perigraft seroma may be relevant too, because fluid around the prosthesis is a sign of failure of graft incorporation. We hypothesized that endarterectomy of the aneurysm sac at the time of open AAA repair—and thus removal of the atherosclerotic, calcified and poorly vascularized intima—would improve graft incorporation and

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reduce the incidence and extent of fluid collection around the aortic prosthesis. In the present trial we compared standard thrombectomy of the aneurysm sac with thrombectomy plus endarterectomy in elective open AAA repair, by assessing the fluid collection around the graft by abdominal CT scan, 1 year after surgery.

Methods

From July 2005 to 2008 all patients who underwent elective open AAA repair for an asymptomatic or symptomatic aneurysm at the Centre for Vascular Surgery Aarau-Basel were alternately treated with mere conventional thrombectomy or thrombectomy plus endarterectomy of the aneurysm sac (pseudo-randomization). Exclusion criteria for the study were ruptured aneurysm, inflammatory aneurysm, aneurysm due to Marfan syndrome or other connective tissue disease, and aneurysmatic dilatation of chronic aortic dissection. Of the 136 patients in the series, 132 were operated with a transabdominal approach; the other 4 patients were operated with a retroperitoneal access. Endarterectomy of the aneurysm sac was performed bluntly, using a Kelly clamp to find the layer of dissection between the intimal and adventitial layer of the aneurysm wall (see Fig. 1). Endarterectomy was usually performed immediately after opening the aneurysm, before oversewing bleeding lumbar arteries and before performing the arterial anastomoses. If bleeding from lumbar arteries was severe, they were oversewn first; the suture then usually had to be removed for the endarterectomy and the lumbar artery had to be oversewn again. The endarterectomy was terminated ~2 cm away from the aortic anastomoses, so as not to compromise them. Endarterectomy was restricted to the aortic aneurysm, even in patients with an aneurysm that

extended into the iliac arteries. The endarterectomy required approximately 10 min in all patients. If the intimal layer was strongly adherent, we did not insist on a perfect endarterectomy, and patches of intima were left behind. All patients had a standard-walled polytetrafluoroethylene (PTFE) bifurcated or tube graft (Gore-Tex) implanted. In all patients the aneurysm sac was tightly closed over the prosthesis using a continuous suture after completion of the arterial anastomoses. Five board-certified vascular surgeons, all using the identical surgical technique, were involved in either performing the procedure or instructing three trainee vascular surgeons.

Abdominal CT with 1.5 mm sections and intravenous contrast enhancement was performed 1 year postoperatively. All CT scans were analyzed on a work station by a single vascular surgeon (T.W.) who, at the time of analysis, was blinded as to the procedure performed. The maximum width of the fluid collection between the prosthesis and the original aneurysm sac on any axial image was assessed. In case of a bifurcated graft, the maximum distance perpendicular to the tangent joining the two graft limbs and the original aneurysm sac was measured (Fig. 2). If no fluid between the graft and the aneurysm sac was visible, this was taken as radiological evidence of complete graft incorporation. A fluid collection wider than 10 mm was considered a perigraft seroma.

Statistical analysis

In a first step we used multiple linear regression to study the effect of endarterectomy (and tube graft versus bifurcated graft) on the maximum fluid width. Because the assumption of normal distribution of the outcome for linear regression was not fulfilled, we log transformed the fluid width values, which resulted in appropriately fitting linear

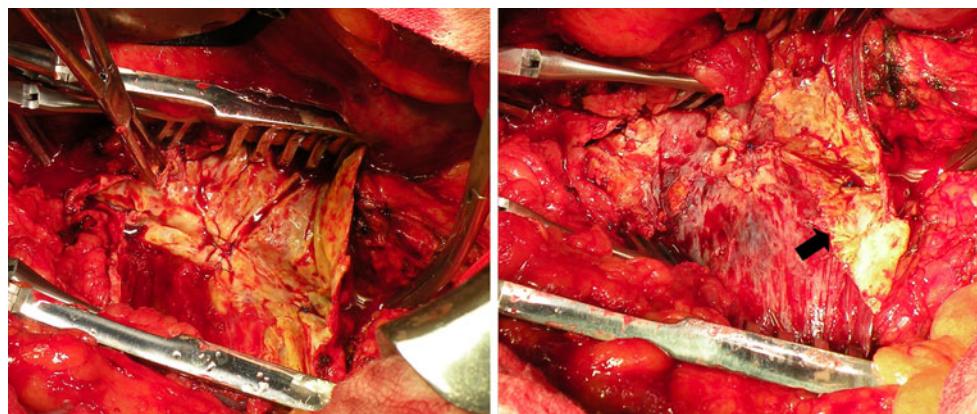


Fig. 1 Technique of endarterectomy: the image on the *left* shows blunt dissection between the thickened intima and the adventitia of the aneurysm sac with a Kelly clamp. The image on the *right* shows

the aneurysm sac after endarterectomy. The intima near the aortic neck (arrow) was not removed so as not to compromise the proximal anastomosis

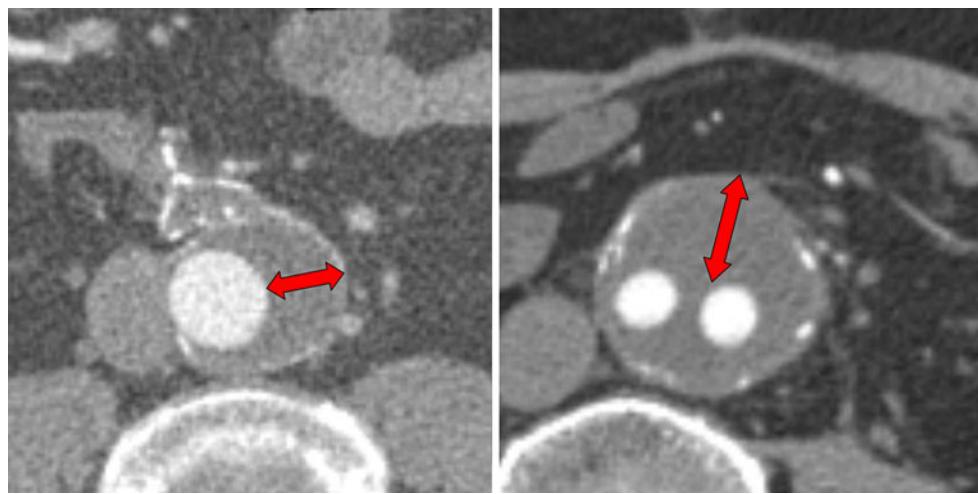


Fig. 2 Measurement of perigraft fluid width on CT scans. The maximum width of perigraft fluid on any axial section was identified. In case of a bifurcated graft, the maximum distance between the tangent to the two graft limbs and the aneurysm sac wall was measured

models. For the log transformation, the patients with no visible fluid (maximum width of 0 mm) were assigned a maximum fluid width of 1 mm, to avoid log of zero (i.e., infinity), which is standard practice in the analysis of non-normally distributed outcome data. We back-transformed model estimates to the normal scale using inverse log function. In a second step, we performed logistic regression analysis to study the effect of endarterectomy (and tube graft versus bifurcated graft) on the frequency of either absence of any fluid or presence of fluid wider than 10 mm. We always first fitted univariate regression models and extended the models for type of graft (tube or bifurcated) to see whether the estimates change after adjustment. For all models, we also evaluated the interaction terms to see whether the effect of endarterectomy was different in patients with a tube graft rather than a bifurcated graft.

Results

A total of 136 patients were enrolled, comprising all patients with open repair of a non-ruptured aortic aneurysm during the study period. The 30-day mortality was 2.2% (3 patients). Endarterectomy of the aneurysm sac did not lead to any intraoperative or postoperative complications. A total of 10 patients died within the first year after surgery, and 12 patients declined follow-up. One patient had his postoperative CT shortly before his death within 1 year of the operation. A CT scan 1 year after surgery was thus available for 115 patients, 56 of them had undergone endarterectomy of the aneurysm sac; and 59 had not. The average age of the patients was 71 years (range: 50–86 years), 101 patients were male, and 14 patients were female. The average maximum diameter of

the aortic aneurysm was 62 mm (range: 33–97 mm). The patients with aneurysms smaller than 50 mm in diameter were operated for concomitant large iliac artery aneurysms. Fifty-seven of the patients had a tube graft, 58 had a bifurcated graft. Median maximum width of the perigraft fluid collection in the original aneurysm sac was 4.0 mm (interquartile range: 0–6.0 mm) in patients with endarterectomy of the aneurysm sac and 8.0 mm (interquartile range: 4.0–15.5 mm) in the patients without endarterectomy ($P < 0.0001$; Fig. 3a). The effect remained unchanged after adjusting for the type of graft (tube graft or bifurcated graft). The fluid collections showed density corresponding to a seroma; no patient had evidence of a communication to the lumen of the graft or aorta. A fluid collection wider than 10 mm was found in 8 patients with endarterectomy compared to 28 patients without endarterectomy (OR 0.18, 95% CI 0.07–0.46, see Fig. 3b). Conversely, 17 patients with endarterectomy had no visible fluid collection around their graft, a sign of complete graft incorporation, whereas this was the case for only 6 patients without endarterectomy (OR 3.85, 95% CI 1.39–10.66; Fig. 3c). The average maximum width of perigraft fluid was larger by a factor 2.04 (95% CI 1.44–2.94) in patients with bifurcated grafts than in patients with tube grafts (Fig. 3d). From statistical interaction tests, there was no evidence for the presence of differential effects of endarterectomy for the type of grafts (tube or bifurcated) on perigraft fluid [$P = 0.99$ for the linear model, $P = 0.66$ and 0.49 for the logistic models of no fluid and >10 fluid, respectively (Fig. 3e)]. The ratio of tube to bifurcated grafts was identical among patients with endarterectomy and those without. The width of the perigraft fluid did not correlate with the preoperative AAA diameter (Fig. 3f).

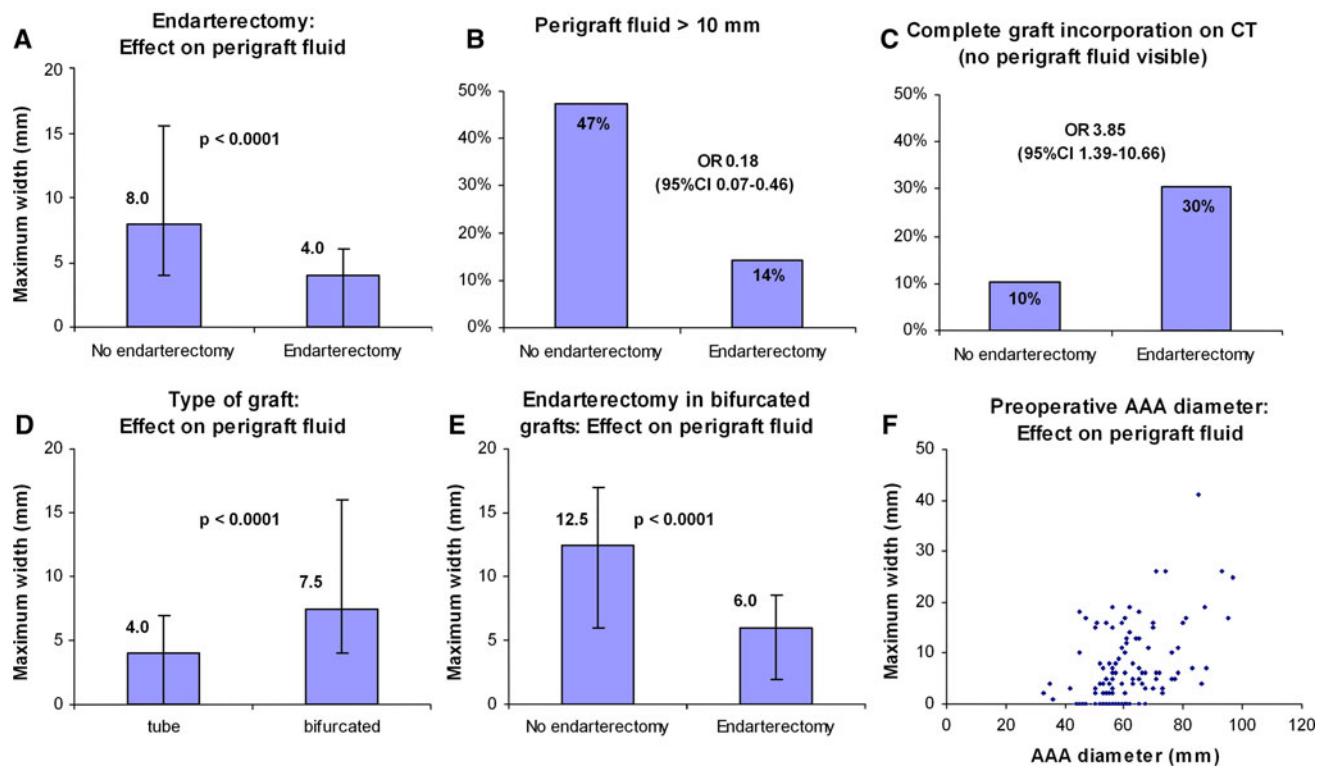


Fig. 3 **a** Maximum width of perigraft fluid was greatly reduced in patients with endarterectomy of the aneurysm sac. Values are given as median. Bars represent second and third quartiles. **b** The incidence of perigraft fluid collections wider than 10 mm is greatly reduced in patients with endarterectomy of the aneurysm sac. **c** The incidence of signs of complete graft incorporation on CT is greatly increased in

patients with endarterectomy of the aneurysm sac. **d** Perigraft fluid collections were significantly larger in patients treated with a bifurcated graft than in patients treated with a tube graft. **e** The effect of endarterectomy remained even when only assessing patients treated with a bifurcated graft. **f** The width of perigraft fluid collections did not correlate with the preoperative AAA diameter

Discussion

This is the first study that investigates endarterectomy of the aneurysm sac in open AAA repair. Endarterectomy led to a significantly smaller perigraft fluid collection in the original aneurysm sac, to a significantly lower incidence of fluid collections wider than 10 mm, and to a significantly higher incidence of radiological signs of complete graft incorporation 1 year after surgery. Our study thus suggests that this simple, brief, and non-dangerous modification of standard surgical practice for open AAA repair may improve graft incorporation.

Incomplete graft incorporation is thought to predispose to graft infection and is thus an important issue, even in the absence of symptoms. Furthermore, the interaction of graft material and the vessel wall is of scientific interest, as it is most relevant for the anchoring of a stent graft in endovascular repair.

This is also the first study that systematically assesses the incidence and extent of perigraft fluid after open aortic aneurysm surgery. There is only one publication available, where the authors attempt to assess the incidence of symptomatic perigraft seroma after open AAA repair [7].

In that article, the authors report 5 patients with proven symptomatic perigraft seroma during a period in which 256 patients were operated with a PTFE graft. The authors thus estimate the incidence of symptomatic perigraft seroma to be 2.7%. This figure contrasts with the results of large follow-up studies after open AAA repair with PTFE grafts (e.g., [8]), where perigraft seroma was not recognized as a common complication. Neither does this figure correspond to our own clinical experience, where we have treated only 3 patients with symptomatic perigraft seroma over the last 15 years. The present study suggests that asymptomatic perigraft seroma is very common: After standard treatment (i.e., no endarterectomy), 47% of patients had a fluid collection wider than 10 mm 1 year after surgery. There are no studies available that this figure could be compared against, as systematic follow-up by CT has not been performed in any of the large follow-up studies after open AAA repair. It is important to note that none of the patients in the present study developed a symptomatic perigraft seroma, although there were patients with fluid collections ranging up to 45 mm in width and a maximum aneurysm sac diameter of up to 70 mm. The clinical significance of our observation is thus unclear. We do not intend to suggest

that asymptomatic perigraft seroma in the absence of signs of infection require any treatment. We are planning to reassess our study patients by CT scan 5 years after surgery, which we hope will shed more light on the clinical relevance of the fluid accumulations found 1 year after surgery and will allow us to describe their natural course.

Treatment allocation in this study occurred by alternating between successive patients (pseudo-randomization). Formal randomization was not used because it was felt that the increased complexity of the process would impair protocol adherence. The robust results obtained suggest that it is unlikely that they would have been affected by formal randomization.

Our study does not shed any light on how endarterectomy of the aneurysm sac decreases perigraft fluid accumulation. Four different possible etiologies for the formation of perigraft seroma are discussed in the literature. The first of these, low-grade infection, particularly with pathogens producing biofilms, may lead to perigraft fluid collections [6]. None of our patients had other radiological systemic signs of vascular graft infection. This renders infection as a cause extremely unlikely. Second, hyperfibrinolysis of clotted blood in the aneurysm sac has been proposed as a possible mechanism for perigraft seroma formation [9]. Third, an immunological response to the graft material that prevents tissue ingrowth and graft incorporation has been discussed [10, 11]. This hypothesis is supported by the evidence of a factor inhibiting fibroblast growth in perigraft seroma and the successful treatment of perigraft seroma by plasmapheresis [12]. Fourth, and in our eyes most important, ultrafiltration of the graft has been discussed as a mechanism for seroma formation [6]. This explanation is supported by the fact that all reported cases of perigraft seroma after open AAA repair occurred in patients operated with a PTFE graft. Ultrafiltration is a flow-related phenomenon that is well recognized as occurring with PTFE grafts in vitro and in vivo. Increased ultrafiltration in first-generation PTFE grafts for endovascular aneurysm repair has also been implicated in graft failure with aneurysm size increase due to endotension [13]. It would, however, be premature to conclude from our observations that the potential formation of a perigraft seroma is an inherent disadvantage of PTFE grafts, which would mandate a change in surgical strategy or follow-up regime. As mentioned, several long-term studies (e.g., [8]) prove the safety and durability of PTFE aortic grafts, which are in no way inferior to polyester grafts.

It was of interest to note that all surgeons involved in the present study reported that the aneurysm sac could be closed more tightly and accurately over the prosthesis when endarterectomy of the aneurysm sac had been performed, particularly if the intima had been heavily calcified

or thickened. The reduced perigraft fluid may thus be due to tighter and more precise closure of the aneurysm sac over the graft and not to the removal of the intima *per se*. This observation suggests that details of the surgical technique used may be important for the development of fluid collections in the original aneurysm sac.

We also found that perigraft fluid collections were larger after placing a bifurcated aortoiliac graft rather than an aorto-aortic tube graft. This is presumably primarily because the stiff aneurysm wall cannot be approximated to the two limbs of a bifurcated graft as snugly as around a tube graft. The fluid accumulation around the main body of the bifurcated graft tended to be similar to that around the equivalent portion of the tube graft.

In summary, we present evidence that asymptomatic fluid collections around PTFE grafts 1 year after open AAA repair are much more common than previously thought. Endarterectomy of the aneurysm sac at the time of open AAA repair proved to be a simple and safe procedure that led to significantly fewer perigraft seromas, as well as to radiologically improved graft incorporation, and endarterectomy may thus be generally recommended.

References

- Cuff RF, Thomas JH (2005) Recurrent symptomatic aortic sac seroma after open abdominal aortic aneurysm repair. *J Vasc Surg* 41:1058–1060
- Kat E, Jones DN, Burnett J et al (2002) Perigraft seroma of open aortic reconstruction. *Am J Roentgenol* 178:1462–1464
- Lucas LA, Rodriguez JA, Olsen DM et al (2009) Symptomatic seroma after open abdominal aortic aneurysm repair. *Ann Vasc Surg* 23:144–146
- Stierli P, Gurke L, Hess P et al (2000) Aneurysm sac enlargement after conventional inflammatory aneurysm repair with a polytetrafluoroethylene aortobiiliac graft. *Eur J Vasc Endovasc Surg* 20:484–486
- Sugimoto T, Kitade T, Nishikawa H et al (2004) Large perigraft seroma after aortobiiliac bypass with an expanded polytetrafluoroethylene graft: report of a case. *Surg Today* 34:698–700
- Williams GM (1998) The management of massive ultrafiltration distending the aneurysm sac after abdominal aortic aneurysm repair with a polytetrafluoroethylene aortobiiliac graft. *J Vasc Surg* 28:551–555
- Thoo CH, Bourke BM, May J (2004) Symptomatic sac enlargement and rupture due to seroma after open abdominal aortic aneurysm repair with polytetrafluoroethylene graft: implications for endovascular repair and endotension. *J Vasc Surg* 40:1089–1094
- Shah DM, Darling RC III, Kreienberg PB et al (1997) A critical approach for longitudinal clinical trial of stretch PTFE aortic grafts. *Cardiovasc Surg* 5:414–418
- Risberg B, Delle M, Eriksson E et al (2001) Aneurysm sac hygroma: a cause of endotension. *J Endovasc Ther* 8:447–453
- Ahn SS, Machleder HI, Gupta R et al (1987) Perigraft seroma: clinical, histologic, and serologic correlates. *Am J Surg* 154:173–178

11. Ahn SS, Williams DE, Thye DA et al (1994) The isolation of a fibroblast growth inhibitor associated with perigraft seroma. *J Vasc Surg* 20:202–208
12. Sladen JG, Mandl MA, Grossman L et al (1985) Fibroblast inhibition: a new and treatable cause of prosthetic graft failure. *Am J Surg* 149:587–590
13. Haider SE, Najjar SF, Cho JS et al (2006) Sac behavior after aneurysm treatment with the Gore excluder low-permeability aortic endoprosthesis: 12-month comparison to the original excluder device. *J Vasc Surg* 44:694–700