Endotension in an experimental aneurysm model

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Purpose: The purpose of this study was to design an experimental model of endotension and to investigate whether attachment site failure without endoleak results in higher aneurysm sac pressure (ASP).

Methods: Infrarenal aortic aneurysms were created in canines with an elliptical knitted polyester patch. Pressure transducers were inserted into the aneurysm. Group I (n = 4) underwent endovascular stent graft exclusion of the aneurysm. An attachment site failure was formed in group II (n = 5) before aneurysm exclusion. ASP measurements were obtained for 3 weeks, and the ratio of mean ASP to mean systemic blood pressure (ASP/BP) was calculated. Before explant, norepinephrine was administered and ASPs were recorded at varying systemic pressures. Stent graft cuff exclusion of the attachment site failure was performed in group II.

Results: Intraoperative arteriography and duplex ultrasonography did not reveal an endoleak in either group. ASP/BP in group I was 0.39 ± 0.02 compared with 1.01 ± 0.02 in group II (P < .001). Mean systemic pressure varied from 55 to 177 mm Hg after norepinephrine administration. Within this interval, ASP/BP was 0.51 ± 0.10 in group I compared with 0.91 ± 0.10 in group II (P < .001). ASP/BP before cuff deployment in group II was 0.98 ± 0.08 compared with 0.46 ± 0.04 after cuff deployment (P < .001).

Conclusion: Systemic pressure is transmitted to the aneurysm sac through an attachment site failure, despite no endoleak, resulting in endotension. Cuff exclusion of the attachment site failure decreases ASP. ASP may help determine the need for future intervention after endovascular aneurysm repair. (J Vasc Surg 2002;36:814-7.)
6-cm longitudinal arteriotomy to create an infrarenal AAA. Before aneurysm formation in group II, a 3-cm segment of a 10F Argyle shunt (Kendall, Mansfield, Mass) was secured to the proximal aortic wall so that 1.5 cm of the shunt extended into the aneurysm (Fig 1). The shunt had an outer diameter of 3.3 mm and an inner diameter of 2.2 mm. The purpose of the shunt segment was to create an attachment site failure once the endograft was deployed. All aortic side branches in proximity to the AAA were ligated and divided.

An implantable silicon strain-gauge pressure transducer (P, 4.5; Konigsberg Instruments, Pasadena, Calif) was secured to the luminal side of the knitted polyester component of the aneurysm wall before stent graft deployment. After aneurysm exclusion, a graft tunneler was used to pass the cable and compensation module of the transducer to an area within the subcutaneous tissue between the scapulae of the animals. External access was achieved with a skin button appliance, allowing for daily measurements of aneurysm sac pressure (ASP). The aneurysm was excluded in both groups of animals with 8-cm × 8-mm stent grafts (Talent; Medtronic, Santa Rosa, Calif) inserted through the right common iliac artery.

Assessment of abdominal aortic aneurysm exclusion. Intraoperative arteriography was performed after stent graft deployment to assess for the presence of endoleaks. Before explantation of the AAA and stent grafts, color flow duplex ultrasonography was performed intraoperatively for endoleak assessment and AAA dimensions were obtained. Arteriography also was performed at explantation.

Pressure measurements. Daily ASPs were obtained for 3 weeks with connection of an analog-digital recorder (Model 2700; Keithley Instruments, Cleveland, Ohio) to the pressure transducer. In addition, daily forelimb phonomanometry was performed. The ratio of the mean ASP to the mean systemic blood pressure (ASP/BP) was calculated to account for differences in BPs among the animals. The ratio of the systolic pressure in the aneurysm sac to the common iliac artery.

Correlation of aneurysm sac pressure with systemic pressure. Three weeks after the initial surgical procedures, a catheter was positioned in the abdominal aorta of the animals proximal to the excluded AAA, via the right common iliac artery. The catheter was connected to a standard pressure transducer for determination of systemic BP. Baseline ASP and systemic pressures were obtained. To investigate the relationship between ASP and increasing systemic pressure, intravenous norepinephrine was administered and ASP and systemic pressure were recorded.

Exclusion of attachment site failure. To test whether exclusion of the attachment site failure decreased ASP, a 5-cm stent graft cuff (Talent) was deployed (3 weeks after the initial surgical procedure) to cover the attachment site failure in group II. Arteriography was performed to assess for the presence of endoleak. ASP and systemic pressure were recorded at baseline and again after norepinephrine administration to investigate whether exclusion of the attachment site failure reduced the pressure within the aneurysm sac.

Statistical analysis. ASP ratios are expressed as the mean ± standard error of the mean. SigmaStat version 2.0 for Windows (Jandel Scientific Software, San Rafael, Calif) was used to perform t tests on the mean ASP ratios for the two groups.

RESULTS

One dog in group I died in the recovery room of respiratory arrest. Two dogs in group 2 were killed because of hindlimb paralysis. All animals with patent stent grafts were included in the analysis.

Assessment of endoleak. Intraoperative arteriography performed at the time of initial AAA exclusion and at explant did not reveal endoleak in any of the animals. In addition, intraoperative color flow duplex ultrasonography was performed at explant and confirmed the absence of endoleak in both groups of animals.

Aneurysm size and characteristics. The mean aneurysm diameter at the time of explant was 16 ± 1.7 mm in group I and 16.4 ± 1.5 mm in group II. The mean aortic diameter proximal to the aneurysm was 7.5 ± 0.2 mm in group I and 7.7 ± 0.15 mm in group II. Organized thrombus was seen within the aneurysm sac in both groups and within the shunt in group II. The relative cross-sectional area of the shunt to the patent stent graft lumen was approximately 17%. No deformation of the shunt by the proximal stent was found.

Aneurysm sac pressure measurements. During the 3-week study period, the mean ASP/BP in group I was 0.39 ± 0.02 compared with 1.01 ± 0.02 in group II (P < .001). Similar findings were observed when the systolic pressure ratios were calculated (Table). The ASP ratio remained systemic over 3 weeks in group II (Fig 2).

Correlation of aneurysm sac pressure with manipulation of systemic pressure. Before explantation, mean ASP/BP before administration of norepinephrine was

Fig 1. Drawing of aneurysm model, with group I (control) and group II (attachment site failure).
Mean and systolic ASP/BP

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<th>ASP/BP (mean)</th>
<th>ASP/BP (systolic)</th>
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<tr>
<td>I. Control</td>
<td>0.39 ± 0.02</td>
<td>0.38 ± 0.02</td>
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<tr>
<td>II. Attachment site failure</td>
<td>1.01 ± 0.02</td>
<td>0.94 ± 0.03</td>
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DISCUSSION

Experimental and clinical studies show that type I and II endoleaks result in higher pressure within the aneurysm sac. However, whether pressure exists in the aneurysm sac in the absence of flow (endotension) is controversial and has been a topic of heated debate. The presence of endotension has largely been inferred from the observations of aneurysm rupture or enlargement after endovascular AAA repair in which no endoleak could be identified.

This study investigated endotension in an experimental animal model. We hypothesized that pressure could be transmitted to the aneurysm sac through thrombus. A proximal attachment site failure was created with securing an Argyle shunt to the canine aorta, with deployment of the stent graft just below the proximal portion of the shunt. Ligation and division of all outflow vessels resulted in thrombosis of the aneurysm sac and thrombus formation within the shunt. Thus, there was no flow into the aneurysm sac through the shunt, which was confirmed with intraoperative arteriography and duplex ultrasonography.

The group of animals with an attachment site failure showed mean and systolic ASP ratios that were basically systemic (1.01 and 0.94, respectively). These ratios were significantly higher than those of the group without the attachment site failure (0.39 and 0.38, respectively). The mechanism for the observed endotension in this experimental model appears to be the transmission of aortic pressure to the aneurysm sac through the attachment site failure.

An additional finding from this study is that the mean ASP ratio remained fairly constant with increasing systemic pressure. This observation supports the hypothesis that pressure transmission through the attachment site failure is the mechanism for endotension in this model. It would also suggest that BP control is especially advantageous in the clinical situation when endotension is suspected.

We were also able to show that exclusion of the attachment site failure with deploying a stent graft cuff proximal to the shunt resulted in markedly reduced ASP ratios, similar to the control group. This intervention is analogous to deploying a proximal extender cuff for a type I endoleak after AAA repair and may also represent a treatment option for endotension.

Potential limitations exist in this study. The mean aneurysm diameter was 1.6 cm, twice the diameter of the proximal canine aorta. This aneurysm model is smaller than that encountered clinically. Whether the same forces and physical properties exist within a larger aneurysm is not known. Multiple factors likely contribute to the pressure within the aneurysm sac. Systemic BP, aneurysm sac diameter and volume, mechanical properties of the aneurysm wall and stent graft device, and the size of the attachment site failure or thrombus layer are important considerations.

What the short-term and long-term consequences are of a pressurized aneurysm sac without an endoleak is unknown. The law of LaPlace states that the wall tension in the aneurysm sac is directly proportional to diameter and...
pressure. The presence of endotension within the aneurysm sac after endovascular AAA repair may signify treatment failure and risk of aneurysm rupture. Rupture may not lead to catastrophic hemorrhage if the proximal and distal stent graft attachment sites remain secure. However, endotension may also be the result of a sealed type I endoleak, and rupture could have disastrous consequences in that situation.1

Stent graft migration also has been implicated as a cause of aneurysm rupture after endovascular AAA repair.2,3 Insecure proximal fixation from an angulated neck or deployment too low in the neck may be responsible for subsequent graft migration in some of these cases.3 Another possible explanation is that endotension may contribute to the risk of stent graft migration or failure. The presence of a pressurized aneurysm sac could lead to aneurysm growth or dilation of the native aorta at the proximal or distal attachment sites. This could result in graft migration, type I endoleak, or rupture.

Clinical detection of endotension has been limited by the inability to noninvasively measure ASP. Postoperative surveillance after endovascular stent graft repair is primarily performed with serial computed tomographic scans to measure aneurysm diameter or volume and to assess for the presence of endoleak. However, endoleak (apart from type I) appears to be a poor predictor for risk of aneurysm rupture.2,3,9 Demonstration of endotension in patients whose aneurysms enlarge or fail to shrink in size may be a more reliable indicator of treatment failure. Given the current state of technology, deployment of a small computerized chip into the aneurysm or onto the body of the graft at the time of endovascular repair that would allow serial noninvasive measurements of ASP may be possible. As our understanding of the consequences of endotension evolves, ASP may help determine the need for future intervention after endovascular AAA repair.

REFERENCES

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