Sac enlargement due to seroma after endovascular abdominal aortic aneurysm repair with the Endologix PowerLink device

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A patient who had undergone endovascular repair of an abdominal aortic aneurysm with the Endologix PowerLink bifurcated system presented with delayed aortic aneurysm enlargement due to assumed endotension. He was treated with aortic sac evacuation and wrapping of the endograft. This is the first report of endotension and aneurysm sac enlargement after implantation of the PowerLink endograft. (J Vasc Surg 2006;43:169-71.)

Sac enlargement after endovascular treatment of an abdominal aortic aneurysm (EVAR) implies increased pressure in the aneurysm sac and so far has been associated mainly with endoleaks. Recently, several authors have reported cases of sac enlargement after EVAR with the Excluder (W. L. Gore & Associates, Sunnyvale, Calif) (a polytetrafluoroethylene [PTFE] system) that were associated with the presence of a sac hygroma but no endoleak.[1-5] Thoo et al[6] also reported five cases of symptomatic sac enlargement and rupture due to seroma after open repair of abdominal aortic aneurysm (AAA) with a PTFE graft. The exact extent of this phenomenon is not known, but it is correlated with the PTFE vascular grafts.

We present a case report of a patient who underwent EVAR with the use of a PTFE endograft, the Endologix (Endologix Inc, Irvine, Calif) PowerLink bifurcated endograft, and presented with delayed aortic aneurysm enlargement partially due to endotension.

CASE REPORT

A 71-year-old man with a history of severe ischemic coronary artery disease underwent EVAR of a 56-mm AAA with a 25-16-175 RDL Endologix PowerLink Bard bifurcated endograft. The choice of a nonmodular endograft was due to the particular morphology of the patient's aneurysm. Circular thrombotic material was present in the aneurysm; it resulted in a narrow lumen that could present difficulties in trying to enter the iliac gate for the contralateral iliac branch placement of a modular endograft. These difficulties were avoided with the implantation of a nonmodular endograft. The postimplantation angiographic control showed no endoleak of any kind. Computed tomography scanning (CT) 6 months after the procedure showed a decrease in the maximal aneurysm diameter to 54 mm. At 12 months, the CT maximal aortic diameter was 58 mm, with no signs of endoleak. The CT at 24 months showed a maximal aortic diameter of 63 mm; at 36 months, the maximal diameter was 68 mm, with no detectable endoleak even with a late-phase CT scanning technique. Angiographic examination at 36 months had also shown the absence of any kind of endoleak. The patient was asymptomatic, but because of the tendency of the aneurysm to increase its diameter and, potentially, its risk of rupture, open surgical treatment was chosen.

Median laparotomy was performed, and surgical preparation of the aorta and the iliac arteries for endograft explantation and conventional graft interposition was performed. The aneurysmal sac was punctured with an 18-gauge needle for measurement of the intrasac pressure, but no pressure was detectable. However, the aneurysm was pulsative. The sac was opened without aortic cross-clamping, and a large amount of straw-colored, rubbery, gelatinous, semifluid material (Fig 1) was released under tension and evacuated. No blood was present, and no endoleak was detected. However, careful examination of the endograft showed a point of blood impregnation of the PTFE graft (Fig 2). Therefore, wrapping of the endograft with a knitted Dacron vascular graft (DuPont, Wilmington, Del) was performed, and the sac was closed with imbricating sutures (Fig 3). Culture of the sac contents was negative. At 6 months after this procedure, CT scanning showed no sac enlargement or endoleak.

DISCUSSION

Aneurysm sac enlargement is an important finding in the follow-up of patients treated with EVAR. Persistent pressurization of the AAA sac can eventually result in rupture, but in the presence of little or no intrasac flow, it may not result in the patient's death. However, continued expansion of the AAA sac can result in dilation of the infrarenal proximal aortic neck and the iliac arteries that may threaten the integrity of proximal and distal endograft fixation and sealing.[5,7] The treatment of this situation is undefined. Many surgeons would proceed to open surgery with removal of the endograft and placement of a conventional vascular graft. Although this conversion creates a permanent solution, it can be associated with significant morbidity,[5] and such conversion is associated with a peri-

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Competition of interest: none.

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0741-5214/$32.00

doi:10.1016/j.jvs.2005.09.011

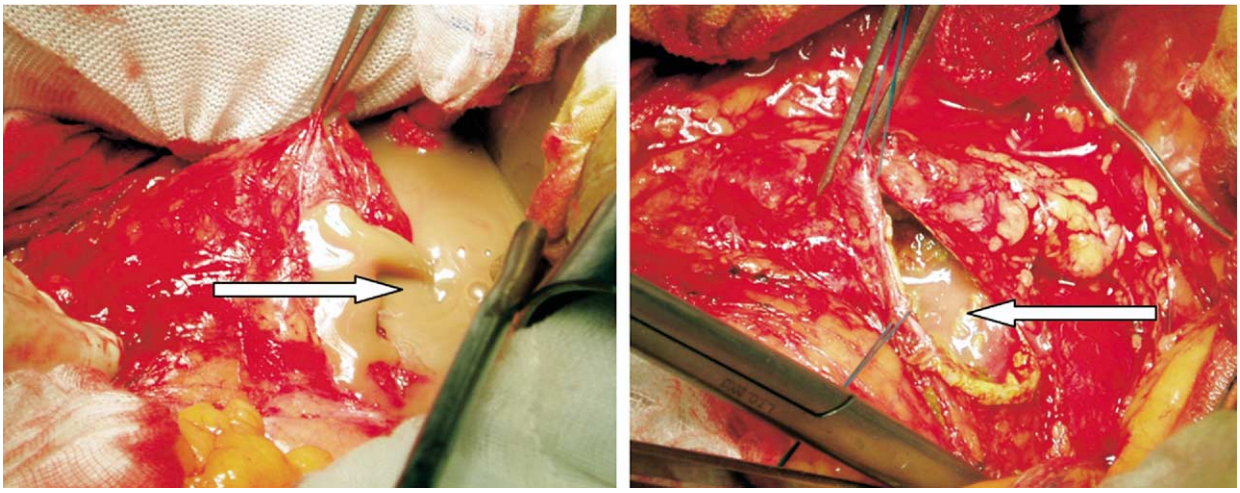

Fig 1. Intraoperative findings. The arrows show the straw-colored, rubbery, gelatinous, semifluid material found in the aneurysmatic sac.

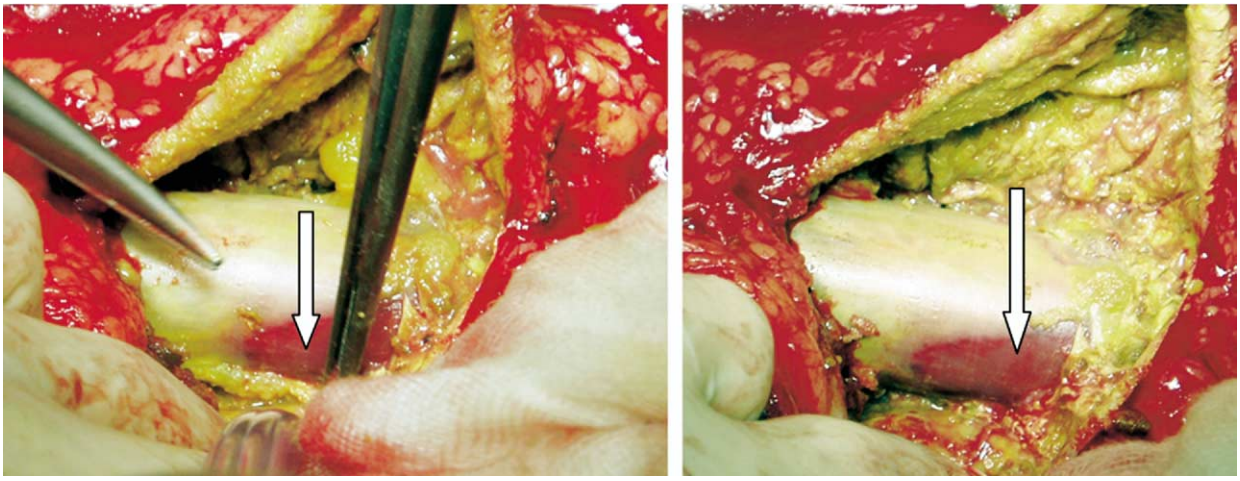

Fig 2. The *arrows* show the point of blood impregnation of the polytetrafluoroethylene.

operative mortality of 20% to 30%.[8] Additionally, there is no proof in the literature that sac enlargement after EVAR will always lead to sac rupture, and recently Thoo et al[6] showed that conservative treatment with only observation is not unreasonable in selected patients.

In the case presented here, sac enlargement was not associated with an endoleak on preoperative imaging studies, and when the aneurysm sac was opened, there was no evidence of blood flow or an attachment site endoleak. It seems that the most likely cause of sac enlargement in this case was transgraft passage of fluid from the endograft lumen. Therefore, we chose to treat this seroma with an endograft wrapping and imbricating sutures of the aneurysm sac without removing the endoprosthesis, thus avoiding aortic cross-clamping in a high-risk patient (Fig 3).

Several authors have described seromas, endotension, and aneurysm sac enlargement without evidence of endoleak associated with PTFE grafts[6] and endografts.[1-5] The cases reported in the literature of hygroma after EVAR were related to the Excluder endograft.[1] This is the first report to our knowledge that correlates aneurysm sac seroma and endotension with the PowerLink endograft, which is also a PTFE stent-graft system. Carpenter,[9] in a multicenter trial of the Endologix PowerLink bifurcated system, showed a mean aneurysm diameter reduction of 2 mm in 6 months, 4 mm in 12 months, and 7 mm in 24 months. However, in the same study, 2 patients demonstrated aneurysm diameter expansion without a detectable endoleak. It is interesting to note that Arya et al[10] recently reported a case of endotension and sac enlargement after open repair of AAA with a knitted polyester graft. Large studies with long-term

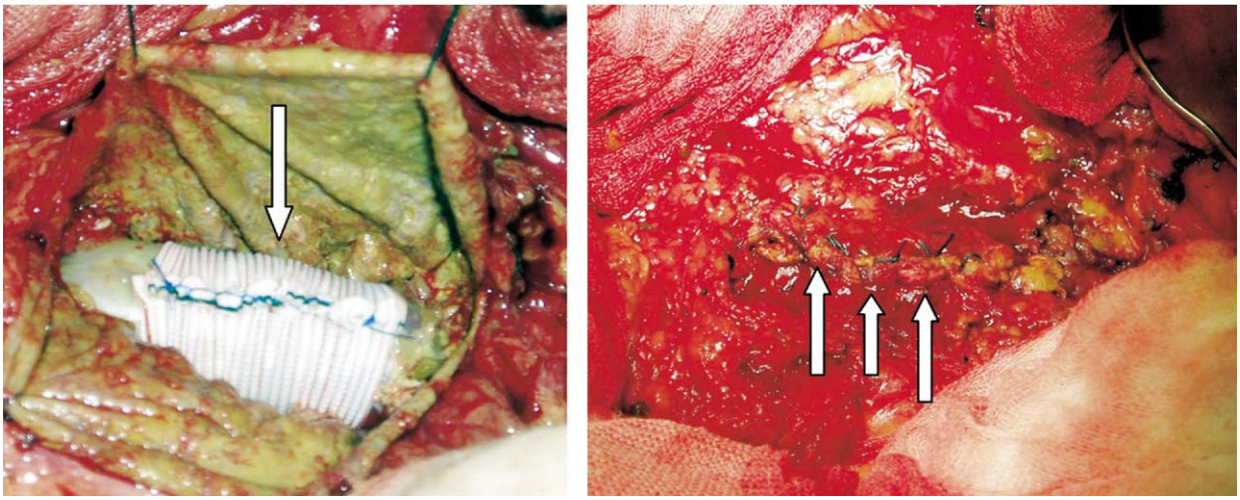

Fig 3. Treatment: the *arrows* show the wrapping of the endograft and the sac imbrication.

results after open repair and EVAR with knitted polyester and PTFE grafts and endografts would be very useful for the better understanding of the exact dimension of the phenomenon and its correlation with specific vascular materials.

The exact mechanism of persistent endotension without detectable endoleak remains unclear. Some researchers propose that endotension could be the result of direct pressure transmission from the adjacent aortic lumen to the aneurysm sac,[11-13] whereas Lin at al[4] suggested that endotension may represent a low-flow endoleak that is not visualized with conventional imaging techniques. This is supported by the fact that aneurysm enlargement cannot occur without expansion of the intrasac volume.

The intraoperative findings (Fig 1) in this case were similar to those published by other authors.[4,6] An interesting exception is the finding of a point of blood impregnation of the PTFE graft (Fig 2). However, we do not know whether this was correlated with the intrasac seroma. Intrasac pressure measurement was not feasible because of the gelatinous, semifluid nature of the material within the sac, but the sensation of the pulse that the sac gave was of a systemic-like pressure that was not the result of transmitted pulsation from the endograft. Thoo et al[6] proposed that sac imbrication with sutures is sufficient treatment of this problem. In the case presented here, the presence of a type III microendoleak that was not bleeding at the time could not be excluded, so wrapping of the endograft with a Dacron graft was preferred. Even if no blood flow were present at the time, blood staining of the graft was a very unusual finding that suggested a graft deficit might be present that could lead to type III endoleak.

In conclusion, the pathogenesis of endotension and aneurysm sac enlargement without detectable endoleak remains unclear, and its treatment is undefined. Multicenter trials with long-term results after EVAR and open repair with PTFE and knitted polyester endografts are needed to define the exact extent of this phenomenon.

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Submitted Jul 26, 2005; accepted Sep 8, 2005.