The management of massive ultrafiltration distending the aneurysm sac after abdominal aortic aneurysm repair with a polytetrafluoroethylene aortobiiliac graft

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Collections of serous fluid surrounding prosthetic grafts can be caused by infection or transudation of serum, and making the distinction is often troublesome. Bergamini and his colleagues[1] developed a dog model of low-grade prosthetic graft contamination with *Staphylococcus epidermatis*. All animals developed evidence of graft infection, and 13 of 18 dogs developed a fluid-filled perigraft cyst. Signs of systemic infection, however, were present in only 1 animal, and the *Staphylococcus epidermatis* study strain was isolated from the tissue surrounding the graft in only 1 dog. The authors had to disrupt the biofilm to achieve positive cultures in 14 of 18 animals. This animal model seemed to conform to clinical experience and placed great emphasis on the role of indolent infections in the pathogenesis of perigraft fluid collection.

It is equally clear that perigraft fluid collections may result from transudation of fluid through the prosthetic surfaces, which act similar to a dialysis membrane under certain circumstances.[2–6] Noninfectious seromas are characterized generally by the accumulation of clear serous fluid with a protein and glucose content of serum and the lack of acute inflammatory cells when the sediment is examined.

The need to distinguish between these 2 forms of fluid accumulation became important in the treatment of a 62-year-old man who was seen 2½ years after the repair of an abdominal aortic aneurysm with an aortobiiliac stretch polytetrafluoroethylene (PTFE) prosthesis. There was no evidence of infection, and there was a 12 cm cystic mass surrounding a patent PTFE prosthesis. (J Vasc Surg 1998;28:551-5.)

CASE HISTORY

The patient, a 62-year-old active minister, underwent repair of an 8 cm infrarenal abdominal aortic aneurysm with a PTFE aortobiiliac prosthesis. The large size of the aneurysm made the proximal dissection a bit more difficult than usual, but the procedure was otherwise straightforward, and the patient' s early recovery was uneventful. However, the patient had difficulty eating and, after a protracted period of intestinal intubation, underwent lysis of adhesions 2 weeks postoperatively. A long intestinal tube was threaded to the ileocecal valve, but, despite this, the patient required a second celiotomy for lysis of adhesions 5 weeks postoperatively. Recovery after this second operation was slow, but sure.

The patient returned 1 year later for follow-up examination, and a midline mass was palpated. A large collection of fluid circumferentially surrounding the arterial prosthesis was demonstrated by means of CT. Calcification at the rim of this cystic mass was interpreted to be the aneurysm sac that had been closed around the prosthetic graft during the initial operation.

There was no evidence of anastomotic interruption or stenosis. There was no fever or leukocytosis. A decision was made to observe the patient conservatively.

On a follow-up CT scan 2 months later, the appearance of the graft and aneurysm sac were unchanged. However, the patient was experiencing some discomfort, and all were anxious to discover the etiology of this problem. The patient was referred to the Johns Hopkins Hospital and underwent aortography and aspiration of the cyst in the angio suite. Clear serous fluid was withdrawn. The fluid contained no polymorphonuclear leukocytes and no organisms on gram stain. The glucose was 100 mg / dL, protein 7.3 g / dL, amylase 37 U / L, and lactate dehydrogenase 217 U / L. After 3 days, culture results were reported positive for coagulase negative staphylococcus species exquisitely sensi-

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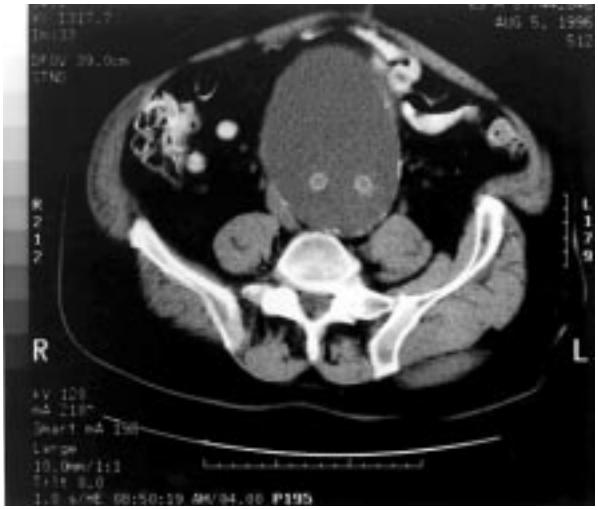

Fig. 1. Preoperative CT scan demonstrating massive distension of the aneurysm sac overlying the iliac limbs of the PTFE graft.

tive to penicillin (1 μg / mL). A decision was made to withhold antibiotic therapy, because the fluid had most of the characteristics of a seroma and contained no polymorphonuclear leukocytes characteristic of biofilm infections.

The patient remained well, with the exception of increasing lower abdominal discomfort. Further enlargement of the aneurysm sac to a diameter of 13.3 centimeters was disclosed 10 months later by means of a second CT scan (Fig. 1), and the patient was admitted for surgery.

On admission, the patient was afebrile and well-nourished. Examination was unremarkable, with the exception of the abdomen, which had a huge midline mass, almost protruding through the midline scar. Pulses were all present and normal, with the exception of the popliteal pulses, which were widened. Laboratory study results included a serum urea nitrogen level of 18 mg / dL, a creatinine level of 1.3 mg / dL, and a white blood cell count of 8100 / μL with a normal differential. Cholesterol was elevated at 230 mg / dL. On Aug. 20, 1996, the patient was taken to the operating room, where the midline incision was reopened. Adhesions were taken down sufficiently to expose the entire anterior surface of the old aneurysm sac, which was very tense. The sac was incised longitudinally, and a large volume of straw-colored, slightly turbid fluid was removed. The graft was found unattached to any tissues except the anastomoses. The surface of the graft was covered with patchy white material that was not slimy. This material was removed from the graft surface and proved to be fibrin without acute inflammatory cells. There was no gross "weeping" of serum from the graft. The aneurysm sac was partially excised. It was very thick-walled, but could be loosely approximated about the graft with sutures, leaving a central defect for a "window." Because the patient was subject to forming adhesions capable of sealing the communication between the cyst and the abdominal cavity, we elected to suture a PTFE "membrane" to the retroperitoneum. The exterior circumference of this square "donut" membrane was sutured to the retroperitoneum with absorbable sutures, and the central donut hole was sutured to the opening left in the aneurysm sac (Fig. 2). There was concern when the patient had a temperature of 39.1°C the evening of surgery, but he remained afebrile thereafter, and all cultures proved to be negative. The persistence of a small cystic cavity, with a maximum diameter of 4 mm, containing the graft was revealed by means of a CT scan performed on the day of discharge (Fig. 3, *A*). This represented the residual aneurysm sac. The patient returned for a follow-up examination 10 months later, and a slightly smaller sac was disclosed by means of a CT scan (Fig. 3, *B*). The patient remains well and is working full time.

DISCUSSION

This case history is presented for 2 reasons. First, it dramatizes the importance of establishing the etiology of the prosthetic fluid collection, and second, we found no other report of managing a huge intra-abdominal periprosthetic cyst by creating a cyst peritoneal window. It was essential to determine whether this dramatic failure of incorporation of the PTFE graft was the consequence of bacterial biofilm infection. Towne et al,[7] reporting on their experience with 20 cases, characterize biofilm infections as "an absence of systemic sepsis, a fluid-filled cavity surrounding the graft, a draining sinus tract, and microorganisms that must be removed from the fabric prosthesis for bacterial cultures." Additionally, evidence of acute inflammation, namely polymorphonuclear leukocytes (PMNs) but not often bacteria, are seen on cellular analysis of aspirated fluid. Our patient could have had a bland infection, and the lack of a sinus tract could be explained by the absence of groin incisions and the deep-seated location of the graft.

The obvious step was to sample the fluid and judge by its composition whether we were dealing with a exudate or transudate. When the fluid more closely mimicked serum concentrations of glucose and protein and failed to have any PMNs, we felt secure in the diagnosis of perigraft seroma. The late report of a positive culture for *S epidermatis* was unsettling, but we felt that the absolute absence of PMNs and anastomotic difficulties outweighed the positive culture.

Much of the literature on perigraft seromas appeared before there was much information on biofilm infections. It is possible, even likely, that many patients thought to have ultrafiltration may have had an infectious etiology. However, there is

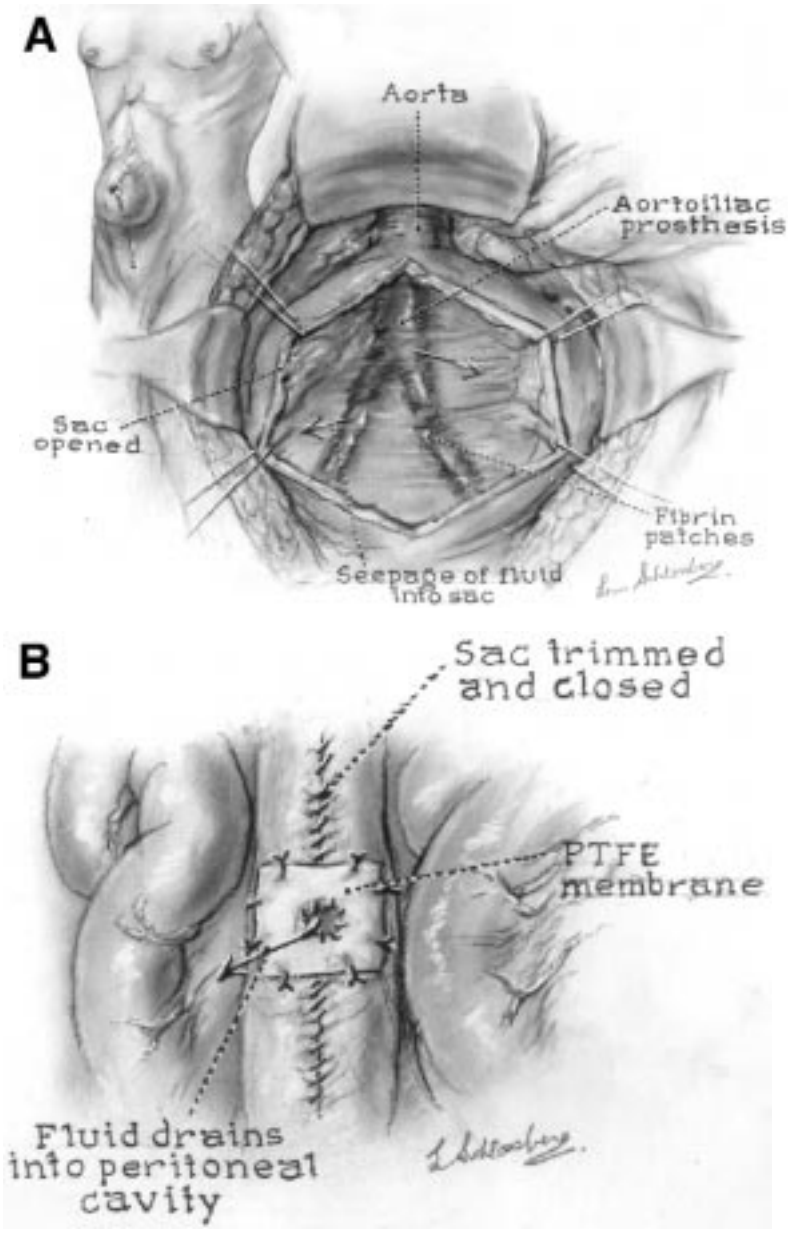

Fig. 2. Operative findings. A, There was a huge aneurysm sac containing the graft. B, Method of creating a functional peritoneal "window," using PTFE membrane to prevent adhesions.

strong evidence clinically and experimentally that ultrafiltration may occur and pose major problems. Although it can be argued that fluid surrounding prosthetic grafts used for hemodialysis access is more likely caused by repetitive needle punctures with introduction of bacteria, there is little likelihood that infection plays a role in the high frequency of seromas associated with subclavian artery-pulmonary artery shunts for the treatment of congenital cyanotic heart disease. In the mid-1980s, 5 reports documented problems with serous fluid leakage posing significant clinical problems.[3,4,8–10] LeBlanc et al[3] reported seromas in 18.8% in 138 children treated with a modified Blalock-Taussig operation using PTFE grafts. Four children required a second operation, 3 for evacuation of the seroma mass and 1 to treat cardiac tamponade. Nine children had the shunt wrapped in silicone sheathing to prevent adhe-

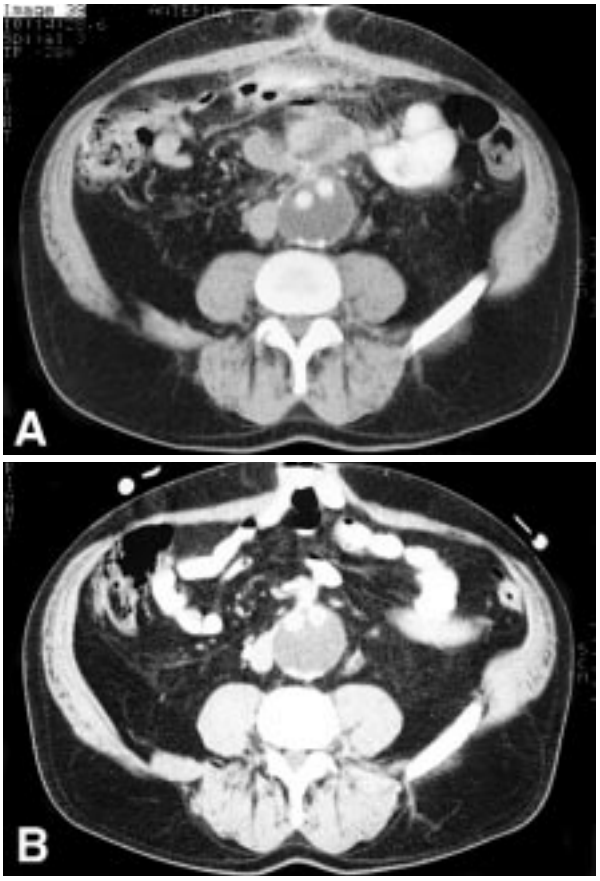

Fig. 3. Follow-up CT scans. A, Discharge CT scan. The transverse "cut" is through the window, and there are inflammatory changes anteriorly. B, Resolution of the inflammatory changes with slight reduction in the size of the sac demonstrated by means of a CT scan 10 months postoperative.

sions, simplifying the takedown of the shunt at the time of total correction. Seroma formation occurred in 5 of these 9. A very short segment central shunt between the aorta and pulmonary artery seems to alleviate this problem.[11]

In vitro studies[2] were undertaken to evaluate mechanisms of transudation from segments of PTFE.[2] Using citrated blood, segments of PTFE were perfused under controlled conditions of pressure and flow. Increases in pressure up to 181 mm Hg had little effect on transudation of fluid, whereas increases in flow caused increased transudation from the PTFE graft segment. This may explain, at least in part, why seroma formation is more common after dialysis access and systemic pulmonary shunt operations; both are associated with high flows.

Why do some grafts leak, and why don't others? In the case of PTFE, the normally hydrophobic quality of the Teflon may become "wettable" when exposed to agents such as alcohol, betadine, tissue fluid, or blood. In some patients, droplets may appear on the exterior surface of the prosthesis, a situation commonly corrected by reversal of heparin with protamine. A more intriguing etiology is the failure of fibroblasts to incorporate the graft in some patient. Sladen[6] reported that serum taken from patients with perigraft seromas inhibited fibroblast proliferation in vitro. Based on this finding, 2 patients underwent plasmapheresis with disappearance of recalcitrant seromas. In a subsequent report by Ahn,[12] serum from 3 patients was studied and found to inhibit the proliferation of autochthonous fibroblasts. The inhibitor disappeared from serum collected after graft removal in 1 patient and after spontaneous resolution in a second.

CONCLUSION

Perigraft fluid collections are caused by multiple factors. Because bacterial biofilm infections are so insidious, they may mimic sterile processes ranging from changes in the graft matrix to failure of fibroblast incorporation of the graft. I hypothesize that aspiration of cyst fluid will discriminate between infectious and sterile etiologies. This hypothesis was tested in the management of the case presented. The creation of a peritoneal seroma window is not novel and was reported by Lowery[13] in the case of axillary-femoral bypass recalcitrant to other measures. It was chosen over graft replacement, covering the graft with microfibrillar collagen,[14] and resection of the aneurysm sac as a safe and more direct method for achieving decompression in a patient prone to the development of adhesions.

Finally, it must be acknowledged that bacterial biofilm infections and transudation or ultrafiltration of fluid are 2 separate and distinct mechanisms responsible for the collection of fluid around a prosthetic graft. We treated this patient uneasily for a bland, noninfectious transudation of fluid from his PTFE graft chiefly because there were no acute inflammatory cells present in the fluid surrounding the graft. Support for this stance came from the experimental studies reported by Bergamini et al[1] and from the clinical report by Towne et al. In the experimental study, Bergamini reported that the perigraft fluid typically contained polymorphonuclear leukocytes, but no organism on Gram' s stain. This finding formed the basis of our subsequent therapy. In the clinical material reported by Towne

et al., all 20 patients found to have biofilm infections had presenting symptoms of infection. There were false aneurysms with perigraft fluid in 10, inflammatory groin mass in 6, and sinus tracts in 4. Although this patient did not have prosthetic material in the groin, there was no evidence preoperatively, intraoperatively, or postoperatively of anastomotic difficulties. Thus if ultrafiltration can occur, and there is ample evidence it can, the presence or absence of acute inflammatory cells in the perigraft fluid may predict which patients may and which may not require graft excision.

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