Surgical repair of an abdominal aortic aneurysm (AAA), sometimes called endoaneurysmorraphy, is performed within the aneurysm sac.\(^1\) After placement of the prosthetic graft, the remaining aneurysm wall can be sutured back into place around the graft to reinforce and isolate the prosthesis.\(^2\) As a result, the graft can be covered by aneurysm wall, approximating the configuration in transluminal stent graft repair.\(^7\) After this operation, disruption of the anastomosis,\(^3\),\(^4\) backbleeding from a lumbar artery\(^5\) or a defect in the graft\(^6\) may cause enlargement and rupture of the former aneurysm sac now repositioned around the graft. This phenomenon is similar to endoleak following transluminal stent graft repair,\(^7\) and might termed endoleak after surgical repair of the AAA.\(^3\),\(^6\) We report a type I endoleak-like phenomenon occurring 12 years after surgical replacement of an AAA. Evidence of the endoleak was detected by computed tomography (CT) 4 months before rupture of the aneurysm sac.

**Case**

A 69-year-old man presented with acute abdominal pain and diaphoresis. He had a history of AAA repair 12 year earlier (hospital records for this previous operation were already lost and the type of graft and suture was unknown). Following the AAA repair, he still had a remnant hypogastric artery aneurysm 2 cm in diameter. CT, therefore, was performed periodically to reassess the hypogastric aneurysm. On such an examination 3 years preceding admission, the abdominal aortic aneurysm sac that had been surgically replaced, measured 2.8 cm in diameter by CT. Four months preceding admission, it had increased to 3.5 cm in diameter, and thrombus was identified outside the prosthesis. At that time, the dilation was not considered clinically significant (Fig. 1A, B).

On admission blood pressure was 112/80 mmHg. Body temperature was normal. Red blood cell count was 4.02 × 10\(^6\), and hemoglobin was 13.2 g/dl. White blood cell count
was $9.8 \times 10^3$, and C-reactive protein was 0.3 mg/dl. Emergency CT showed enlargement of the former aneurysm sac to 4.5 cm in diameter. A massive hematoma was present in the left retroperitoneal space (Fig. 1C). At emergency laparotomy, the aorta was clamped between the origin of the renal arteries and the former proximal graft anastomosis. The aneurysm sac, which covered the prosthesis, was opened. Organized thrombus was present within the aneurysm. The iliac portion of the former bifurcation graft was clamped bilaterally. The lumen of the aneurysm was examined. The former proximal anastomosis had been carried out in an endoaneurysmorrhaphy fashion but had detached at the posterior wall. The aneurysm wall was nearly intact except for a tear in the left posterior wall (Fig. 2). The former anastomosis was divided, and a 16-mm knitted Dacron graft was interposed between the infrarenal aorta and the aortic portion of the original bifurcation graft.

The postoperative course was uneventful. The right hypogastric artery aneurysm was repaired 2 years after the emergency operation after it was shown to have enlarged to 3.5 cm in diameter.

**DISCUSSION**

Following conventional graft replacement of the AAA, Chan et al.\(^3\) reported six cases of type I endoleak, while Yow et al.\(^5\) reported a case of type II endoleak, requiring emergency surgery. In these cases, the second
operation necessitated by endoleak was performed within 18 months after the first replacement surgery; in five of the cases, the second operation was performed within 2 months. In these cases, the endoleak probably was caused by technical problem. However, when endoleak occurs several years after graft replacement surgery, the anastomosis may have broken down because of degeneration of the host artery. In this situation, a pseudoaneurysm usually develops at the anastomosis, but anastomotic leakage into the replaced aneurysm sac may occur instead. In our case, dehiscence at the proximal anastomosis caused bleeding into the aneurysm sac, dilating of the sac and ultimately causing rupture. The tear of the aneurysm sac occurred at its posterior wall, which had not been incised in the first replacement operation. This situation resembles the type I endoleak following stent graft therapy of the AAA. Gawenda et al.\(^4\) reported such a case in which type I endoleak caused a sac rupture 4 years after conventional AAA replacement surgery. A case of type III endoleak caused by a defect in the mid-portion of the body of the bifurcation graft was also reported 14 years after replacement of the AAA.\(^5\)

We recently studied regression of 35 surgically replaced AAA that had been 5 cm or larger according to preoperative contrast-enhanced CT.\(^6\) In the replacement surgery, a Dacron graft was anastomosed intraluminally, and the aneurysm wall was closed with continuous polypropylene suture after graft insertion. The aneurysm sac was observed regularly after surgery by CT. From 1 week to 3 months after surgery, the major and minor diameter of the aneurysmal sac both decreased significantly (major diameter, from 49 ± 12 mm to 32 ± 8 mm; minor diameter, from 39 ± 10 mm to 26 ± 7 mm). At 12 months, the major diameter was 30 ± 7 mm and the minor diameter was 24 ± 4 mm. Shrinkage therefore was nearly complete 1 year after conventional replacement surgery with a Dacron graft. At 1 year, the attached aneurysm sac directly covered the prosthesis.\(^7\) Regression was rapid and complete as compared with findings after stent graft repair, where complete regression is relatively rare. In patients who undergo AAA surgery with a polytetrafluoroethylene graft, however, aneurysm sac regression may follow a different course, where a perigraft seroma can produce similar aneurysm sac expansion.\(^8\)

If anastomotic leakage occurs into a shrunken aneurysm sac following replacement surgery with a Dacron graft, expansion of the aneurysm sac may result. This expansion is dangerous, as is true for typical anastomotic aneurysms; the replaced, shrunken aneurysm sac is likely to be more fragile than previously, and prone to rupture. It is probably due to the sac wall atrophy of the AAA that shrunk once. This phenomenon was also observed after transluminal stent graft repair.\(^9\) In our case, the aneurysm sac had dilated about 10 mm in the final 4 months, even though the resulting diameter was only 4.5 cm. Careful examination and treatment is necessary if an aneurysm sac expands after initial shrinkage following replacement surgery. After 1 year or more, dilation of a surgically replaced AAA sac may be a sign of anastomotic leakage with an endoleak requiring a second repair. Life-long imaging surveillance may be necessary following surgical replacement as well as stent graft repair of the AAA.

**REFERENCES**