Progression of Perianeurysmal Inflammation after Endovascular Aneurysm Repair for Inflammatory Abdominal Aortic and Bilateral Common Iliac Artery Aneurysms

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The use of endovascular aneurysm repair (EVAR) to treat inflammatory abdominal aortic aneurysms (IAAAs) has been reported, and this procedure appears to be preferable to open surgical repair because of intraoperative difficulties related to inflammation. We herein report a case of IAAA and bilateral inflammatory common iliac artery aneurysms that was successfully treated with bifurcated stent grafting. The perianeurysmal inflammation worsened postoperatively, requiring the placement of a ureteric stent. EVAR is feasible in cases of inflammatory aneurysms; however, the potential for an inflammatory response should be taken into account when considering the application of EVAR in patients with IAAA.

Inflammatory abdominal aortic aneurysms (IAAAs) constitute a minor subgroup of the total number of abdominal aortic aneurysms, with an incidence ranging from 2.2−18.1% according to published data.[1] Although IAAAs are traditionally treated with conventional open surgical repair (OSR), the consequent inflammatory reaction results in significant adhesion formation within the retroperitoneum, and the inflammatory process frequently involves neighboring structures, such as the ureters, duodenum, and vena cava.[2] Therefore, the perioperative mortality associated with OSR of IAAAs is higher than that observed in patients with noninflammatory aortic aneurysms.

Recently, several reports have indicated that endovascular aneurysm repair (EVAR) for IAAAs is an excellent alternative treatment, with promising perioperative and long-term results.[3] EVAR can be successfully and safely used to treat IAAAs; however, the benefits of this procedure in improving perianeurysmal inflammation (PAI) and subsequent renal complications, such as hydronephrosis, remain controversial.[4] We herein report the case of a patient whose inflammatory aneurysms were treated with EVAR, although postoperative hydronephrosis progressed because of PAI.

CASE REPORT

A 77-year-old man presented with lower abdominal pain. He had a history of hypertension, coronary artery disease, and chronic obstructive pulmonary disease. On admission, his body temperature was 36.8°C, and a physical examination showed a pulsatile abdominal mass, although there were no other remarkable findings. The laboratory results included a normal white blood cell (WBC) count ($7.8 \times 10^3/\mu L$), high level of C-reactive protein (CRP; 6.0 mg/dL), and elevated level of serum creatinine (1.11 mg/dL). All other laboratory values were within the normal range. Contrast-enhanced computed tomography (CT) showed an abdominal aortic aneurysm (maximum diameter, 71 mm), right common iliac artery aneurysm measuring 23 mm, and left common iliac artery aneurysm measuring 22 mm (Fig. 1). All the aneurysms exhibited PAI with fibrotic tissue. These laboratory and

Conflict of Interest: K.I. and all coauthors have nothing to declare.

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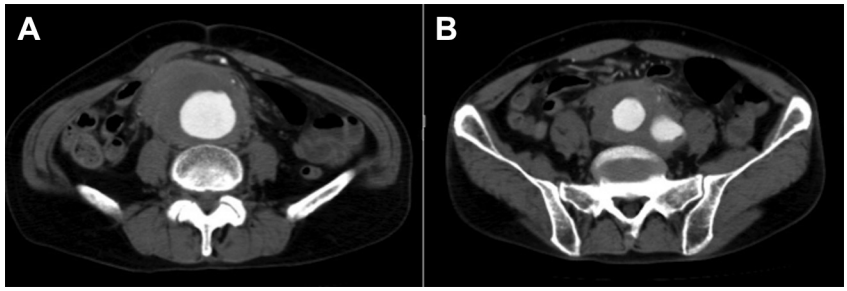

Fig. 1. Preoperative contrast-enhanced computed tomography showed an abdominal aortic aneurysm measuring 71 mm **(A)** and bilateral iliac artery aneurysm **(B)**. These aneurysms revealed perianeurysmal inflammation.

radiologic findings suggested a diagnosis of IAAA and bilateral inflammatory common iliac artery aneurysms. The patient also had severe systemic comorbidities and was therefore considered a high-risk candidate for open surgical therapy; therefore, EVAR was selected.

A Gore Excluder™ bifurcated stent prosthesis (W. L. Gore and Associates, Flagstaff, AZ) was implanted without complications via bilateral femoral access after coil embolization of the bilateral internal iliac arteries. Complete angiography showed a patent stent graft without endoleaks or enhancement of the abdominal aortic or bilateral common iliac artery aneurysms. The patient's postoperative course was uneventful, without the development of ischemic complications. On the seventh postoperative day, a CT scan showed a patent stent graft with type II endoleaks from the inferior mesenteric and lumbar arteries, which did not affect the enlargement of the abdominal aortic aneurysm (maximum diameter, 70 mm). In addition, the laboratory findings showed no remarkable changes in the WBC count ($5.4 \times 10^3/\mu L$), CRP level (4.3 mg/dL), or serum creatinine level (1.2 mg/dL), and the patient was discharged from the hospital 11 days after surgery.

The patient was subsequently readmitted with anorexia 2 months after undergoing surgical repair. The laboratory findings showed no remarkable changes in the degree of inflammation (WBC count, $6.2 \times 10^3/\mu L$ and CRP, 7.9 mg/dL); however, the renal function had deteriorated, with a serum creatinine level of 26.6 mg/dL. A plain CT scan showed worsening of the hydronephrosis in the bilateral kidneys (Fig. 2), although the abdominal aortic aneurysm had not increased in size (maximum diameter, 70 mm). Because aneurysmorrhaphy had been accomplished without enlargement of the aneurysm, we decided that open surgical conversion was not required to treat the hydronephrosis. Therefore, bilateral ureteric J-J stents were placed, and temporary hemodialysis was applied. One month after the procedure, the patient's renal function improved (serum creatinine, 1.63 mg/dL) without steroid therapy, and the bilateral ureteral stents were removed. A plain CT scan showed regression of the hydronephrosis in the left kidney, whereas that in the right kidney remained unchanged (Fig. 3).

Eighteen months after surgery for aneurysm repair, the laboratory findings showed resolution of the inflammatory changes (WBC count, $6.3 \times 10^3/\mu L$ and CRP, 0.5 mg/dL); however, the patient displayed chronic renal dysfunction, with a serum creatinine level of 1.5 mg/dL. Although ultrasonography showed no enlargement of the abdominal aortic aneurysm (maximum diameter, 65 mm), persistent hydronephrosis continued in the right kidney.

DISCUSSION

IAAAs are characterized by the presence of varying degrees of inflammatory infiltrates within the aneurysmal wall and perivascular and perineural tissues.[5] This inflammatory process obscures the normal tissue planes, resulting in injury to the adjacent structures, which tend to bleed more normal tissue. Patients with IAAAs sometimes present with serological autoimmune abnormalities, such as positivity for autoantibodies, or systemic autoimmune diseases.[6] Recently, a close relationship has been suggested between the titer of immunoglobulin G4 (IgG4) and the development of idiopathic IgG4-related sclerosing lesions. Kasashima et al.[7] reported that some IAAA patients also have IgG4-related sclerosing lesions and that IAAAs can be classified into IgG4-related and non−IgG4-related cases. Patients with IgG4-related lesions tend to exhibit an elevated serum IgG4 level and diffuse infiltration of IgG4-positive plasma cells in the aneurysmal adventitia. In the present case, the serum IgG4 level was within the normal limits (14.2 mg/dL), and the resected aneurysmal adventitia showed no filtration of IgG4-positive cells; therefore, the patient was diagnosed with non−IgG4-related IAAA.

Although IAAAs are sometimes treated with OSR, the procedure is often complicated because of the presence of inflammatory changes, such as fibrosis, around the aortic wall and adhesion to the duodenum and/or ureter. Such cases are associated with higher intraoperative and postoperative morbidity and mortality rates in patients with IAAAs than in those with