Endovascular stent-graft repair for penetrating atherosclerotic ulcer in the infrarenal abdominal aorta

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Purpose: **Penetrating atherosclerotic ulcer (PAU) is an ulceration of an atherosclerotic plaque penetrating through the intima, which may lead to intramural hematoma, aneurysm formation, or rupture. This disease is predominantly found in the thoracic aorta and is uncommon in the infrarenal aorta. The effectiveness of endovascular repair of PAU in the infrarenal aorta was retrospectively investigated.**

Methods: **From 1999 to 2002, PAU was diagnosed with computed tomography and magnetic resonance imaging in the abdominal aorta in four patients. All patients were men; their average age was 78 years. All four patients had hypertension, and two patients had concomitant coronary artery disease. Three patients had abdominal pain or lumbago.**

Results: **All patients underwent endovascular grafting with a Gianturco Z-stent covered with thin-wall woven Dacron graft. Indications for endovascular intervention were aneurysm formation with or without intramural hematoma in two patients and contained rupture with extraaortic hematoma in two patients. The postoperative course was uneventful in all cases, and no endoleak or aneurysm expansion was recognized during follow-up (4-32 months; average, 14 months).**

Conclusions: **Infrarenal aortic lesions caused by PAU were generally localized, and endovascular grafting appears to be a feasible alternative to surgical repair. (J Vasc Surg 2003;38:383-8.)**

Penetrating atherosclerotic ulcer (PAU) of the aorta is ulceration of an atherosclerotic plaque penetrating through the intima, and it may be complicated by aortic intramural hematoma, adventitial pseudoaneurysm formation, or aortic rupture[1,2] (Fig 1). PAU occurs predominantly in the descending thoracic aorta, and it is uncommon in the infrarenal aorta.[3,4] We reviewed clinical features of PAU in the infrarenal aorta and investigated the usefulness of endovascular repair for this rare lesion.

METHODS

Patient characteristics. Between 1999 and 2002, PAU of the abdominal aorta was diagnosed in four patients (Table). All patients were men, and their ages ranged from 66 to 89 years (average age, 78 years). Three patients previously had abdominal pain or lumbago. Hypertension was preoperatively detected in all four patients, and coronary artery disease was detected in two patients.

PAU was diagnosed at computed tomography (CT) and magnetic resonance imaging (MRI). Intramural hematoma with aortic expansion (case 1; Fig 2*A*,), adventitial pseudoaneurysm formation without intramural hematoma (case 2; Fig 3*A*,), and contained ruptures with extra-aortic hematoma (cases 3 and 4; Fig 4*A*,) were recognized. Aortography was performed to assess the femoral access route, anatomy of the abdominal aorta (Fig 4*B*), and location of PAU (Fig 2, *B*; Fig 4, *B*).

Endovascular procedure. The stent graft was constructed of a self-expanding Gianturco Z-stent (GZV; Cook Inc, Bjaeverskov, Denmark) covered with thin-wall Dacron graft (UBE woven graft, porosity 150 mL/min/cm^2; Ube Inc, Yamaguchi, Japan).

After systemic heparinization and preoperative aortography, an 18F delivery sheath catheter (catheter introducer kit; Medikit Co, Tokyo, Japan) with the stent graft loaded inside was introduced into the femoral artery over the guide wire. The stent graft was deployed over the lesion under fluoroscopic guidance and tug-of-wire maneuvering. Balloon dilation was usually used also. Finally, completion aortography was performed to confirm successful deployment of the stent graft.

In three cases endovascular stent grafting was performed with the patient under local anesthesia in an angiography suite. One patient who had adventitial pseudoaneurysm after PAU and acute cholecystitis underwent simultaneous repair of both complications in the operating theater. With the patient under general anesthesia, endovascular stent grafting was performed first, from the femoral approach, followed by cholecystectomy through a right subcostal incision.

To evaluate endoleak, stent-graft migration, and aneurysm diameter, follow-up contrast material–enhanced CT was performed before hospital discharge and every 3 to 6 months thereafter.

RESULTS

Deployment of the stent graft was technically successful in all patients. Aortograms obtained after the procedure demonstrated no endoleak and complete exclusion of

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Competition of interest: none.

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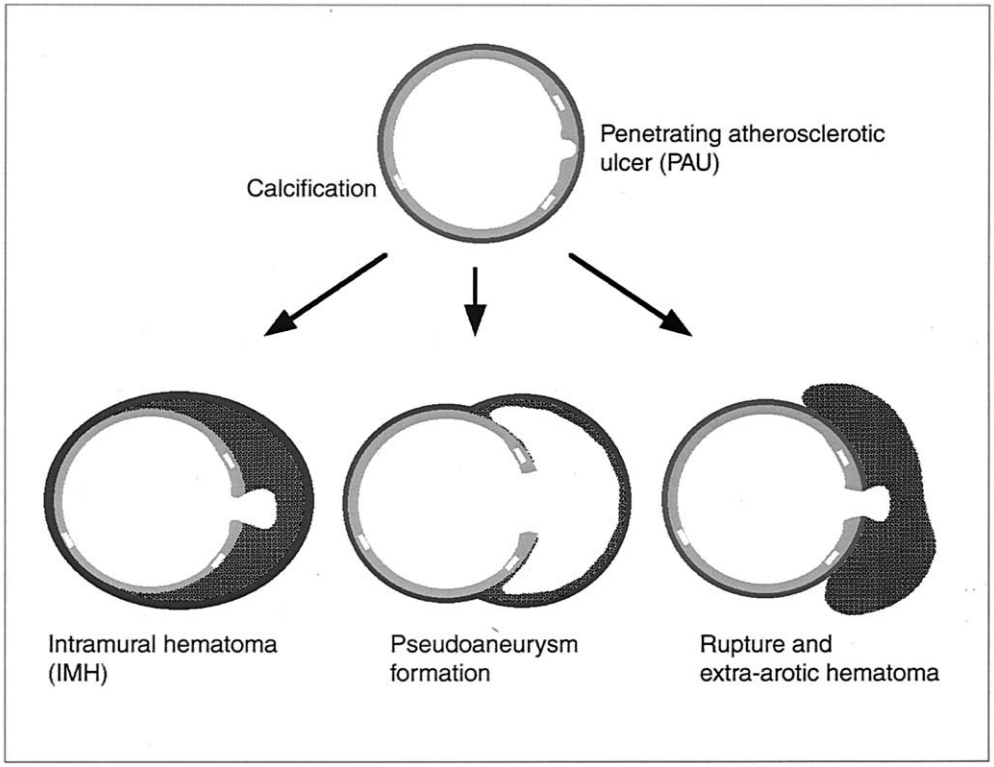

Fig 1. Schema of clinical course of penetrating atherosclerotic ulcer of the aorta. This lesion may lead to aortic intramural hematoma, adventitial pseudoaneurysm, and rupture with extra-aortic hematoma. Calcified plaques detected on plain CT scans are useful markers for differential diagnosis of intramural hematoma and intraluminal thrombus, and true aneurysm and adventitial pseudoaneurysm.

Table 1. Clinical characteristics of patients with PAU in infrarenal abdominal aorta

							Radiologic findings			
Patient	*Age (y)*	*Gender*	*Onset*	*Symptoms*	*HT*	*CAD*	*Shaggy aorta*	*Calcification*	*Associated condition*	*Prognosis (mo)*
1	72	M	Chronic	None	+	−	−	+	Intramural hematoma	Alive (32)
2	89	M	Acute	Abdominal pain	+	NA	−	+	Adventitial pseudoaneurysm	Death (7)
3	85	M	Chronic	Abdominal pain	+	+	+	+	Contained rupture with hematoma	Alive (11)
4	66	M	Chronic	Lumbago	+	+	+	+	Contained rupture with hematoma	Alive (4)

CAD, Coronary artery disease; *HT*, hypertension; *NA*, not assessed; *PAU*, penetrating atherosclerotic ulceration.

PAU or pseudoaneurysm (Fig 2*C*). Mean operative time was 115.5 minutes (range, 90-165 minutes). Mean blood loss was less than 100 g, and no blood transfusion was needed.

Postoperative course in all patients was uneventful. Contrast-enhanced CT scans obtained before hospital discharge demonstrated that all stent grafts were in the correct position, and all types of endoleak were absent (Fig 3*B*). Mean hospital stay after the operation was 13.8 days (range, 8-26 days).

During follow-up of 4 to 32 months (average, 14 months) no endoleak or aneurysm expansion was recognized in any patient. Average aneurysm shrinkage ratio (postoperative/preoperative aneurysm diameter) was 0.9 (range, 0.8-1.0) at 6-month follow-up and 0.87 (range, 0.73-1.0) at 12 months. One 89-year-old man died of nonrelated disease 7 months after the operation. The remaining three patients are alive without recurrence of disease.

DISCUSSION

PAU of the aorta was first described as a distinct clinical and pathologic entity by Stanson et al[1] in 1986. The condition is characterized by ulceration of an atheroscle-

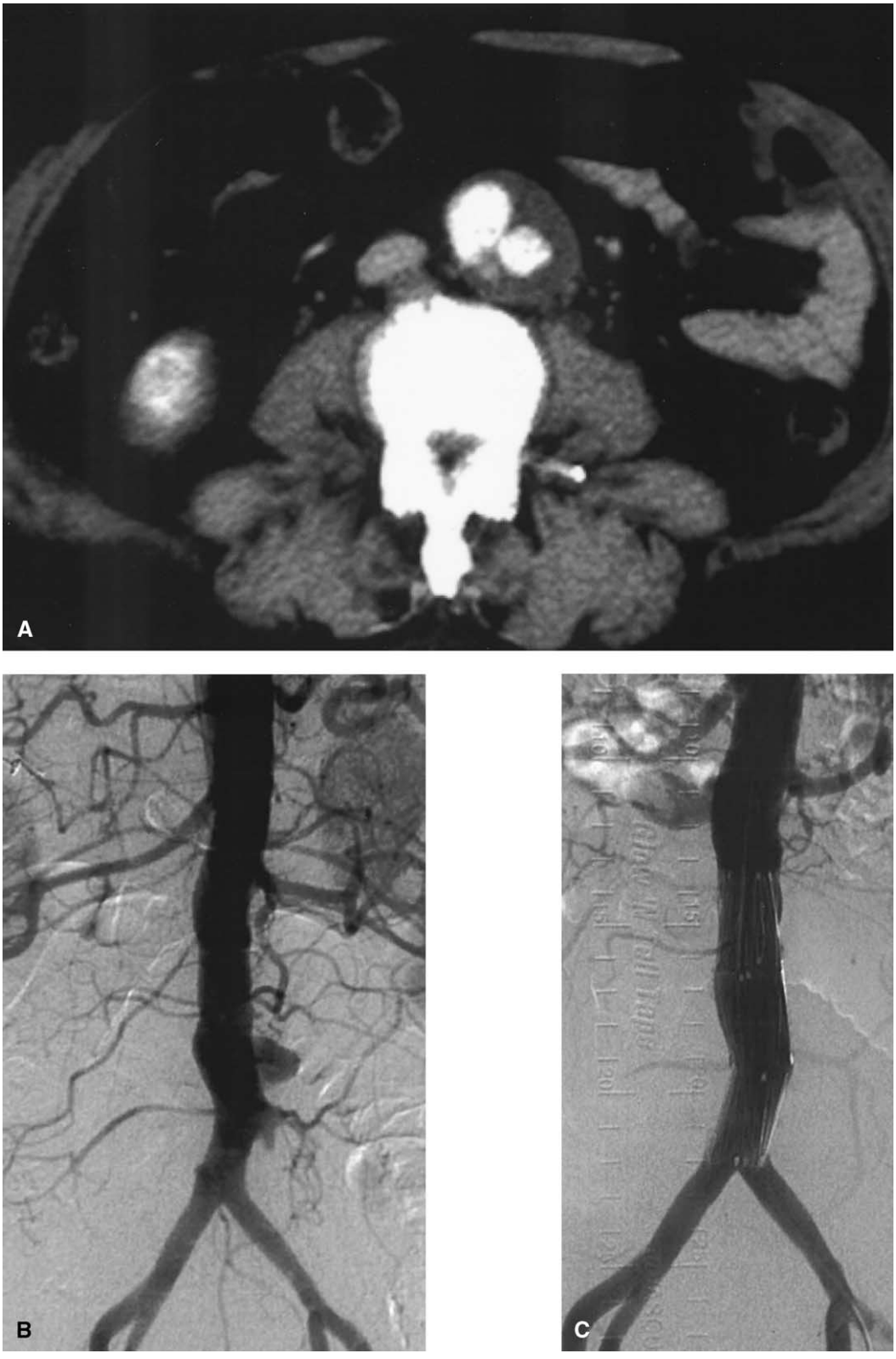

Fig 2. Patient 1. Penetrating atherosclerotic ulcer in the abdominal aorta, with intramural hematoma. **A,** Preoperative contrast-enhanced CT scan. **B**, Preoperative aortogram shows localized contrast-filled outpouching in the infrarenal aorta. **C**, Aortogram after deployment of the stent graft demonstrates that the lesion has been completely sealed.

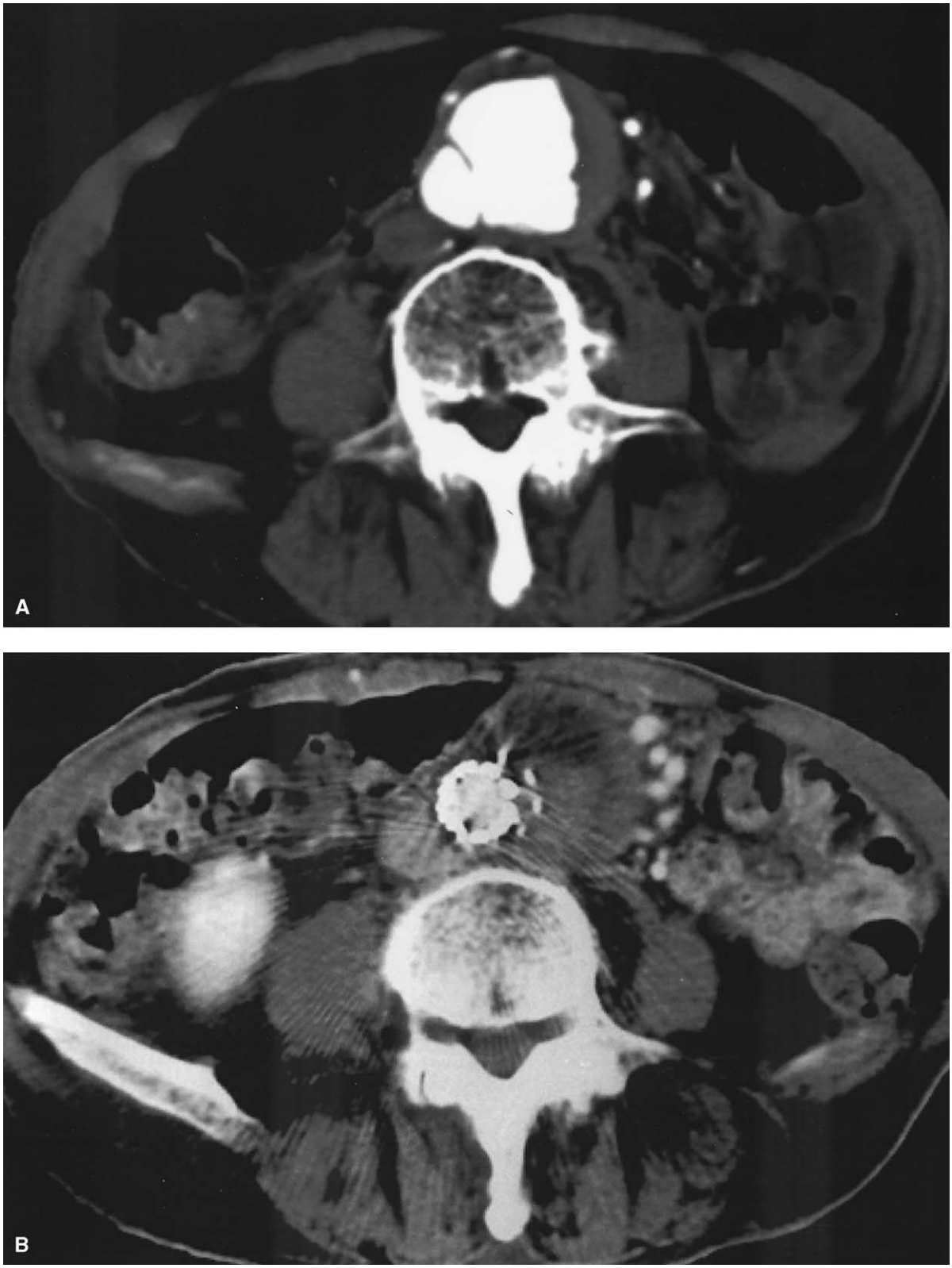

Fig. 3. Patient 2. Penetrating atherosclerotic ulceration in the abdominal aorta with pseudoaneurysm. **A,** Preoperative contrast-enhanced CT scan. **B**, Contrast-enhanced CT scan obtained 8 days after endovascular stent grafting demonstrates complete exclusion of the lesion and the adventitial pseudoaneurysm.

rotic plaque that penetrates through the elastic lamina and into the media. Subsequently, hematoma formation may extend along the media, and in some cases hematoma extension causes stretching of the weakened aortic adventitia, forming a saccular pseudoaneurysm. Intramural hematoma and pseudoaneurysm associated with PAU may eventually rupture, and it may be speculated that some spontaneous aortic ruptures in elderly patients may be due to perforation of PAU. PAU should be distinguished from true aneurysm and classic aortic dissection; however, differentiation of these disease entities is sometimes difficult, and there seems to be confusion regarding the concept of PAU.[5]

PAU generally affects elderly patients with advanced athrosclerosis. As a result, PAU is associated with a high incidence of morbidity, eg, hypertension, coronary artery disease, and carotid artery occlusive disease. All four patients in this series have received antihypertensive drugs, and two patients had coronary artery disease.

PAU may be diagnosed at CT, MRI, and conventional aortography; however, a high degree of clinical suspicion is necessary for diagnosis of PAU. Extensive aortic calcification is often detected on plain CT scans, and development of intramural hematoma after PAU versus intraluminal thrombus can be differentiated by the location of calcified plaques in relation to the thrombus on plain CT scans. On contrast-enhanced CT scans, PAU is recognized as a contrast-filled, pouchlike aortic protrusion without a dissection flap or false lumen. The intramural hematoma, advential pseudoaneurysm, and rupture with extra-aortic hematoma may also be seen. However, it is sometimes difficult to differentiate extra-aortic hematoma after rupture of PAU and periaortic tumors invading the aortic wall.[6] MRI is useful in differential diagnosis in such cases, and MRI has the additional advantage of allowing multiplane imaging without use of contrast medium. [7,8]

Some authors believe immediate surgical treatment is not always required, because most PAU have a benign clinical course.[3,4] However, early intervention is recommended when PAU is complicated with aneurysm expansion, rupture, embolic symptoms, or uncontrolled pain. Open surgical repair with graft interposition has been used for such symptomatic PAU, but patients with PAU are generally not ideal candidates for open repair because of advanced age and poor general status. High operative morbidity and mortality associated with open repair have been reported.[1,9]

As a less invasive treatment for this disease, endovascular stent grafting was first used by the Stanford group.[10,11] Subsequently, several reports of successful endovascular treatment of PAU have been published.[4,6,12-16] Review of the 32 cases reported to date, including our own, reveals that deployment of the stent graft was successful in all. Early complications have included transient or permanent paraplegia in three patients, and primary endoleak, which spontaneously regressed, in four patients. During follow-up, aneurysm recurrence was reported as a treatment-related complication in two patients, and was suspected to be

Fig. 4. Patient 4. Penetrating atherosclerotic ulceration in the abdominal aorta with extra-aortic hematoma. **A,** Preoperative contrast-enhanced CT scan. **B**, Preoperative aortogram shows contrast-filled protrusion in the infrarenal shaggy aorta.

caused by persistent endoleak and iatrogenic intimal injury during deployment of the stent graft. As most patients who received the stent grafts to repair PAU were in a high-risk cohort, the advantages of stent grafting are changing the strategy for treatment of symptomatic PAU. The lower morbidity and mortality after stent-graft repair also supports a more aggressive approach to this lesion, even in patients with symptoms.

PAU typically involves the descending thoracic aorta, and it is comparatively rare that PAU develops in the infrarenal abdominal aorta. To our knowledge, 18 patients with PAU in the infrarenal abdominal aorta have been reported. Fourteen were followed up medically, and 4 underwent open surgical repair.[2-4,17,18] We have found no report dealing with endovascular stent grafting for treatment of PAU in the infrarenal abdominal aorta.

In conclusion, infrarenal aortic lesions caused by PAU are generally localized, and endovascular stent grafting for repair of this disease appears to be a feasible alternative to surgical repair. Initial results of endovascular treatment of PAU are satisfactory; however, further investigations of the long-term results of this procedure are necessary.

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