International Journal of Surgery (2007) 5, 172–175

INTERNATIONAL JOURNAL OF SURGERY
www.theijs.com

Endovascular repair of abdominal infrarenal penetrating aortic ulcers: A prospective observational study

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KEYWORDS
Abdominal penetrating aortic ulcers;
Endovascular repair

Abstract *Objective:* Penetrating atherosclerotic ulcer generally occurs in elderly patients with systemic atherosclerosis, predominantly in the descending thoracic aorta, and it is uncommon in the infrarenal aorta. We reviewed our experience of endovascular treatment of penetrating aortic ulcer in the infrarenal aorta.
Methods: In the last 4 years, out of 348 patients who underwent abdominal aortic procedures, a total of 13 patients (12 men and 1 woman) were found to have an abdominal penetrating aortic ulcer, corresponding to an incidence of 3.7%. Mean age was 73 ± 7 years. All patients had hypertension. Three lesions were discovered incidentally and 10 were symptomatic. All patients underwent endovascular treatment in the operating room. Follow-up included CT-A control at 1, 4 and 12 months after the intervention, and yearly thereafter.
Results: Primary technical success was 100%. No postoperative death was observed. Mean operative time was 100 ± 29 min. Mean blood loss was 168 ± 133 ml. No patient required intensive care unit stay. We observed one major complication (transient ischemic attack). Mean hospital stay was 4 ± 1 days. During a mean follow-up period of 26 months no endoleak, aneurysm evolution or stent graft failure was recognized in any patient. One patient died 24 months after the intervention after a stroke.
Conclusions: In our experience, endovascular or repair of infrarenal aortic ulcer appears feasible, and midterm results satisfactory.

Introduction

Penetrating atherosclerotic ulcer (PAU) of the aorta is defined as atherosclerotic lesion with ulceration of the aortic intima and media, and rupture of the internal elastic

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doi:10.1016/j.ijsu.2006.06.004

lamina.[1–3] PAU occurs in elderly patients with systemic atherosclerosis, predominantly in the descending thoracic aorta, and it is uncommon in the infrarenal aorta.

Although first described by Shennan in 1934,[4] their natural history still remains unclear and treatment is controversial; it may be complicated by aortic intramural hematoma, adventitial pseudoaneurysm formation, or aortic rupture.[5]

We reviewed the clinical features of PAU in the infrarenal aorta and investigated the usefulness of endovascular repair for this uncommon lesion.

Materials and methods

In the last 50 months, 348 patients underwent abdominal aortic procedures at our institution for atherosclerotic disease (aneurysms ($n = 293$), obstructive disease ($n = 55$)); overall, 251 patients were treated endovascularly, 97 with open surgery. A total of 13 patients were found to have an abdominal PAU, corresponding to an incidence of 3.7%. They were analyzed in a retrospective manner: there were 12 men and 1 woman in this series; mean age was 73 ± 7 years (range 56–84, median 77). All patients had hypertension (on anti-hypertensive medication); additional co-morbidities and risk factors included peripheral obstructive arterial disease ($n = 6$), diabetes ($n = 5$), chronic obstructive pulmonary disease ($n = 3$), cerebrovascular insufficiency ($n = 3$), chronic renal failure ($n = 2$), hostile abdomen ($n = 2$), coronary artery disease ($n = 2$), and hyperlipidemia ($n = 1$).

Three lesions were discovered incidentally during aorto-iliac duplex ultrasound (US) screening examination and were treated electively; of the 10 patients with symptoms, five had claudication and the presence of the PAU was suspected during preliminary US, three had critical ischemia of the lower extremities due to arterial embolism, and two complained of abdominal pain (one associated with syncope that suggested a ruptured abdominal aorta).

In every patient, PAU was diagnosed at computed tomography angiography (CT-A): on CT-A, PAU is recognized as a contrast-filled, pouch-like aortic protrusion without a dissection flap or false lumen (Fig. 1A,B). Intramural hematoma with aortic expansion, adventitial pseudoaneurysm formation without intramural hematoma, and contained ruptures with extra-aortic hematoma were recognized. Because they were treated electively, it was not possible to obtain an histological diagnosis to support the CT diagnosis of PAU. CT-A also demonstrated associated atherosclerotic arterial lesions in eight patients: stenosis of an iliac artery ($n = 5$; four bilateral, one unilateral), and stenosis of the internal carotid artery ($n = 2$). CT also revealed thrombus in the abdominal aorta ($n = 5$) and extensive aortic calcifications ($n = 4$).

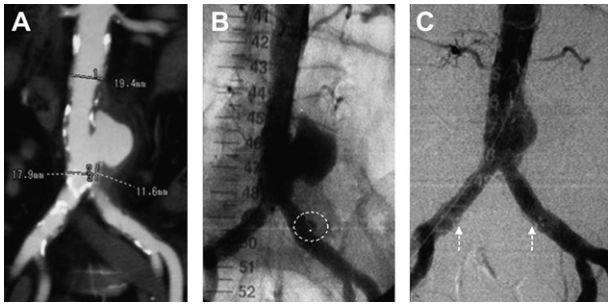

Figure 1 Preoperative CT-angiography of an infrarenal PAU with a 55 mm aortic expansion (A). Intraoperative angiogram (B) confirmed the presence of the PAU just above the aorto-iliac bifurcation and the ulceration (ring) of a plaque of the left common iliac artery; both the lesions were excluded with two abdominal ''cuffs'' and a ''kissing''-stent grafts (arrows) reconstruction of the aorto-iliac bifurcation (C).

All patients in this series underwent endovascular treatment with stent graft (SG); the devices used were cuffs of Excluder® (W.L. Gore and Associates, Flagstaff, AZ, USA), Zenith® (Cook Inc., Bloomington, IN, USA), and Lifepath® (Edwards Lifesciences Corp., Irvine, CA, USA). Repairs were performed in the operating room with the patient under loco-regional anesthesia ($n = 11$), or general anesthesia with endotracheal intubation ($n = 2$); a cell-saver system (Compact-Dideco®; Modena, Italy) was available in the event that surgical conversion was needed. Patients were prepared and draped for either femoral arteriotomy and traditional transperitoneal approach. Every patient received short-term antibiotic prophylaxis (vancomycin 1 g b.i.d.). The common femoral artery was exposed in standard fashion for device access in all patients. Completion digital subtraction angiography (DSA) was routinely performed at the end of the procedure to confirm adequate position of the SG, the complete exclusion of the lesion and detect potential endoleaks (Fig. 1C).

All patients underwent follow-up CT-A control, at 1, 4 and 12 months after the intervention, and yearly thereafter. Data for variables were expressed as mean values. Data were prospectively collected and analyzed in a retrospective manner. Primary outcome measures were the exclusion of the PAU and patient survival.

Results

Overall, lesions were recognized as follows: PAU ($n = 6$), pseudoaneurysm ($n = 6$), and rupture ($n = 1$). Deployment of the SG was technically successful in all patients. No postoperative deaths occurred. A total of 17 SGs were used: ten patients received one SG, two patients two SGs, and one patients three SGs. Mean SG diameter was 25 ± 3 (range 20–28, median 24). Aortograms obtained after the procedure demonstrated complete exclusion of PAU and no endoleak. Additional procedures included bilateral iliac arteries PTA/stent ($n = 4$), iliac artery PTA ($n = 1$), and internal carotid artery stenting ($n = 2$). Mean operative time was 100 ± 29 min (range 60–150, median 80). Mean blood loss was 168 ± 133 ml (range 50–500, median 120); blood transfusion (2 units of packed red blood cells) was needed in the patient with a ruptured PAU.

Intensive care unit stay was never required; postoperative course was uneventful in all but one patient. We observed a cerebrovascular event that spontaneously resolved in a patient who underwent simultaneous carotid stenting. Mean hospital stay after the operation was 4 ± 1 days (range 3–8, median 4).

During a mean follow-up period of 26 months (range 3–60, median 24) no endoleak, aneurysm evolution or SG failure was recognized in any patient. In this series no patient was lost to follow-up; one patient died 24 months after the intervention after a stroke.

Discussion

PAU of the aorta was first identified by Shennan in 1934, but described as a distinct clinical and pathologic entity by Stanson in 1986.[3,4] Stanson defines a PAU as an atherosclerotic lesion with an ulceration that penetrates the internal elastic lamina and the media of the aortic wall; 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. PAU generally affects elderly patients with advanced atherosclerosis: as a result, PAU is associated with a high incidence of morbidity, such as hypertension, coronary artery disease, and carotid artery occlusive disease.[6] All patients in this series have received antihypertensive drugs, and three patients had multiple (carotid, coronary and peripheral) arterial disease. Of interest, in terms of age we noted a significant difference between patients with PAU if compared to patients who received aortic repair for peripheral obstructive disease (73 ± 7 vs 66 ± 10, $p < 0.05$), but no statistical difference if compared to patients with aneurysmal disease (73 ± 7 vs 72 ± 8, $p =$ NS).

PAU typically involves the descending thoracic aorta, and it is comparatively rare that PAU develops in the infrarenal abdominal aorta; few reports have dealt with endovascular stent grafting for treatment of PAU in the infrarenal abdominal aorta.[7–9] The true incidence of PAU has yet to be determined. No literature study to date has appropriately addressed this question. The incidence of thoracic PAU varies from 2 to 7% while abdominal PAU has been considered responsible for 1–5% of all aortic ruptures.[3,5–7] In our series abdominal PAU accounted for 3.7% of all aortic procedures over almost 4-years.

It has been reported that the incidence of these lesions also depends on the diagnostic method; in fact, most cases reported in the medical literature regarding the imaging appearance of PAU or endovascular treatment do not have histologic proof and the diagnosis of atherosclerotic lesion is assumed but unconfirmed. Differentiation of these disease entities is sometimes difficult, and there seems to be confusion regarding the concept of PAU.[3,5,7,10] For these reasons, Quint referred to these lesions as ''ulcerlike lesions'' of the aorta rather than PAU. PAU may be diagnosed at CT, magnetic resonance imaging (MRI), and conventional aortography; 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, pouch-like aortic protrusion without a dissection flap or false lumen.[2,5,11] An intramural hematoma, advential pseudoaneurysm, and rupture with extra-aortic hematoma may also be seen. We believe CT is more accurate, and is the most commonly used technique. MRI appears more effective because it reduces the rate of false negative diagnoses, but it is less available than CT; in our series, we detected six PAU, six pseudoaneurysm, and one rupture.

PAU are the subject of considerable controversy with respect to definition as well as natural history. The natural history of abdominal PAU remains unclear and little information is available in the literature.[5] The unpredictable course of PAU was previously emphasized; in particular, some patients had distal ischemia caused by embolism from a PAU.[3] Embolization from penetrating aortic ulcers has been considered rare, but lower limb embolisms were reported to be more frequent with abdominal PAU than with thoracic PAU.[3,12,13] Harris reported three patients with complicated forms of abdominal PAU: two patients had distal ischemia caused by embolism, one of them with recurrent embolism that required amputation. Our series confirms that when there are no signs suggesting a cardiac origin, discovery of lower limb embolism should prompt a search for an aortic cause, and in particular PAU.

The treatment of PAU remains controversial too. Although some authors believe immediate surgical treatment is not always required, because most PAU have a benign clinical course, early intervention has been recommended when PAU is complicated with aneurysm expansion regardless of size, rupture, embolic symptoms, or uncontrolled pain.[1,2,5,10,11] Open surgical repair with graft interposition has been used traditionally, but patients with PAU are generally not ideal candidates for open repair because of advanced age and poor general status.[1,2,7–10] In fact, high operative morbidity and mortality associated with open repair have been reported.[7] Use of aortic SGs could probably change the strategy for treatment of PAU. This less invasive procedure is suitable for high risk patients, and can also be used in cases of rupture.

As a less invasive treatment for this disease, endovascular stent grafting was advocated and several reports of endovascular treatment of PAU have been published.[7–9,13,14] Early complications for abdominal PAU included endoleak mainly; peripheral embolization could be a potential intraoperative complication as well as in the post-operative course. We opted to use a SG in order to prevent potential debris migration or fragmentation from the original PAU during the positioning or deployment of the device, and to exclude completely the potential aneurysm evolution of the PAU from the systemic flow. Review of all cases reported to date, including our own mid-term results, reveals that deployment of the SGs was successful in all; thus, the lower morbidity and mortality after endovascular repair could also support a more aggressive approach to this lesion, even in patients without symptoms.

Conclusions

In conclusion, this is the largest experience of stent graft treatment for abdominal PAU. Infrarenal aortic lesions caused by PAU are generally localized, and endovascular SG repair of this disease appears to be a feasible alternative

to surgical repair. Mid-term 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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