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Does the Presence of an Iliac Aneurysm Affect Outcome of Endoluminal AAA Repair? An Analysis of 336 Cases[†]

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Objective: to determine whether the presence of an iliac aneurysm compromises outcome of endovascular exclusion of AAA and to ascertain the fate of the iliac aneurysmal sac.

Patients and methods: between April 1997 and March 2001, data on 336 consecutive patients undergoing endovascular repair for AAA were entered in a prospective database. Suitability for endovascular repair was assessed by preoperative contrast-enhanced computed tomography. A maximum common iliac artery (CIA) diameter ≥ 20 mm was defined as iliac aneurysm. Patients with and without iliac aneurysms were compared to early (immediate conversion or perioperative death) and late failure (increase in aneurysm diameter or persisting graft-related endoleak, or late AAA rupture or conversion).

Results: fifty-nine patients (18%) had iliac aneurysms, 19 were bilateral, for a total of 78 aneurysmal iliac arteries (median diameter 23 mm; range 20–50 mm). A distal seal was achieved by landing in 33 external iliac arteries, in 20 ectatic CIAs, and in 25 normal CIAs. Operating time differed significantly between patients with and without CIA aneurysms (153 ± 71 vs 123 ± 55 min, $p = 0.0001$), whereas no statistically significant differences were found with respect to early and late failure (2% vs 3%, $p = 0.5$ and 14% vs 8%, $p = 0.11$, respectively). There were no cases of buttock or colon necrosis. At a median follow-up of 14 months (range 0–46; i.q.r. 7–27 months) common iliac diameter decreased ≥ 2 mm in 49 cases, remained stable in 25, and increased ≥ 2 mm in 3.

Conclusion: the presence of iliac aneurysm rendered endoluminal AAA repair more complex but did not affect feasibility and long-term outcome of the procedure. In our experience internal iliac exclusion was never associated with significant morbidity. These data may be useful when considering endovascular repair in high-risk patients with challenging anatomy.

Key Words: AAA; Aortoiliac aneurysm; Endovascular treatment.

Introduction

Patient selection for endoluminal AAA repair depends on aortoiliac anatomy, which must meet the requirements of available endografts. The common iliac artery (CIA) serves as the distal endograft implantation site and must be of suitable diameter to allow adequate haemostatic seal and fixation. The presence of a CIA aneurysm represents a major limitation to accomplishing full exclusion of the aneurysm with the stent graft and may compromise technical success of endoluminal repair. Different techniques can be employed to lengthen the distal anchoring site in the presence of CIA such as direct revascularisation of the internal iliac artery, followed by proximal ligation and

transection of the vessel, or occlusion of the internal iliac artery. In the case of ectatic, non-aneurysmal iliac arteries, an aortic extension cuff can be inserted into the distal portion of the iliac limb of a bifurcated endograft, resulting in a flared iliac limb or “bell bottom” configuration.¹ Aim of the present study was to determine whether the presence of an iliac aneurysm affects outcome of endovascular exclusion of AAA in terms of incidence of graft-related endoleaks, AAA rupture, AAA growth, and to ascertain the fate of the iliac aneurysmal sac.

Methods

Patients

Between April 1997 and March 2001, 336 consecutive patients underwent endovascular repair for AAA at the Unità Operativa di Chirurgia Vascolare, Policlinico

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Monteluce, Perugia, Italy. Preoperative, operative, and follow-up data of patients were entered in a prospective database. Indications for treatment of AAA included AAA of 5 cm or greater, or AAA ≥ 4 cm that increased rapidly in size (>0.5 cm in the previous 6 months), or became tender, or was associated with an iliac aneurysm greater than 3 cm.

Preoperative imaging

AAA diameter was assessed with computed tomography (CT), considering the maximum external diameter of the vessel. When the aneurysm diameter had an elliptical shape, the smaller diagonal was considered, thus avoiding errors in size evaluation due to aortic tortuosity.² Selection criteria for endoluminal repair included a proximal aneurysm neck length ≥ 15 mm and a diameter ≤ 30 mm, aortic neck angulation $<75^\circ$, external iliac artery >6 mm in diameter without excessive tortuosity, calcification, or stenosis.

Anatomical features were evaluated with a combination of pre-procedural imaging techniques including contrast-enhanced, conventional, or spiral CT scan with 5 mm cuts or less, colour duplex scan, and angiography or angio-magnetic resonance with gadolinium. Aneurysm extension was recorded according to the EUROSTAR classification.³ CIAs with diameter ≥ 20 mm involving at least 2/3 the length of the vessel were considered aneurysmal (EUROSTAR classification class D or E).

Procedures

All endovascular procedures were performed in the operating room with patients under general or spinal anesthesia. The surgical team included two vascular surgeons and one interventional radiologist. Intraoperative radiological imaging was performed with a portable C-arm fluoroscopic device (9000 and 9800 OEC, Diasonics) with digital imaging and road mapping capability on a compatible operating table. Initial assessment of endograft function and position, and verification of satisfactory exclusion of the AAA was evaluated by intraoperative post-deployment angiography, pre-discharge colour duplex scan, and plain abdominal radiographs.

When an adequate segment of CIA distal to the aneurysm was present, the distal stent of the endograft was positioned in the CIA proximal to the internal iliac artery origin. If the CIA was aneurysmal at the level of the bifurcation, the endograft was extended into the external iliac artery and, selectively, these

patients were considered for simultaneous internal iliac coil embolisation to prevent retrograde flow, or internal iliac revascularisation. In the case of CIA diameter between 20 and 22 mm within 1 cm of the internal iliac, a bell-bottom approach was used.¹

Based on the anatomical features of patients, different endografts were used: 228 (68%) AneuRx, Medtronic (Santa Rosa, CA, U.S.A.), 11 (3%) Anaconda Sulzer-Vascutek (Edinburgh, U.K.), 27 (8%) Zenith Cook (William Cook Europe, Biaeverskow, Denmark), 2 (0.6%) Endologix (Irvine, CA, U.S.A.), 39 (12%) Excluder Gore-Tex (WL Gore and Associates, Flagstaff, AZ, U.S.A.), 29 (9%) Talent Medtronic (World Medical-Medtronic, Sunrise, FL, U.S.A.).

Surveillance

Clinical evaluation, colour duplex scan (ATL HDI 3000), and plain abdominal radiography were repeated 1, 6, 12 months after the procedure and every 6 months thereafter. Contrast-enhanced CT scan was repeated 1 month after surgery and then annually. CT images were stored in a computer database. Two vascular specialists examined CT imaging for the presence of endoleak, and AAA and iliac diameter assessment. Measurements of CIA were obtained at each follow-up visit with the use of geometric calipers at the maximum diameter perpendicular to the supposed vessel axis on axial reconstruction. To determine the evolution of the iliac aneurysmal sac, maximum iliac diameters on the preoperative CT scan were compared to those of the last available CT in follow-up. Immediate conversion to open repair or perioperative death (within 30 days) were considered early failures; increase of aneurysm diameter during follow-up, or persistent graft-related endoleak, or AAA rupture, or conversion to open repair were considered late failures of the endovascular procedure.

Statistics

Patients without iliac aneurysm (Group A, EUROSTAR class A–C) and with CIA aneurysm (Group B, EUROSTAR class D–E) were compared to early and late failure. Data was expressed as median (with interquartile range) when appropriate. Comparison between patients in the two groups was performed with two-sample *t*-test or Mann–Whitney *U*-test (when appropriate) for continuous variables and χ^2 or Fisher exact test for discrete variables. Statistical analysis was conducted with SPSS software

(SPSS Inc., Chicago, IL, U.S.A.). $p \leq 0.05$ was considered statistically significant.

Results

Three hundred and thirty-six patients underwent endoluminal abdominal aortic aneurysm repair in the study period: 277 patients did not have CIA aneurysms (82%, Group A), while 59 (18%) had concomitant iliac aneurysms (Group B). Nineteen patients had bilateral CIA aneurysms for a total of 78 aneurysmal iliac arteries with a median CIA diameter of 23 mm (i.q.r. 21–28.5 mm; range 20–50 mm). Seven aneurysms involved the internal iliac artery. Demographics and risk factors of the two study groups are shown in Table 1.

Median AAA diameter was 50 mm (i.q.r. 45–55 mm; range 40–86 mm) in group A and 52 mm in group B (i.q.r. 48–56 mm; range 40–74 mm; $p = \text{NS}$). In group B, distal seal was achieved by landing: (1) in the external iliac artery excluding the internal iliac artery in 33/78 (42%) cases (bilateral in five), (2) in the ectatic CIA adjacent to the CIA aneurysm in 20/78 (26%) cases and (3) in the distal third of CIA with normal calibre in the remaining 25 cases. In 8/33 (24%) patients with a distal landing site in the external iliac artery, internal iliac embolisation was performed to exclude refilling into the aneurysm sac. In one of the seven aneurysms extended to iliac internal artery, embolisation was performed. Operative details of the two study groups are shown in Table 2.

There was one immediate conversion to open repair in Group B and five conversions in Group A. Outcome measures of the two study groups are displayed in Table 3. Overall perioperative major morbidity rate was 5% and included two acute myocardial infarctions, two congestive heart failures, one stroke, one pancreatitis, one renal infarction, one

intraparenchymal renal haemorrhage, one bleeding duodenal ulcer, four limb graft occlusions, one above-knee amputation in a patient affected by critical limb ischaemias and aortic pseudoaneurysm, two colonic mucosal ischaemia not requiring surgical therapy (both in group A), one graft infection (femoral–femoral bypass in a patient with aortouniliac device). Perioperative mortality occurred in four patients (1.4%), all in group A. Causes of perioperative deaths were congestive heart failure occurring two days after endoluminal repair in a patient with severe respiratory and cardiac disease, pulmonary oedema (28° post-operative day), massive haemorrhage from intraprocedural aortic rupture requiring immediate conversion to open repair, and sepsis in a patient affected by chronic leukaemia and tender AAA.

The 30-day endoleak rate was 8% in both groups respectively (Table 3). Three type 1 endoleaks (all proximal) and no endoleaks type 3 were detected in Group A whereas one type 3 endoleaks (refilling between distal cuff and iliac branch) and no type 1 endoleak occurred in Group B.

At a median follow-up of 14 months (i.q.r. 7–27 months; range 1–46 months), overall late mortality occurred in 21 patients (6%): 18 patients (6.5%) in

Table 2. Operative data for Group A and Group B patients. Mean values (\pm SD).

	Group A <i>n</i> = 277	Group B <i>n</i> = 59	<i>p</i> value
Surgical time (minutes)	123 (\pm 55)	153 (\pm 71)	0.0001
Blood loss (ml)	293 (\pm 692)	445 (\pm 521)	NS
Medium contrast used	148 (\pm 59)	182 (\pm 75)	0.0001
Fluoroscopy time	22 (\pm 10)	27 (\pm 15)	0.001

NS, not significant.

Table 3. Outcome parameters in Group A and Group B.

	Group A <i>n</i> = 277 (%)	Group B <i>n</i> = 59 (%)	<i>p</i> value
Median hospital stay in days (25–75% IQR)	2 (2–3)	2 (2–4)	0.045
Early failures	8 (3)	1 (2)	NS
Perioperative death	4 (1)	0	
Early conversion	5 (2)	1 (1)	
Endoleak at 30 days*	21 (8)	5 (8)	NS
Late failures	21 (8)	8 (14)	NS
Late conversions	3 (1)	1 (2)	
AAA diameter increase	17 (6)	4 (7)	
Persistent type 1 endoleak	1 (0.4)	3 (5)	
AAA rupture	1 (0.4)	0	
Reinterventions	15 (6)	4 (7)	

* In Group A, eighteen endoleaks were type 2 (reperfusion), and three endoleaks were type 1 (proximal). In Group B, four endoleaks were type 2 (reperfusion) and one endoleak was type 3 (between distal cuff and iliac branch).

Table 1. Risk factors of AAA (Group A) and iliac aneurysm (Group B) patients.

	Group A <i>n</i> = 277 (%)	Group B <i>n</i> = 59 (%)	<i>p</i> value
Mean age	70	72	NS
Gender (males)	260 (94)	56 (95)	NS
Smoking	158 (57)	38 (64)	NS
Hypertension	179 (64)	40 (68)	NS
Diabetes	26 (9)	3 (5)	NS
Cardiac disease	121 (44)	36 (61)	0.02
Respiratory disease	142 (51)	39 (66)	0.04
Renal insufficiency	30 (11)	7 (12)	NS
ASA Class = 4	24 (9)	15 (27)	0.001

NS, not significant.

group A and 3 (5.1%) in group B. In all cases late mortality was not related to the endovascular procedure.

Two patients in group A and 5 in group B experienced buttock claudication ($p=0.001$): all occurred in patients with a covered or coiled internal iliac artery.

The mean decrease of the maximum AAA diameter was 4.3 mm in group A and 4.5 mm in group B ($p=NS$). Of 326 AAA successfully implanted, 182 (56%) showed a decrease of maximum aneurysmal diameter >2 mm, 127 (39%) had unchanged diameter, and 21 (6% 17 in Group A and 4 in Group B) showed a diameter increase >2 mm. The mean decrease of CIA aneurysm diameter in group B was 2.1 mm. Of 77 iliac aneurysms treated without immediate death and/or conversion, 46 (60%) showed a decrease of maximum iliac aneurysmal diameter >2 mm, 28 (36%) had unchanged diameter, and 3 (4%) showed a diameter increase >2 mm.

Nineteen reinterventions were performed during follow-up (see Table 3). In only one case the indication for reintervention was the increase of iliac aneurysm diameter. Four extra-anatomic by-passes for branch occlusion, one inferior mesenteric artery clipping, four conversions to open repair and 10 endovascular reinterventions (nine adjunctive proximal cuffs and one adjunctive double proximal and iliac cuffs) were performed.

Discussion

Endovascular AAA treatment in patients with concomitant iliac aneurysms requires adjunctive technical procedures that significantly lengthen surgical and fluoro time, increase intraoperative contrast load, and recovery time. Yet, according to our data, extensive iliac aneurysmal disease does not affect significantly early and mid-term success rates after AAA endoluminal repair.

Successful endovascular repair of AAA requires secure fixation of the endograft to the native arteries at the proximal and distal necks, ensured by mechanical forces of the metal components of the graft. Usually the self-expanding grafts are oversized 10–20% to provide continuous radial force to maintain fixation and haemostatic seal. Iliac artery anatomy plays a crucial role in aortic endografting: a segment of appropriate diameter (usually less than 14 mm) and length (at least 10–15 mm) should be present at this level to ensure adequate sealing of most of the currently available endografts. These requirements are difficult to meet. In a morphometric study by Schumacher *et al.*, 31.8% of 242 patients evaluated for AAA endografting had aneurysmal dilatation down

to the iliac bifurcation, while 14.5% showed partial CIA aneurysmal involvement.⁴

In such cases endoluminal treatment is still feasible only if the endograft is deployed at the distal portion of non-aneurysmal CIA, deployed into the ectatic CIA, or if the graft is extended into the external iliac artery with concomitant exclusion or reimplantation of the internal iliac artery.⁵

Occlusion of one internal iliac artery has proved to be a relatively safe procedure in several studies in the obstetrics and gynaecology, urology and trauma literature. Yet, unilateral and bilateral interruption has been reported to be associated with considerable morbidity including buttock necrosis, severe lower extremity neurologic deficits, ischaemic colitis, impotence, and gluteal claudication.^{6–13}

In our experience 33 internal iliac arteries were excluded in 28 patients: in three cases bilateral interruption of internal iliac artery was with re-implantation or by pass, as described by Parodi.¹⁴ This adjunctive procedure was not undertaken in a patient with colonic cancer who underwent resection the day after aortic endografting, without any finding of colonic ischaemia, and was not accomplished in a second patient who presented heavily calcified CIA and internal iliac artery at surgical exploration. In 25 cases embolisation of the internal iliac artery was not performed either because the graft itself occluded the ostium, or because the preoperative angiogram showed that hypogastric refilling was unimportant. In the remaining five cases internal iliac artery was embolised yet, the coils were deployed in the main trunk of the artery, thus preserving patency of the distal branches. This conservative approach may explain the absence of serious ischaemic complications in our series. Yet, it should be emphasised that incidence of buttock claudication was significantly higher in patients with concomitant iliac disease. Collateral circulation plays an important role in preventing pelvic ischaemia. In a study by Iliopoulos *et al.*, internal iliac artery pressures and variations after intermittent clamping of the pelvic arteries were recorded, and the crucial role of the collateral pathway coming from the ipsilateral external iliac–deep femoral arteries was demonstrated.¹⁵

In 20 patients with ectatic CIA distal to the aneurysm (between 15 and 20 mm in diameter), we used the “bell bottom” technique, placing the endograft at the distal extremity of the iliac aneurysm. Some of the currently available endografts provide custom or standard made iliac limbs with flared configuration, while others allow to obtain the flared bottom end by placing an aortic extender cuff into the iliac limb of the graft. This technique may help in broadening

the spectrum of anatomical settings in which endoluminal repair can be performed. In a study by Hodgson this technique allowed recruitment of an additional 15% of patients for aortic endografting, without sacrificing the internal iliac artery.¹⁶ Long-term durability of this approach is under study. However, natural history of CIA aneurysms appears benign. In a retrospective review by Santilli *et al.*, the expansion rate of CIA aneurysms less than 30 mm maximum diameter at CT and/or duplex examination was 0.11 ± 0.02 cm/year, causing no symptoms or ruptures at a mean follow-up of 31 months.¹⁷ Similar results were reported for patients with concomitant AAA and CIA aneurysms. Lavee *et al.* reported no diameter increase in ectatic iliac arteries 4 years after aortic tube graft open repair.¹⁸ Similarly Provan *et al.* documented no enlargement of CIAs up to 30 mm in diameter 3–5 years after open AAA repair.¹⁹

In our experience, CIA aneurysms enlarged significantly in follow-up only in one case. This patient underwent iliac endografting and internal iliac artery exclusion 18 months after the primary procedure. This patient also had aortic neck enlargement with graft migration that required contemporary proximal aortic extension.

Close postoperative surveillance allows avoiding major complications deriving from diameter changes, endoleaks or graft migration.

In summary, different techniques allow extension of indications for endoluminal repair in patients with short or absence of distal neck at the CIA level. However, considering that this approach has potentially serious complications, it is our opinion it should be restricted to patients with poor surgical risk. True incidence of peculiar complications like pelvic and colon ischemia may have been underestimated in our patients with internal iliac artery interruption because patients in this group had limited sexual and walking activities, and because routine colon endoscopy was not performed.

In conclusion, the presence of an iliac aneurysm does not affect the outcome of endoluminal AAA repair. However, treatment of extensive iliac aneurysmal disease needs adjunctive procedures and carries higher risk of disabling claudication. Endoluminal repair of aorto-iliac aneurysms, avoiding bilateral internal iliac artery interruption may be an advantageous option in patients at high risk for open repair. A longer follow-up is needed to examine outcome of these patients and the fate of the iliac aneurysmal sac in the long-term.

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