Cardiovascular Topics

EVAR: critical applied aortic morphology relevant to type-II endoleaks following device enhancement in patients with abdominal aortic aneurysms

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Summary

Endovascular repair (EVAR) of abdominal aortic aneurysms (AAA) is an established alternative option to conventional surgery for AAA, provided optimal anatomical morphology of the aneurysm sac, neck and outflow exists. In most documented series of EVAR, type-II endoleak occurrence is a universal procedural drawback. This is referred to as the Achilles heel of EVAR. This morphological study, addressing predominantly non-aneurysmal aortic anatomy, reveals the dyssynchronous origins of the renal ostia, ectopia of the superior mesenteric artery and median sacral artery, variations in the length of the infrarenal abdominal aorta, multiple mainstem renal arteries, and the presence of accessory renal arteries (in 13% of cadavers). Such potential vascular anomalies need careful consideration pre-operatively prior to EVAR.

In a prospective, clinical study of EVAR in 163 patients over 60 months, using four different aortic stent devices, we demonstrated an intraprocedural type-II endoleak rate, before exclusion, of 3% (5/163). Most were related to patent lumbar arteries. An active policy of intraprocedural aneurysm pressure sac measurement and angiography was used to demonstrate type-I and type-II endoleaks, focusing on the applied anatomy of aortic side branches and variations. Selective intraprocedural coil embolisation and thrombin injection into the sac was utilised to thrombose persisting and large lumbar arteries that predisposed to retroleaks. We recorded a low incidence of persisting type-II endoleaks using this proactive treatment strategy by addressing variant aortic morphology and patent lumbar arteries during EVAR. One aneurysm-related death (0.6%) was observed due to late rupture after EVAR, and a single intraprocedural death was related to unpredictable aneurysm rupture.

In conclusion, comprehensive anatomical knowledge of the abdominal aorta and its main collateral side branches, including variations, is a fundamental prerequisite if satisfactory and predictable results are to be achieved after EVAR, especially regarding prevention, diagnosis and treatment of type-II endoleaks. Intraprocedural aneurysm sac pressure monitoring, coil embolisation and the use of injection of thrombin into the aneurysm sac of selected patients is useful in reducing the incidence of post-EVAR type-II persisting endoleaks.

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The object of definitive exclusion of abdominal aortic aneurysms (AAA), either by conventional aneurysmectomy or endovascular aortic aneurysm repair (EVAR), is to prevent or reduce rupture and predictable aneurysm-related

death.[1-3] Important causes of death following rupture of an abdominal aortic aneurysm include exsanguinating haemorrhage and multiple organ failure.[1] Sadly, death due to aneurysm rupture can be clinically missed, as shown in our cadaver study.

Until 10 years ago, all patients with large AAA, including selected octogenarians declared fit for surgery, underwent conventional aneurysmectomy and prosthetic engraftment. Evidence-based studies show that predictable rupture can be prevented by this treatment.[1] In about 70% of surgical cases, it is feasible to exclude AAA by insertion of a short interposition Dacron or PTFE tubular graft. The Cleveland Clinic Foundation has confirmed, in a large series, that open repair of AAA in properly selected octogenarians is safe and durable.[4] In almost all cases, this meant a single, definitive, cost-effective intervention. This group, over a nine-year period, reported an 8% 30-day mortality rate for 114 octogenarians who had had open surgical repair.[4] Other high-volume vascular units report low in-hospital mortality rates following conventional aneurysm surgery in octogenarians.[5-8] Most series report freedom of aneurysm rupture after aneurysmectomy in excess of 95%, therefore confirming the durability of conventional aneurysm surgery.[4-7]

In 1976, Parodi of Buenos Aires pioneered the concept of endoluminal aneurysm exclusion.[9] EVAR today is a recognised alternative to conventional surgery in selected patients with AAA meeting inclusion criteria.[10-13] About 75% of AAA referrals to high-volume vascular units, including high-risk patients and octogenarians, can be managed by elective EVAR, provided aneurysm morphology is favourable. Motivation for EVAR include reduced hospital stay, less postoperative pain, reduced transfusion requirements and rapid rehabilitation.[10-13] Compared to conventional aneurysmectomy, the Achilles heel of EVAR is the development and consequences of endotension, endoleaks, especially regarding a predisposition to late rupture, and aneurysm-related death.[14-26] Our group believes this is due to a disregard for the anatomy of the aorta and side branches, including variations. A second cost-ineffective drawback is the need for secondary interventions in about 20% of patients post-EVAR.[20,22,23] Single or multiple negative factors such as endoleaks, endotension, stent-migration and stent-fracture have bedevilled the application of early-generation stents. Development of new-generation endografts have steadily overcome these initial drawbacks.[10]

The purpose of this morphological study was to emphasise the importance of clinically relevant surgical anatomy of the abdominal aorta, aberrant vessels, side branches, variations, collateral circulation and juxta-renal region morphology specific to EVAR and type-II endoleaks. Secondly, we report on the incidence and outcome of type-II endoleaks in a series of 163 patients undergoing prospective, elective EVAR and entered into the VASSA Registry during 2003.

Materials and methods

Critical aortic morphology relevant to EVAR

Cadaveric dissection of the abdominal aorta and collateral side branches, inferior to the superior mesenteric artery, was performed in a small cohort (31 human aortas) on predominantly non-aneurysmal aortas. One patient had an undiagnosed aortic aneurysm rupture that resulted in death. Standard and surgical anatomy literature was consulted before and during dissection.[27-31] Special emphasis was placed on dissection of aberrant vessels, aortic anomalies, variations, renal arteries, ostia and take-off and all anatomical features that impact on EVAR. Patency and numbers of all side branches, including variations, were carefully noted, analysed and recorded. Circumferential measurements of the aorta were made using a vernier calliper at varying levels. Anomalies of the abdominal aorta impacted on inclusion and exclusion criteria with regard to EVAR. Special care was taken to study dorsal aortic surface distribution of atherosclerosis, plaques and patency of lumbar arteries and ectopic median sacral ostia.

Regarding the juxtarenal ostial area, circumferential measurements were carefully obtained using a graded vernier calliper. In the clinical situation, this data is needed to facilitate secure proximal stent fixation and the prevention of type-I endoleaks. Readings were obtained from the superior and inferior edges of the renal arteries and compared, in order to assess whether the arteries originated from the abdominal aorta at the same anatomical and horizontal level. This data determines proximal siting of a stent and is useful for predicting the need for transrenal ostial fixation, especially in patients with a short aneurysm neck. Numbers and ostial patency (tested by direct probing and intubation) of the lumbar and median sacral arteries were individually determined. This is a frequent site of type-II endoleaks and perigraft collateral flow after EVAR.[12] Ostial size, patency and diameter of the inferior mesenteric arteries (IMA) were recorded.

Infrarenal aortic measurements were performed and compared, as this data is necessary in the clinical context to determine sizing of the aortic prosthesis (body), thereby ensuring an accurate fixation at the aortic bifurcation. The presence or absence of large accessory renal arteries relevant to EVAR was recorded. This finding is often used as an exclusion criterion for EVAR, especially if the vessels are large and multiple. Posterior abdominal aortic plaque localisation was documented and graded as isolated or confluent (mild, moderate, severe). Ostial splinting of the distal aortic lumbar arteries by atheroma favours endoleak and collateral perigraft flow postprocedurally.[12] For statistical analysis, parametric and non-parametric tests were utilised and data were stored in an Excel database. Data were considered statistically different if $p < 0.05$.

EVAR clinical study

In the clinical component of the study, 207 referred adult patients were prospectively assessed with AAA by a multidisciplinary interventionalist approach, according to the recommendations of the EVAR Consensus Meeting of 2003. Twenty-one per cent (44 of 207) were excluded from EVAR because of co-morbid disease and unfavourable aneurysm anatomy, angulation of the aneurysm neck, short neck, inadequate landing zones and access problems due to narrowed, calcified and angulated iliac arteries.[32]

One hundred and sixty-three patients, with a mean age

of 70.7 years, underwent EVAR over a six-year period. Median AAA diameter was 5.7 cm. American Society of Anaesthesiology (ASA) ratings were as follows: ASA II, 15.9%; III, 57%; IV, 22.6%; V, 2.4%. Patients underwent EVAR using the inclusion criteria recommended by the consensus 2003 Stellenbosch meeting of VASSA. All patients were offered open surgery as an alternative and entered into the VASSA EVAR Trial Registry. The procedure of EVAR, and the inclusion and exclusion criteria have previously been reported by Du Toit and Saaiman.[12] Pre-operatively, patients were assessed by spiral CT and selectively evaluated with conventional angiography.[12] All patients signed informed consent and underwent EVAR by a multidisciplinary interventional team, including vascular surgeons and a cardiologist[12] During the five-year study period, different aortic stent devices were used. All patients underwent pre- and post-EVAR evaluation with duplex Doppler ultrasound assessment of AAA and were followed up at regular intervals indefinitely. Endoleaks were defined as follows:[12]

- type I: graft related (perigraft inflow with and without outflow)
- type II: retroleaks from either the lumbars or inferior mesenteric artery (inflow, with and without outflow)
- type III: prosthetic overlap leak
- type IV: porosity leak (? suture holes)
- type V: unknown origin.

Results: morphological component

Abdominal aorta, variations, relevant to EVAR

The mean age of 31 adult cadavers was 49 years (range 27–90 years). Circumference of the aorta at the level of the superior edge of the renal artery orifice in 30 non-aneurysmal cadavers measured a mean of 4.2 cm (range 2.8–5.6 cm). Circumference at the level of the inferior edge of the renal ostia in the same cohort measured 4.0 cm (range 2.9–5.6 cm) (superior versus inferior circumferences were not significant: $p > 0.05$). In 27 of 30 cadavers (90%), the renal arteries took origin from the aorta at different levels. In three cadavers, the renal arteries were duplicated (10%). In two of the three cadavers, triple renal arteries were present on the left aortic side. In three of these cadavers, an anomalous inferior origin of the SMA was detectable. SMA ostia in these three patients were sited between the renal arteries (Fig. 1). The mean difference in level of origin (measured at the inferior ostial edge) of the left and right renal arteries was 6.9 mm (range 1–17 mm). Sixty per cent (6 of 10) of ostial orifice levels differed by 1 cm or more. The mean distance between the aortic bifurcation and the inferior edge of the left renal artery was 8.8 cm (range 6.6–11.4 cm). On the right side, the mean distance between the aortic bifurcation and the right renal artery was 9.6 cm (range 6.2–12.9 cm). Statistically, this difference was not significant.

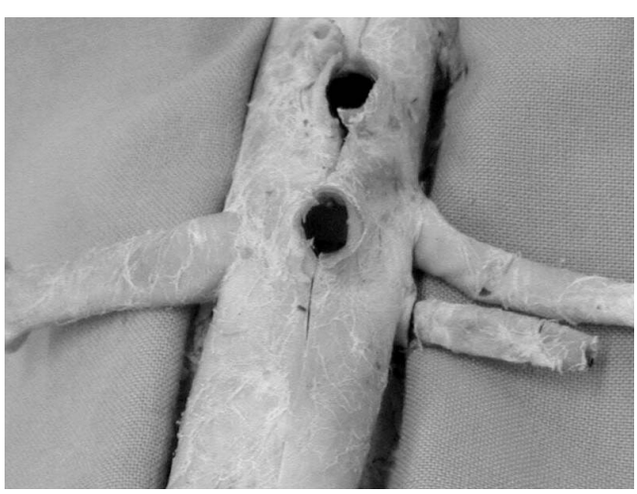

Fig. 1. Anatomical specimen showing an inferior ectopic origin of the SMA sited between the renal arteries (duplicated on the left side). This anomaly would preclude transrenal fixation of a prosthesis during EVAR in a patient with AAA and a hostile neck.

Infrarenal or accessory polar renal arteries occurred in four of 31 cadavers (13%). In one cadaver, a large left-sided accessory artery originated from the abdominal aorta adjacent to the IMA. In three of four specimens, accessory renal arteries were observed on the left side. In all specimens, the inferior mesenteric artery (IMA) was patent. Mean ostial diameter was 3.9 mm (range 1.6–5.9 mm). Regarding the ostial diameters, the following measurements were recorded: 4.4 mm (25%), 4.9 mm (14.3%), 5.9 mm (10.7%) and 1.6 mm (3.6%). The mean distance between the aortic bifurcation and the ostia of the IMA was 3.9 cm (range 2.0–6.1 cm). One hundred and sixty-one lumbar arteries were detected in the cohort studied. In two of 161 (1.2%), the lumbar ostia were not patent. The mean number of lumbars per patient was 5.7 (range 3–9). Ten patients had eight or more lumbar arteries. In three patients, only single lumbars were present: the rest had paired lumbars. In 71% of the cohort studied, the median sacral artery (MSA) was identifiable, and in 60.7%, the ostium was patent. In 46% of cadavers, the MSA ostia were situated in the midline just superior to the aortic bifurcation. In 28% of cadavers, the MSA ostia were situated just to the left of the aortic bifurcation and adjacent to the ostia of the left common iliac artery.

Posterior abdominal aortic plaque occurred in nine of 31 cadavers (29%) at a mean age of 57.7 years (range 40–74 years). Large atherosclerotic and calcified plaques were present at the aortic bifurcation in 6.7% of cases. Plaque formation at the bifurcation and zone inferior to the IMA occurred in 10% of cadavers, some showing identifiable atherothrombosis.

Gonadal (testicular, ovarian) and ureteric arteries were constantly present as paired arteries, but differed in diameter. In one cadaver, the right inferior adrenal artery took origin from the infrarenal aorta, just inferior to the right renal artery.

In one cadaver, a right-sided lumbar ectopic kidney was present (Fig. 2). The right renal artery of this kidney took origin 1.5 cm superior to the aortic bifurcation. An inferior polar artery was visible and took origin from the contralateral, left common iliac artery. Horseshoe kidneys were not detected in this study. Patent MSA ostia are reflected in Fig. 3.

In one cadaver, a large 6-cm ruptured aortic aneurysm was present, which had led to exsanguination and death of

Fig. 2. Anatomical specimen showing the abdominal cavity from anterior, and a right ectopic kidney. The origin of the right renal artery would exclude EVAR in a patient presenting with an AAA and this vascular anomaly. Ostial occlusion will result in extensive segmental renal infarction.

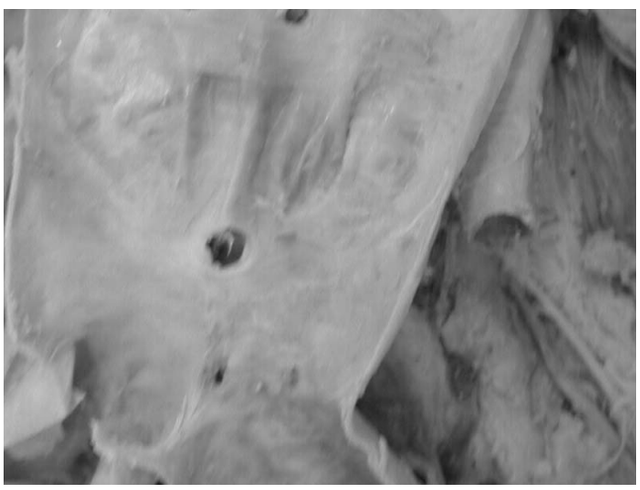

Fig. 3. Anatomical specimen of the inferior aspect of the abdominal aorta. Note paired lumbar ostia surrounded by hard circumferential atherosclerotic plaque, a large median sacral artery (MSA), and small ectopic MSA sited in the orifice of the left common iliac artery. This is frequently the site and origin of lumbar endoleaks after EVAR.

the patient. Fig. 4 shows an infrarenal, ruptured abdominal aortic aneurysm viewed from the ventral surface. A 20-mm, non-angulated aneurysm neck was present. Supra- and infrarenal aortic measurements were 19 and 16 mm, respectively. The origins of the renal arteries were eccentric and no polar vessels were present. Aneurysm diameter was 60 mm, with posterior erosion of three vertebral bodies and 20-mm postero-lateral rupture. Bifurcation diameter was 27 cm. A concomitant 3-by-3-cm right-sided common iliac artery aneurysm was present. Lumens of the external iliac artery ostia measured 11 mm on both sides. Both vessels were patent. Four patent lumbar arteries were detected. One had a large ostium and communicated with iliolumbar arterial collaterals. The length of the aneurysm sac measured 100 mm. Free intraperitoneal rupture was present with a

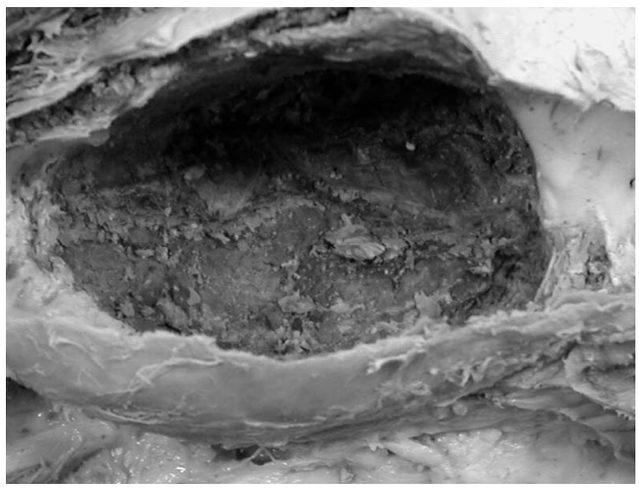

Fig. 4. Anatomical specimen showing a ruptured 6-cm abdominal aortic aneurysm. A concomitant right common iliac artery aneurysm is present. Aneurysm neck morphology and iliac artery measurements favour either conventional surgery or EVAR.

large clot in the left paracolic gutter and pelvic cavity. Aneurysm sac morphology would have permitted either open surgical repair or management by EVAR. Aneurysm neck and landing area morphology favoured endoluminal stenting.

EVAR clinical study

In a 60-month prospective clinical study, successful stent-deployment rate was 99% and rendered a primary patency stent rate of 97%. At 30 days, the results were as follows: surgical conversion 0.6%; procedural or intra-operative mortality 1.2%; 30-day operative mortality 4.3%; and need for secondary interventions 24.5%. This included staged and planned interventions over 30 months after EVAR, such as cuffing or insertion of additional stents to address type-II endoleaks. Follow-up period was a median of 28.3 months (range 1–69 months).

Co-morbidities that contributed to late mortality and attrition included ischaemic heart disease, cardiomyopathy, renal failure, stroke and cancer. Details of late mortality post-EVAR are provided in Table I. One procedural rupture was fatal (0.6%). Two late ruptures occurred, one was successfully endostented and the other patient died after a failed surgical intervention (0.6%). The 30-day outcome and endoleak type, according to endograft used, are reflected in Tables II, III and IV. Intraprocedurally, aneurysm sac pressure measurements together with angiography were utilised to detect type-I or -II endoleaks and obvious patent lumbar arteries. Selective intraprocedural cuffing, coil embolisation and thrombin injection were used to seal endoleaks originating from lumbar arteries. In two patients, coils were used and in another 11, thrombin was injected into angiographically detected endoleaks to afford sealage. Two patients with suspected but unproven type-I persisting endoleaks are being followed up. Surgical or endovascular interventions are under consideration. In three patients, endotension was diagnosed, but an endoleak from a lumbar artery could not be detected (1.8%).

TABLE I. LATE MORTALITY:* CO-MORBIDITIES AND MORTALITY PROFILE AFTER EVAR (*n* = 35)

Co-morbidity status before stenting	n	*Cause of death after EVAR*	n
Cardiac (IHD)	24 (68%)	Stroke[1]	3
Cardiomyopathy	5	Cancer (mean 14.3 months)	9
Respiratory	9	Multi-organ failure[2]	3
Diabetes mellitus	4	Myocardial infarction[3]	7 (20%)
Stroke (ischaemic)	3	Pneumonia	2
EF < 43%	9	Cardiac failure	2
Arrythmia	4	Septicaemia (unknown)	2
Cancer	2	Renal failure[4]	2
Hypertension	16 (45%)	Cardiomyopathy	3
PVD	10	Ruptured AAA	1 (0.6%)
Renal insufficiency	2	Bee sting anaphylaxis	1
Liver cirrhosis	1	Endograft sepsis[5]	1 (0.6%)
Obesity	1	Unknown cause	1
Hostile abdomen	1	Cardiac-related deaths	13 (35%)
AAA rupture	1		
Comorbidity combinations	28 (80%)		
Age > 90 years	1		

*Majority of late deaths post-EVAR occurred between 18 and 60 months.
[1]Non-procedure related (occurred between 18 months and 60 months post-EVAR).
[2]Two cases were associated with aneurysm rupture. Another occurred after 6 months in a patient with cholangiocarcinoma, jaundice and cholangitis.
[3]One acute coronary syndrome occurred after stenting and was successfully salvaged by coronary intervention. Six occurred after 6 months in patients with three-vessel disease and considered high-risk patients.
[4]Non were procedure related. These occurred in two patients with multi-organ failure.
[5]Clinically suspected, but not bacteriologically proven.

TABLE II. EVAR OF AAA 30-DAY OUTCOME (*n* =163)

Variable	*Total*	*Percentage*
Successful deployment	162	99
Surgical conversion	1	0.6
Procedural mortality	2	1.2
30-day mortality	7	4.29
Secondary interventions	40	24.53

TABLE III. TOTAL ENDOLEAK RATE AND LOCATION AFTER EVAR – PROCEDURAL AND BEFORE EXCLUSION

	AneuRx®	*Talent®*	*Powerlink®*	*Vanguard®*
Total pts stented	47	49	62	3
No endoleak	30	35	47	1
Type I	15	13	11	2
Type II	1	1	3	0
Type III	0	0	0	0
Type IV	0	0	0	0
Indeterminate	1	0	1	0
Total endoleaks	17	1	15	2

TABLE IV. ENDOLEAK RATES AND LOCATIONS AFTER EVAR (AFTER EXCLUSION = PERSISTING)

	AneuRx®	*Talent®*	*Powerlink®*	*Vanguard®*
Total pts stented	47	49	62	3
No endoleak	46	49	61	3
Type I				
Type II	1*	0	1**	0
Type III	0	0	0	0
Type IV	0	0	0	0
Indeterminate	0	0	0	0
Total endoleaks	1	0	1	0

*Endoleak excluded at three months, persists at 16 months.
** Patient not yet seen at follow-up.

Discussion

Comprehensive knowledge of aberrant morphological anatomy of the abdominal aorta, collateral aortic side branches, variations and growth evolution of AAA is of critical importance to the interventionalist striving to reduce the incidence of endoleaks and perigraft flow.[1-3,27-31,33] Anomalies of the abdominal aorta impact on stent selection, sizing, placement, fixation and sealage. Anomalies affecting aortic side branches and take-off may well be predictors or risk factors for endoleak formation. Aortic and side branch variations, as described, influence inclusion and exclusion criteria for EVAR. An endoleak is defined by the persistence of blood flow outside the lumen of an endoluminal graft, but within the aneurysm sac.[32]

Endoleaks are classified as type I, II, III and IV.[32] It is accepted that an endoleak is evidence of incomplete exclusion of an aneurysm from the circulation during or after EVAR.[32] Various causes have been cited.[14-17,19-26] Important causes include incomplete seal between the endograft and aortic wall, retrograde blood flow from patent aortic side branches, fabric defects or porosity, poor sealing between the components of a modular prosthesis, and device failure.[19,24]

Pre-operatively, prediction of endoleak incidence is not accurate, despite using spiral CT.[10,12] Type-I endoleaks (ineffective seal at either the proximal or distal graft ends or attachment zones), if not controlled, correlate strongly with delayed aneurysm rupture after EVAR.[18,32] A type-II endoleak is associated with retrograde or perigraft flow from lumbar arteries, the inferior mesenteric artery (IMA), or other collateral vessels.[15,16,19,20,22-25]

Pre-operative assessment of aortic side branches and variations is not consistently accurate. This applies especially to aberrant lumbar artery anatomy in the distal aorta. Origin and outflow patterns have previously been reported by Du Toit and Saaiman and include lumbar–lumbar, lumbar–IMA, accessory renal–lumbar/IMA, hypogastric–lumbar/IMA or undefined.[32] Knowledge of applied anatomy of aortic side branches and anomalies are important to categorise aetiology, diagnosis and treatment strategies of endoleaks.[32] This allows the interventionalist to prognosticate regarding potential obliteration of the retroleak with significant perigraft flow.

Connection of flow to proximal or distal graft end, even in the presence of retrograde flow from a lumbar or IMA vessel, classifies an endoleak as type I.[32,34] The risk of late aneurysm rupture in these patients is prohibitive. Endotension refers to the problem of continued enlargement of an AAA after EVAR, often in the absence of a detectable endoleak.[15,21] Aneurysm sac enlargement under these circumstances may lead to aneurysm rupture.[15,18,21]

Endoleaks and endotension therefore remain an active area of investigation because of the danger of unexpected late rupture and aneurysm-related death after EVAR.[15,19-26]

Endoleaks almost never manifest after conventional open aneurysm repair because lumbar orifices are intentionally ligated to prevent back-bleeding into the sac.[4,6-9] The IMA is ligated or reimplanted into the prosthetic endograft. Endoleak formation following endoluminal treatment of AAA is regarded as the Achilles heal of EVAR, if improperly managed, because late rupture of the stented aneurysm sac may occur.[15,19,23,25]

We believe that it is possible to reduce the implications and negative sequelae of late or persisting endoleaks by adopting a proactive policy of intraprocedural aneurysm sac pressure monitoring, selective angiography and exclusion of perigraft flow by planned and intentional coil embolisation or thrombin injection into the sac. Detailed knowledge of lumbar artery anatomy and variations, including collateral circulation with the hypogastric arteries, is critical in post-procedural decision-making. Precise anatomical localisation of retroleaks by angiography is imperative if coil embolisation is to be considered. Careful study thereof is required, as side-branch anatomy is distorted in patients with large aortic aneurysms and not always accurately visualised by conventional intraprocedural angiography.

An important vascular consensus meeting with a focus on the importance of post-EVAR endoleaks was held in 2002 under the auspices of the Society of Vascular Surgery and the American Association for Vascular Surgery.[15] One of the concerns that was pointed out is that the mid- and long-term durability of endografts used during EVAR and the effectiveness in prevention of rupture remains questionable.[15]

One of the principal reasons for device failure one year after EVAR is the occurrence of significant and persisting endoleaks. In some patients, endoleaks and endotension are critically important problems after EVAR. Consensus at the international conference was that the current classification system of endoleaks, with some important modifications, is adequate.[15] The recognised incidence of type-I and -II endoleaks after EVAR is 0–10% and 10–25% respectively.[15] Many type-II endoleaks will seal spontaneously and are not regarded as being associated with detrimental effects.[15] An enlarging aneurysm after EVAR mandates early surgical or interventional treatment.[15] Consensus and general agreement was reached regarding treatment strategy for the latter entity.

Pre-treatment of patent lumbar and inferior mesenteric arteries in some way before EVAR was considered controversial.[15] Precise localisation of all lumbars is needed if this approach is adopted. The prevailing opinion at the consensus meeting was that 2–15% of type-II endoleaks were associated with an unfavourable outcome.[15] Some interventionalists have commented that the 'bad outcome' may be due to an unrecognised, simultaneous type-I leak.[15] Of interest was the finding that coil embolisation of type-I or type-II endoleaks may be ineffective.[15] Laporoscopic clipping of aortic collateral branches has remained a popular approach by some workers to type-II endoleaks.[15] Knowledge of applied aortic anatomy is desired to achieve positive outcomes, thereby reducing the chances of late aneurysm rupture.

From the published literature, it is clear that much remains to be learned about the causation of endoleaks and endotension, and how they may be optimally prevented and treated.[15] For instance, it is now established that some endoleaks cannot be detected with optimal CT scanning.[32] Many units have shown that duplex ultrasonography, although useful, is not the best method for the detection of the anatomical origin of endoleaks.[32] Ultrasonography, although non-invasive, is not always accurate for the detection and precise localisation of type-II endoleaks.[32]

In the current clinical study, the overall type-I procedural endoleak rate before exclusion was 25.1%. After intraprocedural endoleak exclusion, the incidence was reduced to 1.2%. Other units report an endoleak incidence rate varying from 15 to 25%.[15,19-26] Regarding type-II endoleaks before exclusion, five were detected (3%) either by ultrasonography or spiral CT. All were successfully excluded either by coil embolisation or thrombin infusion into the sac. Intraprocedural aneurysm sac pressure measurement, together with angiography, facilitated planned intraprocedural exclusion thereof. Using this strategy, only two persisting (1.2%) endoleaks were detectable during follow-up duplex Doppler examinations.

The problem of post-EVAR endotension is well documented in the literature and the treatment thereof remains controversial.[15] In the present study, three patients (1.8%) have endotension and are undergoing careful follow-up surveillance. In the current study, no perigraft flow could be attributed to a back-bleed from the IMA. However, two patients (1.2%) developed colonic ischaemia within 30 days of EVAR. Renal atheroembolisation or renal infarction was not detectable procedurally or beyond 30 days. Other workers have reported on the incidence of renal infarction after EVAR.[35]

Results of the morphological studies in dissected laboratory specimens without AAA revealed important applied anatomical anomalies and aberrations relevant to a successful EVAR outcome and potential development of endoleaks.[22] These anomalies are poorly delineated in standard and classic works of anatomy. The origin of the renal arteries, and therefore the ostia, were dyssynchronous in 90% of the cases we examined. In almost all cadavers studied, the left renal ostium was inferior to the origin of the right renal artery. With regard to EVAR, this relation is critical in accurate placement of the proximal edge of the stent, especially in patients with a hostile and angulated neck. This information is also useful if supra- or transrenal fixation is contemplated.[35] Care must be exercised not to occlude inferiorly sited main renal ostia with the stent, or when using extra cuffs to seal intraprocedural type-I endoleaks. In 10% of the cadavers, the SMA took origin between the renal arteries. This anomalous anatomical finding rules out the safe application of trans- or suprarenal fixation of a stent, as SMA occlusion could occur, resulting in total small intestinal ischaemia.[35]

Accessory polar renal arteries were present in 3.3% of cases and this anatomical finding is a predictor of segmental renal infarction if EVAR is considered in such patients.

Also stent closure of the ostia of these vessels may result in segmental renal ischaemia and renal failure.[34] In 6% of cadavers, an inferior ectopic origin of a large right renal artery and accessory renal artery was present. This vessel anomaly altogether excludes safe application of conventional EVAR and will result in infarction of a whole kidney or a large segmental infarct. Many units consider such vascular anomalies as exclusion criteria for EVAR.

Regarding the size of the IMA ostia, 10.7% were greater than 5.9 mm in diameter. This finding may well explain the reason for endoleak formation reported in the literature and colonic ischaemia after EVAR. During conventional surgery, these large vessels are traditionally and selectively re-anastomosed to the prosthetic endograft for the purpose of reducing colonic ischaemia. The mean number of lumbar arteries per cadaver was 5.7 (range 3–9). These numerical data are not always appreciated during insertion of an aortic endoluminal stent. Often, on-table completion angiography does not adequately highlight all these vessels. In 33% of cadavers, eight or more lumbars were present, obviously increasing the chances of type-II endoleaks in the clinical situation, especially if these ostia were patent and large. More than 90% had patent ostia. Twenty per cent of cadavers had large patent lumbar ostia. This finding, theoretically, would favour type-II endoleak formation.[33] Our multidisciplinary group has identified this observation as an important finding, especially with regard to large distal aortic lumbar arteries and retroleaks.

Patency of the median sacral artery (MSA) was demonstrated in 70% of the cadaveric cohort study. Of interest was the finding, in a small cohort, of the orifice of MSA at the origin of the left common iliac artery. This finding is technically relevant as uncontrolled patency of the MSA is also known to be associated with perigraft flow and endoleak. Our study could not confirm or exclude this possibility. Anatomically, smaller aortic side branches are occluded during EVAR: gonadal arteries (95%), inferior ureteric arteries (95%) and inferior adrenal artery (3%). The clinical significance may be unpredictable or unimportant.

Anatomically, when considering EVAR for patients with AAA, ectopic kidneys (3%), multiple main renal arteries (10%), accessory renal arteries (13%) and horseshoe kidneys pose substantial challenges for the inexperienced interventionalist working in low-volume units. This also applies to vascular surgeons considering conventional surgery for AAA. Mean measurement between the aortic bifurcation and left renal artery in 30 cadavers was 8.8 cm (range 6.6–11.4 cm). These differences and discrepancies in infrarenal aortic length may impact negatively on the selection of the optimal length and type of stent body for EVAR. This applies especially to the use of first-generation aortic stent devices.

Theoretically, an anatomical misfit of the body of the stent adjacent to the bifurcation may predispose to poor sealing of inferior lumbars and MSA. Stent–host wall mismatch due to distorted anatomical morphology and varying distances in aortic length may contribute to the development of type-II endoleaks and eventual distal migration of the stent body. The latter problem may be the explanation for the development of late type-I endoleaks and eventual post-EVAR aneurysm rupture. This is a topical point, as the effect of remodelling of the aneurysm on aortic endografts after EVAR is not fully understood and appreciated. We believe this to be an important issue, as 30% of our cadavers (mean age 57.7 years) had severe, posterior abdominal atherosclerotic aortic plaque, theoretically, influencing long-term aneurysm remodelling and contribution to late endoleak formation after EVAR.

In conclusion, type-II endoleaks originating from patent infrarenal aortic side branches are an important drawback of EVAR. Diagnostic and management strategies remain controversial. Pre-operative imaging of aortic side branches cannot always predict the incidence of post-EVAR endoleaks. Comprehensive anatomical knowledge of the aorta and its side branches is a prerequisite, if satisfactory endoleak-free results are to be attained after EVAR, thereby reducing the need for secondary interventions. The incidence of post-EVAR type-II endoleaks can be reduced by intra-operative utilisation of aneurysm sac pressure monitoring, considering important anatomical variations of patent lumbar arteries, coil embolisation and selective injection of thrombin into the sac.

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