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Aortocaval Fistula: Is Endovascular Repair the Preferred Solution?

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Abstract

Background—To compare outcomes of open and endovascular repair of aortocaval fistulas (ACFs) in the setting of abdominal aortic aneurysms (AAAs).

Methods—A literature review was undertaken on Pubmed from 1999 to 2014 to identify reported cases of both endovascular and open repair of ACF, including the index case, presented here. Primary outcomes for endovascular repair were: complications, presence of endoleak, and death. Primary outcomes for open repair were: complications and death.

Results—Forty articles were reviewed with a total of 67 patients, including the index case. Endovascular approach was used in 26 patients (39%). Endoleaks were present in 50%, whereas similarly 46% of patients had a reported complication. Five deaths (19%) occurred in the endovascular group. Open repair was performed in 41 cases (61%). The rate of complication and the death in open repair were 36% and 12%, respectively ($P = 0.327$ and $P = 0.910$, respectively) compared with endovascular. Mean follow-up was 7.7 months for the endovascular group and 8.5 months in the open group.

Conclusions—Previous demonstrations of high morbidity and mortality with open repair of ACF in the setting of AAA have motivated endovascular approaches. However, endoleaks are a significant problem and were present in 50% of ACF cases. The continued presence of an endoleak in the setting of an ACF may result in persistence of the ACF, unlikely thrombosis of the endoleak, and continued sac enlargement. Endovascular repair presents theoretical benefit, yet is not associated with a reduced rate of complication or death versus open repair in this contemporary review.

Introduction

Aortocaval fistulas (ACFs) are a rare entity. Although the most common cause is penetrating trauma, in the setting of abdominal aortic aneurysms (AAAs), the incidence is <1%.¹ Among ruptured AAA, ACF is noted in 2–7% of cases.² ACF was first described by Symes in 1831, and over a century later, Dr. Cooley reported the first successful repair in 1955.³ Dr. Woolley in 1995 reported the first case of aortic exclusion used in the treatment of an

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intraoperatively diagnosed ACF. At the dawn of the endovascular age, most ACF had been repaired primarily in an open fashion and the literature accounted the surgical mortality to be as high as 16–66%.^{4,5} However, after Beveridge et al.⁶ in 1998 reported the first endovascular repair of ACF, many have published reports of ACF describe endovascular management. We sought to review the contemporary outcomes and complications of both methods, present a successful case of open repair, and determine if endovascular repair was associated with improved outcomes.

Case Report

A 55-year-old African-American man with a 42-pack-year smoking history, coronary artery disease, and ongoing dyspnea was referred for an AAA. The patient underwent CT angiography which identified a 6.8-cm infrarenal AAA as well as bilateral iliac aneurysms, measuring 5.0 and 3.5 cm. More important, it also revealed a large ACF (Fig. 1). Previous echocardiograms showed a declining left ventricular ejection fraction (current left ventricular ejection fraction = 35%) and a dilated right ventricle with a pulmonary artery pressure of 70 mm Hg. A right heart catheterization was performed which demonstrated several residual 50–70% coronary stenosis but no lesions which would benefit from further percutaneous revascularization. The coronary arteriography also confirmed a massively elevated right heart filling pressure of 25 mm Hg. His overall deteriorating clinical condition was thought to be secondary to his progressive high-output heart failure and was taken semielectively for an open repair as the obligate delay for a fenestrated device to address his juxtarenal AAA would place him at undue risk for further functional decline. A pulmonary catheter was placed and transesophageal echocardiography performed (Fig. 2). Repair was approached via a generous midline incision during which time massive edema was noted throughout retroperitoneum. There was a thrill in the vena cava, and as the cava was compressed in that area, normalization of the right ventricular hemodynamics occurred. The aortoiliacs were controlled, and the aneurysm sac opened revealing a large ACF. Venous bleeding was controlled manually with compression within the aneurysm sac, and the ACF repaired primarily with running Prolene sutures from within the aortic wall (Video 1). Estimated blood loss was 2000 mL, and the patient received 750 mL of Cell Saver transfusion and 2 units of banked blood. After ACF closure, the patient's pulmonary artery pressures immediately decreased from 61 of 42 to 23 of 15. His heart rate was maintained; however, the patient was placed on a nitroglycerin drip for hypertension. The aortic and iliac aneurysms were repaired with a bifurcated Dacron graft, each distal limb to the iliac bifurcations. His postoperative course was uncomplicated. His cardiac output decreased from 12.0 L/min preoperatively to 5.6 L/min postoperatively. He was discharged home on postoperative day 7. At his 2-month check, he lost 70 pounds, and he was beginning to return previous activities such as swimming and weightlifting. At 1 year of follow-up, his echocardiogram demonstrates favorable remodeling and improved ejection fraction.

Methods

A contemporary literature review was undertaken on Pubmed from 1999 to 2014 using search terms ACF, aortoiliac fistula, and aortovenous fistula to identify reported cases of both endovascular and open repair of ACF. We elected to limit the search over the last 15

years because we postulated that after 1999, surgeons had the option to choose between open and endovascular repair. Only cases involving nontraumatic aortocaval, aortoiliac, or ilioaortic fistulas were included. The case presentation and demographics were summarized and follow-up recorded. In addition, only articles identifying presence or absence of endoleak were included for the endovascular group. Overall, the patients with ACF were divided in 2 categories for comparison according to the treatment received: the endovascular repair group ($n = 26$) and the open repair group ($n = 41$). Primary outcomes for the endovascular repair were: complications, presence of group, and the death. Most authors included endoleaks as a complication if further intervention was required. Primary outcomes for open repair were: complications and death. Rupture was defined as violation of the retroperitoneum and excluded simply the presence of an ACF. Statistical analysis was performed using single-tailed Fisher's exact test for categorical variables given sample size and expected contingency tables.

Results

Demographics

Forty articles were reviewed totaling 67 patients, including our patient presented above. The average age was 68 and 70 years in open and endovascular groups, respectively (Table I). Most patients were men (96%). Most patients presented with acute onset of abdominal or back pain, seen in 75.6% open and 46.1% endovascular repairs. Of note, 24% of patients did present with shortness of breath 19.5% in open group and 30.7% in the endovascular group ($P = 0.223$).

Anatomy

Most patients had infrarenal ACFs; 87.8% open and 84.6% endovascular (Table II). One patient in the open group had a juxtarenal ACF. Iliocaval and aortoiliac fistulas were less common. Five patients, 2 in the endovascular group and 3 in the open group, were noted to have either an inflammatory aneurysm or retroperitoneal fibrosis. There were 25 (60.9%) ruptured aneurysms in the open group and 6 (23.1%) in the endovascular group.

Technique

Table III reviews the intraoperative approach to all 67 patients. Most patients undergoing an open repair had primary repair of the ACF from within the sac. Espinel et al.¹¹ and Madani et al.⁴⁶ used a retrograde balloon occlusion of the inferior vena cava (IVC) for venous control before primarily repairing the ACF. Four patients were repaired with a patch angioplasty of the IVC.^{8,9,12} Endovascular repair included several endograft types: Boston Scientific (Natick, MA), Gore (Flagstaff, CA), Cook (Bloomington, IN), Edwards Lifescience (Irvine, CA), Medtronic (Minneapolis, MN), and La Maitre (Burlington, MA).

Outcomes

There were 15 of 41 patients (36%) in the open group who had a postoperative complication (Table IV). These included renal failure, respiratory failure, deep venous thrombosis, bowel obstruction, acalculous cholecystitis, pseudomembranous colitis, wound infection, paralysis, and lower-extremity ischemia. There were 5 mortalities (12% mortality rate among reported

open cases). Thirteen patients (50%, $P = 0.910$, vs. open repair) of those undergoing an endovascular approach had a reported complication (Table V). When only considering endoleaks, 13 patients had a reported endoleak, either at initial surgery or in a delayed fashion. Of the 13 endoleaks, 5 (38%) were intervened on and 6 (46%) resolved spontaneously. Five deaths (19%) occurred in the endovascular group of which none had a ruptured aneurysm. One case died 3 months postoperative and may have been unrelated to the surgery.²⁷ The remaining deaths included multiorgan failure after pneumonia, worsening acidosis, and massive pulmonary embolism. Mean follow-up was 7.7 months for the endovascular group and 8.8 months in the open group (Tables IV and V).

Discussion

Although ACFs can be traumatic or iatrogenic in nature, the majority are primary because of the spontaneous erosion of an AAA into the neighboring venous structures. Previous to the dispersion of endovascular aneurysm repair (EVAR), these patients were primarily repaired in an open fashion. After 1998, surgeons have increasingly elected for an endovascular method, proposing a decreased risk of complications and death. However, endovascular repairs are not without their own significant morbidity as demonstrated in this contemporary analysis.

Endoleaks are a major concern for these patients. Vetrhus et al.²⁷ proposed that endovascular repair is justified with only a small risk of endoleak. We found an endoleak rate of 50% which is much higher than that reported by Antonio et al.⁴⁷ in 2009. Of the reported endoleaks, less than half resolved spontaneously and certainly this high proportion is connected to the concurrent ACF promoting patency of the aortovenous circuit. Although many would argue that type II endoleaks can be monitored closely, when the patient has an ACF, perhaps these should be addressed during their initial hospital stay, particularly in cases in virulent presentation. LaBarbera et al.³⁷ obtained CT angiography on postoperative day 9 showing persistent ACF and type II endoleak and subsequently decided to pursue coil embolization of the inferior mesenteric artery, sac and placement of Amplatzer VSD occluder across the ACF on postoperative day 13. It is more concerning that some endoleaks are occasionally not appreciated on completion angiogram. Melas et al.³⁶ presented such a case, and the patient represented at 6 months with stable sac but a larger sized ACF and increasing signs of heart failure. This begs the argument that in cases where the potential for endoleak is high and the patient's cardiac performance is at risk, the completion angiogram should not be taken as a reliable guide and adjunctive imaging such as early CT or duplex should be routine.²⁵ In the face of an increased incidence of endoleak, some surgeons have modified their endovascular approach. Godart et al.²⁸ describe placing an Amplatzer duct occluder transfemorally for a persistent ACF after endovascular repair. Perhaps the penultimate repair is the placement of an intravenous stent graft as described by Melas et al.³⁶ whereby they deployed an UniFit (Le Maitre Vascular, Burlington, MA) stent graft to seal the shunt from within the IVC (in addition to aortic repair). Bernstein et al.⁴² encountered thrombosed iliac veins after EVAR repair of ACF for which they recanalized the iliac system and reconstructed it in a "kissing" configuration using a Gore Excluder (Gore, Flagstaff, CA) with Extension and a Gore TAG thoracic endoprosthesis in the IVC. There is continued apprehension for deep venous thrombosis in endovenous stenting of the

caval side of the ACF, and the question of anticoagulation is a difficult one both long term and immediately postoperative. There are little data to guide us on anticoagulation for endovenous stenting in the setting of an endoleak and ACF, but it is concerning that anticoagulation would persist the untoward aortovenous circuit.

It is intuitive that endovascular surgery, which places less physical stress on the body, may be more appropriate for patients who are unlikely to survive an open surgery. However, this statement requires examination. The most obvious choice might be those patients who have a ruptured aneurysm or those patients with decompensated heart failure. Although the Immediate Management of the Patient with Rupture: Open versus Endovascular Repair (IMPROVE) aneurysm trial⁴⁸ reported that endovascular repair was not associated with significant reduction in 30-day mortality in patients with ruptured AAAs, many proponents feel that if patients can be treated by EVAR, survival is superior. The limiting factor in many cases of open repair for ruptured aneurysms with ACF is that the institution does not offer the appropriate endovascular graft “off the shelf.” Siepe et al.¹⁶ presented a hybrid approach in 2009. The patient presented with malperfusion and rapidly deteriorated into shock; 2 aortic stent grafts (Medtronic Inc, Fidelity, MN) were placed into the IVC first then after hemodynamics stabilized, transitioned to an open repair. The juxtarenal anatomy excluded it from an endovascular repair of the AAA, but the idea of a hybrid approach is noteworthy and may be a viable option to stabilize the “symptomatic” ACF.

In addition, those patients with inflammatory aneurysms or retroperitoneal fibrosis and ACF deserve mention. There were 5 patients in this review. It has been reported that ruptured inflammatory aneurysms have a high incidence of ACF.⁴⁹ Certainly with inflammatory aneurysms or retroperitoneal fibrosis, an open dissection proves more difficult with increased bleeding risk. Injuries to the IVC or ureters have also been reported complications of inflammatory aneurysms. Thus, these patients may be best served by endovascular repair if possible.

There still remain patients who are not ruptured and are relatively fit for open surgery. The ongoing concern would be management of the overloaded right ventricle and pulmonary hypertension. Most ACF patients will have some degree of heart failure, but hemodynamic changes caused by the ACF are completely reversed after ACF closure and latitude for operative risk to be improved through surgery is a realizable goal. In most, systemic vascular resistance increases whereas central venous pressure, pulmonary artery pressure, stroke volume, and cardiac output all decrease.⁵⁰ Thus, if patients are not limited by significant coronary artery disease, then even moderate heart failure alone should not preclude a patient from open repair.

When considering a high reported mortality rate for open repair of ACF, many surgeons still look to endovascular repair first; whether the AAA is ruptured or not. Five deaths were reported with EVAR and ACF, which represented 19% of the endovascular group. However, one was 3 months postoperative and may have been unrelated to the surgery.²⁷ Furthermore, Akwei et al.³⁸ reported 4 endovascular cases of which 3 died, but all three of those deaths had been admitted to the hospital as many as 6 days before the diagnosis of the ACF. Thus, delay in diagnosis and treatment likely played a factor in their outcome, and these patients

were not likely to fare better with open surgery. Therefore, if a delayed diagnosis is not a factor, the 30-day mortality is adjusted to 3.8% instead of the crude 19% estimate. This is far less than the 12% mortality rate of open repair demonstrated herein. Indeed the improvement in mortality rate of the ACF condition, we have revealed is duly noted for endovascular and open repair, and compares quite favorably to the historical literature.

Conclusions

Forty papers were reviewed with a total of 67 patients with AAA and ACF, the largest series of ACF known to date. Open surgery remains a viable option to patients who are relatively fit; however, endovascular repair may provide a lower risk of 30-day mortality. Nonetheless, a high incidence and persistence of endoleak with the endovascular approach requires caution. The continued presence of an endoleak in the setting of an ACF may result in several untoward outcomes: probable persistence of the ACF, ongoing heart failure risk, unlikely thrombosis of endoleaks, disseminated intravascular coagulation, and even continued sac enlargement. We would agree for these patients that endoleak management should be aggressively pursued. Numerous techniques have been employed for this complication including endovenous stenting and Amplatzer duct occluders, but longer follow-up is needed. Overall, the endovascular approach demonstrates merit and is a viable option for many ACF. Furthermore, timely diagnosis of ACF to manage intraoperative cardiac performance and to recognize the unique nature of endoleak risk to the patient after ACF repair may be central to promoting the improved mortality risk of ACF we appreciated in this modern assessment of outcomes.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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Fig. 1.
Three-dimensional reconstruction of CT angiography. *White arrow*, large aortocaval fistula.

Fig. 2.
Intraoperative transesophageal echocardiogram. Massively dilated right atrium.

Table I
Demographics and presentation

	Open	Endo	Analysis
	<i>n</i> = 41, <i>n</i> (%)	<i>n</i> = 26, <i>n</i> (%)	(<i>P</i> value)
Demographic			
Age (years)	68 (44–85)	70 (56–80)	–
Male gender	40 (97.5)	24 (92.3)	0.332
Presentation			
Shortness of breath	8 (19.5)	8 (30.7)	0.223
Leg swelling	3 (7.3)	10 (38.4)	0.002
Abdominal bruit	2 (4.8)	1 (3.8)	0.668
Pulsatile mass	3 (7.3)	2 (7.7)	0.709
Abdominal/back pain	31 (75.6)	12 (46.1)	0.015
Hemorrhagic shock	7 (17)	3 (11.5)	0.418
Ruptured	25 (60.9)	6 (23.1)	0.002

Anatomy**Table II**

	Open	Endo	<i>P</i> value
	<i>n</i> = 41, <i>n</i> (%)	<i>n</i> = 26, <i>n</i> (%)	
ACF	36 (87.8)	22 (84.6%)	
Infrarenal ACF	35 (85.3)	22 (84.6)	0.673
Juxtarenal ACF	1 (2.4)	0 (0)	0.612
Iliocaval fistula	2 (4.8)	3 (11.5)	0.291
Aortoiliac fistula	3 (7.3)	1 (3.8)	0.494
Inflammatory/retroperitoneal fibrosis	3 (7.3)	2 (7.7)	0.710
Ruptured	25 (60.9)	6 (23.1)	0.024

Bold indicates $P < 0.05$ for open versus endo.

Table III
Intraoperative approach

Open	
Tsolakis 1999 ⁷	Repaired primarily within sac ³ , IVC ligation ¹
Ferrari 2000 ⁸	Patch angioplasty
Takazawa 2001 ⁹ (2 cases)	Patch angioplasty
Gandini 2002 ¹⁰	Repaired primarily within sac
Espinel 2006 ¹¹	Retrograde balloon occlusion of IVC, repaired primarily within sac
Iriz 2006 ¹²	Patch angioplasty
Maeda 2007 ¹³ (5 cases)	All repaired primarily within sac
Laxdal 2007 ¹⁴	Repaired primarily within sac
Takaseya 2007 ¹⁵	Repaired primarily within sac
Siepe 2009 ¹⁶	IVC stent followed by open primary repair
Pevec 2010 ¹⁷	Repaired primarily within sac
Kondo 2011 ¹⁸ (2 cases)	1. Aortic exclusion (tight closure of aortic sac) 2. Repaired primarily within sac
Oda 2011 ¹⁹	Repaired primarily within sac
Kotsikoris 2012 ²⁰	All repaired primarily within sac
Laporte 2012 ²¹	Repaired primarily within sac
Unosawa 2013 ²²	Repaired primarily within sac
Nakazawa 2014 ²³	Repaired primarily within sac
Madani 2014 ⁴⁶	Retrograde balloon occlusion of IVC, Repaired primarily within sac
Orion 2014	Repaired primarily within sac
Endovascular	
Umscheid 2000 ²⁴	Vanguard bifurcated (Boston Scientific)
Lau 2001 ²⁵	Aorto-uni, custom
Duxbury 2002 ²⁶	Gore limb Excluder
Vetthus 2005 ²⁷ (2 cases)	Tri-Fab Cook ²
Godart 2006 ²⁸	Amplatz duct occluder
Hetzel 2006 ²⁹	Lifepath iliac extension (Edwards Lifesciences)
Kopp 2006 ³⁰	Zenith bifurcated Cook
Fukuda 2007 ³¹	Aorto-uni, custom
Leon 2007 ³²	Gore excluder
Kwon 2008 ³³	Gore excluder
Juszkat 2009 ³⁴	Zenith iliac extension
Guzzardi 2010 ³⁵	Gore excluder, IVC filter
Melas 2011 ³⁶	Aorto-uni La Maitre
LaBarbera 2011 ³⁷	Gore bifurcated, converted to aorto-uni
Akwei 2011 ³⁸ (4 cases)	1. Tri-Fab Cook

Open	
	2. Zenith aorto-uni
	3. Bi-Fab Cook
	4. Zenith Cook aorto-uni
Yuminaga 2012 ³⁹	Zenith Cook with Tri-Fab limb extension
Rapacciuolo 2012 ⁴⁰	Amplatzer plug then delayed Talent stent graft (Medtronic)
Shah 2012 ⁴¹	C3 Gore deployed within sac and extended proximally
Bernstein 2013 ⁴²	Gore excluder intra-aortic, delayed Gore TAG intracaval
van de Luitgaarden 2013 ⁴³	Endurant (Medtronic)
Silveira 2014 ⁴⁴	Gore excluder intra-aortic, Gore aortic cuff intracaval
Nakad 2014 ⁴⁵	Endurant (Medtronic)

Table IV
Open repair–postoperative

Author	Complication	Follow-up (months)
Tsolakis 1999 ⁷	Death in 3 patients (1 also had spinal ischemia)	None
Ferrari 2000 ⁸	Acalculous cholecystitis, pseudomembranous colitis	10
Takazawa 2001 ⁹ (2 cases)	Renal failure in 1 patient, cholecystitis in 1 patient	None
Gandini 2002 ¹⁰	Recurrence of ACF and converted to endovascular repair	24
Espinel 2006 ¹¹	None	None
Iriz 2006 ¹²	Renal failure	0.75
Maeda 2007 ¹³ (5 cases)	Death in 1 patient, paraplegia in 1 patient	None
Laxdal 2007 ¹⁴	Lower-extremity ischemia, respiratory failure	12
Takaseya 2007 ¹⁵	None	None
Siepe 2009 ¹⁶	Renal failure, respiratory failure, atrial fibrillation, wound infection	None
Pevac 2010 ¹⁷	None	None
Kondo 2011 ¹⁸ (2 cases)	Bowel obstruction in 1 patient	1
Oda 2011 ¹⁹	None	None
Kotsikoris 2012 ²⁰	Deep vein thrombosis, death in 1 patient	Average 18.5
Laporte 2012 ²¹	None	None
Unosawa 2013 ²²	None	12
Nakazawa 2014 ²³	None	30
Madani 2014 ⁴⁶	None	None
Orion 2014	None	12
	Mean follow-up	8.8

Table V
Endovascular repair–postoperative

Author	Complication	Endoleak	Follow-up (months)
Umscheid 2000 ²⁴	Compression of right iliac limb	Yes	6
Lau 2001 ²⁵	Stent occlusion, leg ischemia	Yes	12
Duxbury 2002 ²⁶	Prolapse into ACF, IVC thrombus	No	1
Vettrhus 2005 ²⁷ (2 cases)	1. None	1. Yes	1. 12
	2. Death at 3 months	2. No	2. 3
Godart 2006 ²⁸	None	No	6
Hetzel 2006 ²⁹	None	No	None
Kopp 2006 ³⁰	Inferior mesenteric artery coiled	Yes	6
Fukuda 2007 ³¹	None	No	48
Leon 2007 ³²	None	No	1.25
Kwon 2008 ³³	None	No	5
Juszkat 2009 ³⁴	None	No	None
Guzzardi 2010 ³⁵	None	Yes	12
Melas 2011 ³⁶	Enlarging sac requiring IVC stent graft	Yes	36
LaBarbera 2011 ³⁷	Persistent type II, Amplatzer VSD occluder to ACF and Inferior mesenteric artery coiled	Yes	1
Akwei 2011 ³⁸ (4 cases)	1. Death day 3	1. No	1. None
	2. Death day 1	2. Yes	2. None
	3. Death hour 1	3. No	3. None
	4. Persistent type Ia and sac enlargement, extended with Palmaz	4. Yes	4. 12
Yuminaga 2012 ³⁹	None	Yes	1.5
Rapacciuolo 2012 ⁴⁰	None	No	1
Shah 2012 ⁴¹	None	Yes	1
Bernstein 2013 ⁴²	Complete thrombosis of left iliac veins and near thrombosis of IVC, ischemic colitis, death at day 35	Yes	None
van de Luitgaarden 2013 ⁴³	Persistent ACF after 1 year	Yes	12
Silveira 2014 ⁴⁴	None	No	24
Nakad 2014 ⁴⁵	None	No	None
	Mean follow-up		7.7