Outcomes of accessory renal artery occlusion during endovascular aneurysm repair

Jagajan Karmacharya, MD, Shane S. Parmer, MD, James N. Antezana, MD, Ronald M. Fairman, MD, Edward Y. Woo, MD, Omaida C. Velazquez, MD, Michael A. Golden, MD, and Jeffrey P. Carpenter, MD, Philadelphia, Pa

Objective: Accessory renal arteries are frequently encountered when patients are evaluated for endovascular abdominal aortic aneurysm repair (EVAR). Some have considered their presence a contraindication to EVAR in fear of endoleak and the end result of renal function. We sought to determine whether the coverage of accessory renal arteries during EVAR was associated with any adverse sequelae.

Methods: Retrospective review of the medical records and computed tomographic scans of all patients undergoing EVAR (1998 to 2003) was performed. Note was made of the presence or absence of accessory renal arteries, hypertension, and renal function. Preoperative computed tomographic images were compared with postoperative images to determine the presence of renal infarction. A control group of 26 consecutive patients without accessory renal arteries was used for comparison of the results of EVAR.

Results: EVAR was performed in 550 patients over the study interval. The mean follow-up was 16 months (range, 1–48 months). The average age was 74 years (range, 57–90 years). Thirty-five patients (6.6%; 32 male and 3 female) were documented to have accessory renal arteries; the average number of accessory arteries was 2 (range, 1–4). Bilateral accessory arteries were present in 13 patients: all but 1 patient (n = 34) had a left-sided accessory renal artery, and 23 had a right-sided accessory renal artery. EVAR was performed with a variety of endografts: AneuRx (n = 10), Talent (n = 7), PowerLink (n = 7), Zenith (n = 5), Lifecath (n = 4), and Ancure (n = 2). There were no mortalities. Twelve endoleaks were documented: three type I, eight type II, and one type III. The accessory renal arteries were not implicated in any of the endoleaks, and none of these accessory vessels was embolized before or after EVAR. Seven patients (20%) had renal infarcts associated with EVAR that were noted on follow-up computed tomographic scans. The mean follow-up for patients with segmental infarction was 23 months (range, 8–48 months). Hypertensive status did not change in any patient in whom an accessory renal artery had been covered. The average serum creatinine was 1.08 mg/dL (range, 0.6–1.8 mg/dL) before EVAR in patients with accessory renal arteries covered by an endovascular graft and did not change significantly in response to EVAR. Serum creatinine increased almost twofold in two patients but spontaneously resolved in follow-up. The average preoperative creatinine clearance was 79 mL/min (range, 35–166 mL/min) in patients without an accessory renal artery and was 80 mL/min (range, 35–167 mL/min) after EVAR. The average preoperative creatinine clearance was 67 mL/min (range, 31–137 mL/min) in patients with an accessory renal artery and 68 mL/min (range, 45–83 mL/min) in patients with renal infarcts. None of the patients required temporary or permanent dialysis. There was no difference between control patients and patients with covered accessory renal arteries with respect to hypertensive status, presence of renal infarcts, serum creatinine, or creatinine clearance after EVAR.

Conclusions: Occlusion of accessory renal arteries is not associated with clinically significant signs or symptoms, even in patients with mild or moderate renal insufficiency. Sacrifice of accessory renal arteries most commonly does not lead to detectable renal infarction, either clinically or radiographically. When segmental infarction of the kidney does result, it seems to be well tolerated in this group of patients. Accessory renal arteries were not found to contribute to endoleaks and should not be prophylactically embolized. (J Vasc Surg 2006;43:8–13.)

Endovascular abdominal aortic aneurysm repair (EVAR) is a valuable option in selected patients, with acceptable morbidity and mortality rates.1 However, not all cases are amendable for EVAR.2 Inadequate proximal neck, tortuosity, and the size of iliac vessels dictate the operative choice and the feasibility of abdominal stent grafts.

In addition, the presence of renal accessory arteries may disfavor the placement of endovascular grafts in abdominal
short proximal neck and its consequent long-term sequelae remain unknown. Medical literature is scarce in regard to complications of segmental renal infarction. Theoretically, the loss of renal parenchyma could lead to worsening of renal function, as determined by an increase in plasma creatinine or blood pressure. We sought to determine whether coverage of the accessory renal arteries during EVAR was associated with any adverse long-term sequelae.

METHODS

Retrospective review of the 550 consecutive medical records and computed tomographic (CT) scans of all patients who underwent EVAR (1998 to 2003) was performed. Note was made of the presence or absence of accessory renal arteries by imaging studies. Accessory renal arteries were defined as supernumerary renal arteries smaller than and located below the main renal artery. Supernumerary renal arteries located above the main renal artery (and therefore not considered for coverage during EVAR) were not included in the analysis. Patients’ medical history of hypertension was reviewed, as was their renal function. All CT scans were performed in a standard manner. Initially non–contrast-enhanced images in 7.5 helical collimation were performed. This was followed by a test bolus of 16 mL of contrast. High-resolution CT angiograms (2.5-mm cuts) were performed, followed by 4 mL/s intravenous contrast bolus administration of nonionic contrast per CT angiography protocol. A delayed postcontrast examination of the abdomen and pelvis was performed in 5-mm collimation. Preoperative CT scans were compared with postoperative images to determine the presence of renal infarction. Patients were followed up initially at quarterly intervals and subsequently at annual intervals. At each office visit, blood pressure was measured and recorded.

Clinical characteristics of patients with renal accessory artery were compared with those of a series of consecutive patients without renal accessory arteries (n = 26). Statistical analysis was performed by using the Student t test for paired data, with a significance level of P < .05.

RESULTS

EVAR was performed in 550 patients over the study interval (Table I). Of those screened for EVAR candidacy, six were excluded from EVAR because of the presence of supernumerary renal arteries that were thought to be essential. The mean follow-up was 16 months (range, 1-48 months). Thirty-five patients (6%; 32 male and 3 female) were documented to have accessory renal arteries; the average number of accessory arteries was 2 (range, 1-4). Bilateral accessory arteries were present in 13 patients. All but one patient (n = 34) had a left-sided accessory renal artery, and 23 had a right-sided accessory renal artery. The average age was 74 years (range, 57-90 years).

In the control group, the average age was 74 years (range, 61-86 years; Table II). All 26 patients had infrarenal fixation by using the unibody bifurcated graft (PowerLink; Endologix Inc, Irvine, Calif). Five control patients (19%) had small renal infarcts in association with EVAR on follow-up CT scans. The average preoperative blood pressure in this control group was 131/93 mm Hg (range, systolic 93-205 mm Hg, diastolic 55-114 mm Hg; Table III). The average blood pressure on follow-up was 151/85 mm Hg (range, 108-211 over 55-114 mm Hg). The average preoperative blood pressure in patients with segmental renal infarction (in the control group) was 145/69 mm Hg (range, 101-205 over 60-109 mm Hg) and on follow-up was 159/92 mm Hg (range, 150-211 over 73-114 mm Hg). Two patients in this control group had pre-existing high systolic blood pressure. Their hypertensive status remained unchanged after EVAR. Significant differences in serum creatinine values were not appreciated after EVAR for patients in the control group (Table III). The average preoperative serum creatinine was 1.1 (range, 0.7-1.8) and on follow-up was 1.06 (range, 0.6-1.8; not significant). The average preoperative creatinine in patients who developed segmental renal infarcts was 1.42 (range, 1.1-1.8), and the creatinine level was 1.1 (range, 1.1-1.8).
after surgery. Serum creatinine was elevated prior to EVAR in two patients and did not change on follow-up. The average preoperative creatinine clearance was 79 mL/min, and after surgery it was 80 mL/min. The preoperative creatinine clearance in the control group that developed renal infarcts was 68 mL/min (range, 35-91 mL/min), and creatinine clearance was 66 mL/min (range, 35-91 mL/min) on follow-up.

EVAR was performed with a variety of endografts in the patients with covered accessory renal arteries: AneuRx (Medtronic, Minneapolis, MN) (n = 110/10), Talent (Medtronic, Minneapolis, MN) (n = 7), PowerLink (Endologix, Inc, Irvine, Calif) (n = 7), Zenith (COOK, Bloomington, Ind) (n = 5), LifePath (Edwards Lifesciences, Corp, Irvine, Calif) (n = 4), and Ancure (Guidant, Menlo Park, Calif) (n = 2). There were no mortalities. Twelve endoleaks were documented in follow-up: three type I, eight type II, and one type III. The accessory renal arteries were not implicated in any of the endoleaks, and none of these accessory vessels required embolization before or after EVAR. All accessory renal arteries thrombosed after coverage (Fig 1).

Seven patients (20%) had renal infarcts associated with EVAR on follow-up CT scans (Fig 2). The mean follow-up for patients with segmental infarction was 23 months (range, 8-48 months).

Hypertensive status did not change in association with renal segmental infarction or in any patient in whom an accessory renal artery had been covered (Table II). The average preoperative blood pressure in our 35 patients was 142/76 mm Hg (range, 119-180 over 62-96 mm Hg). The average blood pressure on follow-up was 140/74 mm Hg (range, 106-172 over 56-96 mm Hg). The average blood pressure in patients with segmental renal infarction before surgery was 151/72 mm Hg (range, 136-170 over 62-90 mm Hg) and on follow-up was 147/67 mm Hg (range, 102-170 over 56-75 mm Hg). Two patients in our series had pre-existing high systolic blood pressure. Their hypertensive status remained unchanged after EVAR. There was no difference between accessory renal artery and control patients with respect to hypertensive status or blood pressure before or after EVAR.

Significant differences in serum creatinine values were not appreciated after EVAR for patients with covered accessory renal arteries (Table III). The average preoperative serum creatinine was 1.08 (range, 0.6-1.8), and on follow-up it was 1.11 (range, 0.6-1.8; P = .1; not significant). The average postoperative creatinine in patients who developed segmental renal infarcts was 1.05 (range, 0.9-2.7). Serum creatinine increased almost twofold in two patients but spontaneously resolved in follow-up. There were no differences between accessory renal artery and control pa-

<table>
<thead>
<tr>
<th>Variable</th>
<th>Preoperative mean (range)</th>
<th>Postoperative mean (range)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Serum creatinine</td>
<td>Accessory RA (n = 35) 1.08 (0.6-1.8)</td>
<td>1.11 (0.6-1.8)</td>
</tr>
<tr>
<td>Control group (n = 26) 1.1 (0.7-1.8)</td>
<td>1.06 (0.6-1.8)</td>
<td></td>
</tr>
<tr>
<td>Accessory RA with renal infarcts (n = 7) 1.15 (0.9-1.6)</td>
<td>1.28 (0.9-2.7)</td>
<td></td>
</tr>
<tr>
<td>Control group with renal infarcts (n = 5) 1.4 (1.1-1.8)</td>
<td>1.1 (1-1.8)</td>
<td></td>
</tr>
<tr>
<td>Creatinine clearance (mL/min)</td>
<td>Accessory RA (n = 35) 67 (31-137)</td>
<td>67 (25-141)</td>
</tr>
<tr>
<td>Control group (n = 26) 79 (35-166)</td>
<td>80 (35-167)</td>
<td></td>
</tr>
<tr>
<td>Accessory RA with renal infarcts (n = 7) 63 (37-83)</td>
<td>68 (45-83)</td>
<td></td>
</tr>
<tr>
<td>Control group (n = 5) 68 (35-91)</td>
<td>66 (35-106)</td>
<td></td>
</tr>
<tr>
<td>Hypertensive status (mm Hg)</td>
<td>Accessory RA (n = 35) 142/76 (119-180 over 62-96)</td>
<td>140/74 (106-172 over 56-96)</td>
</tr>
<tr>
<td>Control group (n = 26) 131/93 (93-205 over 55-114)</td>
<td>151/85 (108-211 over 55-114)</td>
<td></td>
</tr>
<tr>
<td>Accessory RA with renal infarcts (n = 7) 151/72 (136-170 over 62-90)</td>
<td>147/67 (102-170 over 56-75)</td>
<td></td>
</tr>
<tr>
<td>Control group (n = 5) 145/69 (101-205 over 60-109)</td>
<td>159/92 (150-211 over 73-114)</td>
<td></td>
</tr>
</tbody>
</table>

patients with respect to serum creatinine before and after EVAR.

Both preoperative and postoperative creatinine clearance were 67 mL/min (range, 25-141 mL/min) in patients with renal accessory arteries. Similarly, creatinine clearance did not change significantly for control patients after EVAR (Table III). There were no differences between patients with covered accessory renal arteries and controls with respect to serum creatinine before and after EVAR.

**DISCUSSION**

EVAR is an evolving surgical technique. However, secure fixation and seal within the proximal neck continue to be a challenge. A generally accepted requisite is the presence of an adequate proximal neck length (≥15 mm). Many patients do not fulfill this criterion and are excluded. A relative contradiction to EVAR is the presence of accessory renal arteries, which would be covered by the stent graft. In our prior report of patients excluded for EVAR, we noted six patients whom we excluded from EVAR candidacy on the basis of the presence of accessory renal arteries. In this former article, we excluded these patients from EVAR because of the presence of supernumerary renal arteries in a configuration such that the main renal artery would of necessity be covered during EVAR. In our present study of 550 EVAR patients, 35 patients had coverage of accessory renal arteries. All of these patients had supernumerary renal arteries, but in a configuration such that the smaller accessory arteries, rather than the main renal artery, were covered during EVAR. Although seven patients developed renal infarcts, as documented by postoperative CT scans, there was no significant change in the renal function or hypertensive status of these patients, and there were no differences between patients with covered accessory renal arteries and controls.

The long-term clinical implication of excluding the accessory renal arteries is not fully known. With findings similar to our own, Aquino et al reviewed 311 patients treated with EVAR for abdominal aortic aneurysms. They documented 24 patients with covered accessory renal arteries, using the Ancure device in 23 patients and the Excluder in one. Five patients developed segmental infarction as a result of accessory renal artery exclusion. Of these patients, only one developed significant hypertension; this resolved within 3 months, similar to our own findings. They too noted that serum creatinine remained unchanged during follow-up (mean, 11.5 months) in patients whose accessory renal arteries had been covered during EVAR. Kim et al reported experience with coverage of 11 accessory renal arteries by using the AneurX graft, also with no significant increase in serum creatinine, hypertension, or detectable renal infarctions.

Others have reported the lack of clinical effect associated with segmental renal infarction. In their review of 204 patients who underwent EVAR, noted small segmental infarcts on follow-up CT scans in less than 20% of patients. Hypertension was not noted in their patient population.

Our own findings and those of the others detailed previously question the traditional teaching that accessory renal arteries are end arteries. Sacrifice of these arteries does not inevitably lead to renal infarction, at least such as is detectable by CT scanning. The incidence of detectable renal infarction is much less than that of renal accessory artery sacrifice in all reports to date.

In an attempt to quantify the volume of renal mass perfused by accessory renal arteries, Dorffner et al evaluated the use of selective renal angiography and spiral CT scanning to assess the effect of accessory renal artery sacrifice. The amount of renal mass supplied by the accessory artery was measured by computed tomographic angiography with selective injection of the accessory renal artery. The volume of renal infarction was equal to the renal volume perfused by the artery. The maximum amount of renal tissue that could be safely sacrificed could not be precisely determined, but they arbitrarily suggested safety of coverage of accessory renal arteries that supply no more than 32% of the total renal mass, in normotensive patients with normal renal function.

We noted no effect on blood pressure from renal artery coverage. Gupta and Tello have suggested that the accessory renal arteries are a vascular anomaly with no direct ability to cause hypertension. In their retrospective review of 185 hypertensive patients, 45 had renal accessory arteries. They found that accessory renal arteries did not play a role as an anatomically treatable cause of hypertension. In keeping with this, we found no discernible effect in blood pressure in response to the coverage of accessory renal arteries.

Renal insufficiency is a relative contraindication to EVAR, and the causes of renal dysfunction and renal infarction after EVAR are multifactorial. Serum creatinine increased after EVAR in only two of our patients when accessory renal arteries were covered. Both of these had baseline renal insufficiency, and in both instances, creatinine reverted to baseline in follow-up. Even in the setting of
mild to moderate renal insufficiency, accessory renal artery coverage seemed well tolerated. We continue to monitor our patients and tailor strategies to minimize adverse renal effects. Our routine practice in the setting of renal insufficiency and the possibility of accessory renal exclusion is to vigorously hydrate perioperatively, use osmotic diuretics, and decrease or avoid the use of nephrotoxic contrast, using gadolinium for imaging studies before, during, and after EVAR.

Although we did not note any endoleaks attributable to accessory renal arteries, one such case has been reported by Aquino et al. They described a patent accessory renal artery that provided outflow to a distal type I attachment leak. The accessory renal artery thrombosed after treatment of the attachment site leak. It is unnecessary to prophylactically embolize these vessels when coverage or exclusion is planned. Coverage of accessory renal arteries by endografts results in immediate thrombosis. Exclusion of accessory renal arteries arising from the aneurysm sac poses little threat of type II endoleak, because these are end arteries, with little possibility of back bleeding.

CONCLUSION

In summary, our study and those of others suggest that occlusion of accessory renal arteries is not associated with clinically significant signs and symptoms of renal impairment, even in patients with mild or moderate renal insufficiency. The amount of renal loss associated with sacrifice of accessory renal arteries is difficult to determine, and the consequences are unknown, but sacrifice of accessory renal arteries most commonly does not lead to detectable renal infarction, either clinically or radiographically. Our study and those of others suggest that when segmental renal infarction does result, it is well tolerated. In addition, we did not find that accessory renal arteries were a cause of endoleaks, and therefore preoperative embolization of accessory renal arteries was not necessary to prevent endoleak after EVAR.

AUTHOR CONTRIBUTIONS

Conception and design: JK, JPC
Analysis and interpretation: JK, SSP, JNA, JPC

Data collection: JK, SSP, JNA, JPC
Writing the article: JK, JPC
Critical revision: JK, RMF, MAG, OCV, EYW, JPC
Final Approval: JK, SSP, JNA, RMF, MAG, OCV, EYW, JPC
Statistical analysis: JK, SSP, JPC
Overall responsibility: JPC

REFERENCES


Submitted Apr 23, 2005; accepted Sep 3, 2005.
4. Did you assess the main renal arteries for atherosclerotic disease? Could there have been a subset of patients with the presence of atherosclerotic renal artery disease who then had a worse outcome postoperatively?
5. Did you measure the diameter of the accessory renal arteries that you covered? We know from the open surgical literature that small renal artery size can be sacrificed without sequelae. Could it be that the accessory renal arteries that you sacrificed are of insignificant diameter?
6. Did you expect to see an increase in the baseline blood pressure in patients with renal infarctions?
7. You mentioned that 100% of patients with pre-existing renal insufficiency had worsening of their renal function postoperatively. Has this changed your approach to patients with accessory renal arteries during EVAR in the presence of renal dysfunction? For example, would you suggest the use of Mucomyst, fenoldopam, or bicarbonate infusion preoperatively?
8. How much contrast did you use?
9. You used an assortment of endograft devices. Of those patients who developed renal insufficiency, did you look to see if any of those patients received grafts with suprarenal fixation?
10. Lastly, you mentioned that you know of no incidence in which an accessory renal artery caused a type II endoleak. Dr White et al published a paper in the Journal of Endovascular Therapy in August of 2000 describing an 81-year-old patient who underwent EVAR, subsequently experienced rupture, and died due to an accessory renal artery. How would you correlate this with your data?

Dr Karmacharya. The first question was the incidence of type I endoleak and our overall EVAR experience. Yes, in our paper, we had 3 type I endoleaks. This is a small study and I do not think it reflects our total experience of type I endoleaks in our institution. The incidence of type I endoleaks would be approximately 1% in our series.

When was the type I recognized? We recognized type I leaks in the OR [operating room]. We fixed the leaks using a variety of tools, mostly a combination of stents and deployment of balloon cuffs.

Could we have missed a type II endoleak at the proximal fixation site due to the presence of an accessory renal artery? Yes, it is possible. But in our study, all accessory renal arteries that we covered thrombosed; thus, it did not contribute to an endoleak.

How do we document if there was a type II endoleak? We did not see a type II endoleak on follow-up scans, due to an accessory renal artery, because the accessory renal artery was covered.

The third question was when in the postoperative course was the creatinine level elevated? Could we have missed that resolution of the serum creatinine to baseline, therefore missing the window during which ATN occurred? Do we consider looking at creatinine clearance? I think that’s a very good question. Yes, we could have missed the window, because patients were evaluated at quarterly intervals and then annually, so indeed we could have missed a rise in serum creatinine and ATN that resolved.

Did we consider looking at creatinine clearance? I do not have the creatinine clearance for this presentation. However, prospective studies in the future would be useful.

Did we measure the diameter of the accessory renal arteries covered? No.

And could the renal accessory arteries you sacrifice be of insignificant diameter? Yes, Dorffner et al reported the volumes an accessory renal artery supplied. He used CTA arteriography to evaluate renal mass and vascular volume and found that the size of the renal vessel does correlate with the volume of blood supply and that up to 32% could be sacrificed without any long-term sequelae. But again, his study was a very small study, and I think we need more data.

Did you expect to see a rise in baseline blood pressure in patients with renal infarctions? No, we didn’t. Gupta and Tello actually demonstrated that renal infarcts did not correlate with an increase in blood pressure.

Question 7: you mentioned that 100% of those patients with pre-existing renal insufficiency have developed worsening of their renal function postoperatively. Has this changed our practice? This is in reference to our earlier article of September 11, 2001. We have since altered our practice. We use gadolinium. If we do have to use renal contrast, we do so in very small amounts, about 10 to 20 cc. We would do selective renal angiography to identify the renal vessels and not necessarily do a complete aortogram. We hydrate the patients pre- and postoperatively.

How much contrast do we use in patients with renal insufficiency? We try to avoid contrast at all costs.

Question 9: we used an assortment of endografts. Of those patients who developed renal insufficiency, did you look at any of those patients who received grafts with suprarenal fixation? That’s a very interesting question. And as a matter of fact, we have not in this study, but we have a control study that will soon be presented comparing the use of the PowerLink suprarenal vs infrarenal fixation. And preliminarily, I can tell you that there were no statistical differences.

Question 10: lastly, you mentioned that no instances of accessory renal artery caused a type II endoleak, and then the question refers to Dr White’s paper that came out in August 2000 in Endovascular Therapy where an 81-year-old patient died of a type II leak. How do we correlate this with our finding? Well, we did not specifically find an endoleak type II that could be attributed to the accessory renal artery. Lacrina et al actually had found that their type I leak was the source of a feeding accessory renal artery, which was recognized postoperatively, but we didn’t see that. The main thing, I think, is to recognize your leaks and to treat them. I think that’s what highlights that case report.

Dr Larry Scher (Bronx, Ny). Since this was a retrospective review, I’m wondering if you know if any patients were excluded before EVAR was attempted based on the presence of accessory renal arteries. You might have excluded patients with more significant accessory renal arteries that would skew the data.

Dr Karmacharya. Absolutely, you’re correct. But did we know which patients were excluded based on those findings? No.

Dr Jeffrey P. Carpenter (Philadelphia, Pa). We did write a manuscript about who can’t have a stent graft and why that covered roughly the same time interval, and there were only 6 patients out of our entire population that was screened who were excluded on the basis of what we thought to be an essential accessory renal artery.

Dr Scher. Thank you for that answer.