# The effect of warfarin therapy on endoleak development after endovascular aneurysm repair (EVAR) of the abdominal aorta

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***Objectives:*** **The presence of an endoleak after endovascular abdominal aortic aneurysm (AAA) repair (EVAR) may predispose to sac expansion and potential sac rupture. The incidence of endoleak after AAA repair can be as high as 20% to 30%. We investigated whether warfarin anticoagulation was an independent risk factor for endoleak after EVAR for AAA.**

***Methods:*** **All AAA patients who underwent elective EVAR were prospectively followed-up. Data for demographics, clinical comorbidities, outcomes, EVAR devices, and anticoagulation methods were recorded. All patients underwent routine follow-up at 1, 6, and 12 months and annually thereafter. Computed tomography angiography (CTA) with 3-dimensional (3D) volumetric analysis was also completed.**

***Results:*** **During a 7-year period, 127 consecutive patients with infrarenal AAAs who underwent EVAR were monitored for a mean of 2.14 years. The average age at the time of EVAR was 73.8 years. Warfarin therapy alone was administered to 24 patients, and anticoagulation with antiplatelet therapy alone was administered to 103. During the study period, 38 (29.9%) endoleaks were documented. The overall endoleak rate was 13 of 24 in the warfarin group and 25 of 103 in the antiplatelet group ($P = .004$). CTA 3D volumetric aneurysm sac analysis showed an increase of 16.09% in the warfarin study group and a reduction of 9.71% in the antiplatelet group ($P = .04$).**

***Conclusions:*** **Anticoagulation with warfarin appears to be linked to an increased risk for the development of endoleak after EVAR, specifically type II. Volumetric analysis showed warfarin therapy also contributed to persistent aneurysm sac expansion. These data suggest that patients who require warfarin anticoagulation for other indications should be advised that they might be at an increased risk for the development of endoleaks, subsequent secondary interventions, persistent sac expansion, and possible delayed sac rupture. (J Vasc Surg 2010;52:267-71.)**

Each year, 200,000 abdominal aortic aneurysms (AAAs) are diagnosed in the United States alone, and >15,000 aneurysms reach the size criterion for repair.[1,2] The peak incidence of diagnosis is in men aged 65 to 75 years. With the recent approval for Medicare-sanctioned, one-time, entry AAA ultrasound screening in men qualifying by the US Preventive Services Task Force guideline recommendations, an increase in the number of patients with AAAs requiring monitoring and repair is anticipated.[2]

Although traditional open AAA repair is safe and effective in the elective setting, endovascular aneurysm repair (EVAR) for AAAs has gained significant popularity due to the growing complexity of patients' comorbid conditions, the need to provide cost-effective care, and the push toward minimally invasive and endoluminal therapies. The primary aim of EVAR is to exclude flow from the sac while maintaining distal flow with covered stent grafts. Endoleak is precisely defined as:

> " . . . a condition associated with endoluminal vascular grafts, defined by the persistence of blood flow outside the lumen of the endoluminal graft, but within an aneurysm sac or adjacent vascular segment being treated by the graft . . . ."[3]

Endoleaks are classified as type I through type IV.[3,4] There have been conflicting data about whether AAA patients who require warfarin therapy after EVAR are predisposed to the development of endoleaks[5-7] as well as for the risk factors for endoleak development in general.[8-12] The purpose of this study was to determine if warfarin therapy was associated with increased risk for endoleak, specifically type II, after EVAR in our endoluminal experience.

## METHODS

During a 7-year contiguous period from January 1, 2000, to January 31, 2007, all patients with an AAA who underwent EVAR were prospectively enrolled into a clinical database. Data for demographics, clinical comorbidities, outcomes, EVAR devices, and anticoagulation methods were recorded prospectively in a clinical database. All patients underwent routine follow-up at 1, 6, and 12 months and annually thereafter, which included computed tomography angiography (CTA) with 3-dimensional (3D) volumetric analysis. We then completed a retrospective review of the collected data to determine if warfarin therapy increased endoleak rates in these patients.

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The CTA protocol consisted of 5-mm noncontrast helical imaging through the abdomen and pelvis, followed by contrast-enhanced 1.25-mm helical images acquired during the arterial phase of contrast enhancement and delayed 5-mm helical images. Scanning parameters were kept as consistent as possible: 120 kV; smart/automatic tube current range of 80 to 750 mA or 80 to 440 mA; noise index, 16; section thickness/reconstruction interval, 1.25 mm/0.75 mm; pitch, 0.516:1 to 0.625:1; interval, 0.75 mm; and rotation time, 500 msec. For the arterial phase, 150 mL of intravenous contrast material (iohexol [iodine, 300 mg/mL], Omnipaque 300, GE Healthcare, Princeton, NJ), and subsequently, a 50-mL saline chaser bolus, were injected through an antecubital vein at 5 mL/s. Arterial-phase scanning was performed by using automatic scan start with a 6-second diagnostic delay after crossing the threshold attenuation (100 HU). Late-phase images were acquired 70 seconds after injection of the contrast material.

The 3D reconstruction included segmentation of the aneurysm sac, and the sac volume was calculated using a workstation (Volume Viewer 2 Advantage, GE Healthcare, Waukesha, Wisc). Source images were evaluated for the presence of endoleak by an experienced cardiovascular radiologist. The 3D and multiplanar reconstructions, as well as volume measurements, were evaluated to determine aneurysm configuration and volumetric changes over time.[13]

Patients were analyzed for endoleak stratified by anticoagulation method. Subgroups consisted of antiplatelet therapy alone (aspirin or clopidogrel) and warfarin alone. Type I thru IV endoleaks were classified by published guidelines. Predefined end points of the study included time to endoleak, persistent endoleak requiring reintervention, and aneurysm sac volumetric expansion. Rank-sum and *t*-test analyses were used to assess risk factors and other patient characteristic differences. A value of $P < .05$ was considered statistically significant in all analyses.

## RESULTS

During a 7-year period from January 2000 to January 2007, 127 consecutive patients (14 women, 113 men) with AAAs underwent EVAR at the University of Wisconsin–Madison. All aneurysms were infrarenal in nature and were a mean size of 5.9 ± 12 cm at repair. At the time of repair, patients were a mean age of 73.8 ± 8.3 years and had a mean body mass index (BMI) of 28.3 ± 6.9 kg/m$^2$.

Of the 127 patients, 103 were treated with antiplatelet agents alone (aspirin or clopidogrel). Twenty-four patients required warfarin therapy for atrial fibrillation and other cardiovascular comorbidities before EVAR and continued receiving warfarin therapy during the study. The goal international normalized ratio (INR) therapeutic target was 2.0 to 3.0 in all patients. Mean follow-up was 2.14 years.

We attempted to identify outcome-specific differences based on the anticoagulation regimen in our study population. There were no significant differences in age ($P = .37$), gender ($P > .99$), aneurysm size ($P = .80$), BMI ($P = .36$), American Society of Anesthesiologists' (ASA) score ($P = .28$), endograft type ($P = .48$), or average length of follow-up ($P = .38$) between the antiplatelet and warfarin groups (Table I). The incidence of coronary arterial disease (CAD) was higher in the warfarin group (91.7% vs 62.1%, $P = .006$); however, all other comorbidities were equal between the groups (Table I).

**Table I.** Demographic and risk factor analyses[a]

| *Category* | *Antiplatelet group (n = 103)* | *Warfarin group (n = 24)* | P *value* |
|---|---|---|---|
| Gender,[b] No. | | | >.99 |
| Male | 91 | 22 | |
| Female | 12 | 2 | |
| Age,[c] years | 74.1 ± 8.3 | 72.3 ± 8.5 | .37 |
| Aneurysm size,[c] cm | 5.77 ± 1.2 | 6.34 ± 1.2 | .80 |
| BMI,[c] kg/m$^2$ | 28.1 ± 6.8 | 29.5 ± 7.5 | .36 |
| ASA score[c] | 3.09 ± 0.5 | 3.28 ± 0.5 | .28 |
| Comorbidities,[b] No. (%) | | | |
| None | 9 (8.7) | 0 (0) | .21 |
| CAD | 64 (62.1) | 22 (91.7) | .006 |
| COPD | 36 (35.0) | 6 (25.0) | .47 |
| Diabetes mellitus | 18 (17.5) | 8 (33.3) | .096 |
| Hypertension | 68 (66.0) | 17 (70.8) | 0.81 |
| Endograft type,[b] No. | | | 0.48 |
| Ancure[d] | 23 | 4 | |
| AneuRx[e] | 33 | 8 | |
| Zenith[f] | 15 | 3 | |
| Excluder[g] | 32 | 8 | |
| UniGraft KDV[h] | 0 | 1 | |
| Follow-up,[c] days | 743.5 | 948.3 | 0.38 |

*ASA,* American Society of Anesthesiologists; *BMI,* body mass index; *CAD,* coronary artery disease; *COPD,* chronic obstructive pulmonary disease.
[a]Overall, there were no significant differences between the antiplatelet group and warfarin group. Five endovascular devices were used during the study.
[b]Risk factor comparison by Fisher exact test.
[c]Risk factor comparison by Wilcoxon rank-sum analysis.
[d]Guidant, Endovascular Solutions, Melo Park, Calif.
[e]Medtronic, AVE, Santa Rosa, Calif.
[f]Cook Medical Inc, Bloomington, Ind.
[g]W. L. Gore and Assoc., Flagstaff, Ariz.
[h]Guidant Endovascular Systems.

The incidence of endoleak significantly increased in the warfarin group ($P = .004$). A total of 38 endoleaks were documented during the course of this study, including 8 type I, 28 type II, 1 type III, and 1 type IV; of which 13 occurred in the warfarin group (n = 24) and 25 occurred in the antiplatelet group (n = 103). Type II endoleak was significantly more common in the warfarin group ($P = .02$). Clinical progression was documented in nine type II endoleaks associated with warfarin therapy, with sac volumes increasing an average of 46.8 cm$^3$ compared with the mean sac volume change of –3.1 cm$^3$ in the 19 antiplatelet-related type II leaks. Of these type II endoleaks, 4 of 9 (44%) in the warfarin group resolved without secondary intervention, whereas 9 of 19 (47%) in the antiplatelet group resolved similarly. There was no significant difference in the overall mean time to the first detection of endoleak between the two groups (201 days antiplatelet, 158 days warfarin; $P = .40$).

**Table II.** Endovascular aneurysm repair outcomes[a]

| *Category* | *Antiplatelet group (n = 103)* | *Warfarin group (n = 24)* | P *value* |
|---|---|---|---|
| Time to endoleak detection,[b] days (range) | 142 (28-1677) | 158 (31-728) | .4 |
| Endoleaks,[c] No. (%) | 25 (24.3) | 13 (54.2) | .004 |
| Leak type[c] | | | .02 |
| I | 5 | 3 | |
| II | 19 | 9 | |
| III | 1 | 0 | |
| IV[d] | 0 | 1 | |
| Initial volume,[b] $cm^3$ | 195.11 ± 64.8 | 181.84 ± 62.4 | .4 |
| Final volume,[b] $cm^3$ | 174.34 ± 64.5 | 213.34 ± 113.9 | .4 |
| Volume change,[b] $cm^3$ (%) | −18.9 (−9.71) | +29.3 (+16.09) | .04 |
| Repeat interventions,[c] No. (%) | 8 (32) | 6 (46) | .03 |
| EVAR explantations, No. | | | |
| Continued expansion | 0 | 3 | NA |
| Delayed rupture w/explant | 1 | 0 | NA |
| Infected graft | 1 | 0 | NA |
| Overall survival,[b] days (range) | 968.6 ± (54-2432) | 1069.2 ± (55-1812) | .8 |

*NA,* Not applicable.

[a]Warfarin therapy was associated with a significant increase in endoleak formation, predominantly type II. Volumetric analysis by computed tomography angiography showed increased aneurysm sac volumes despite endovascular aneurysm repair.

[b]Parameter comparison by Wilcoxon rank-sum analysis.

[c]Parameter comparison by Fisher exact test.

[d]The type IV endoleak was experienced with a first-generation device. This patient demonstrated continued sac expansion without computed tomography angiography evidence of a type I-III endoleak; at explantation, the mean sac pressure was 60 mm Hg with blunted pulsatility.

The CTA 3D volumetric analyses showed no significant differences in initial aneurysm sac volumes based on preoperative studies (195.11 $cm^3$ antiplatelet, 181.84 $cm^3$ warfarin; $P = .4$). At the conclusion of the study, however, mean sac volume in the warfarin group had significantly increased to 213.34 $cm^3$ compared with a mean decrease in the antiplatelet group to 174.34 $cm^3$. This marked an overall aneurysm sac volume increase of 16.09% (29.3 $cm^3$) in the warfarin study group compared with a reduction of 9.71% (18.9 $cm^3$) in the antiplatelet group ($P = .04$). This disparity was most evident in the proven endoleak groups, with an average change of +38.5 $cm^3$ in the warfarin endoleak-positive group compared with −8.9 $cm^3$ in the antiplatelet endoleak-positive group ($P = .04$). A similar although not statistically significant trend was seen in those patients without a proven endoleak (warfarin endoleak-negative group, +6.2 $cm^3$; antiplatelet endoleak-negative group, –23.5 $cm^3$; $P = .14$).

There were 14 repeat interventions during the course of this study: 8 of 25 (32%) in the antiplatelet group required repeat intervention vs 6 of 13 (46%) in the warfarin group, with endoleak-specific reinterventions in 7 and 6, respectively ($P = .03$; Tables II and III). There were five endograft explantations with conversion to open repair, three in the warfarin group and two in the antiplatelet group. All three in the warfarin group were for continued aneurysm sac expansion despite reintervention. The two in the antiplatelet group were for delayed rupture and infection. One patient in the antiplatelet group with a known type II endoleak refused reintervention and died of delayed aneurysm sac rupture.

## DISCUSSION

EVAR has provided an alternative to traditional open repair that is associated with less perioperative risk, decreased postoperative mortality, and shorter hospital stays.[1,14-17] This, however, comes with a tradeoff of a slight increase in postoperative repair failure, increased need for repeat intervention, and the endoluminal-exclusive complication of endoleak after EVAR.[3,18]

Endoleak predisposes to the risk of further aneurysm sac expansion and, potentially, delayed sac rupture.[3] There have been conflicting data on the role of warfarin anticoagulation in the pathogenesis of endoleak formation.[5-7] Iyer et al[7] described the phenomenon of reversible endotension during periods of excessive anticoagulation associated with supratherapeutic INR while patients received warfarin therapy.[7] Others have observed decreased sac contracture after EVAR in those patients requiring warfarin anticoagulation.[6] In this study, we aimed to investigate the role of warfarin in endoleak formation and to monitor aneurysm sac expansion by using 3D volumetric analysis.

We found that warfarin therapy was associated with an increased endoleak incidence after EVAR, and together, these were a major cause of continued AAA sac expansion after EVAR. Interestingly, warfarin appeared to be associated with continued sac expansion regardless of endoleak status, although the greatest sac expansion occurred in the subset of patients who were endoleak-positive and receiving warfarin therapy. Those patients maintained on antiplatelet agents alone continued to have sac shrinkage even if they were endoleak-positive on average. These observations would suggest that warfarin therapy alone may pre-

**Table III.** I. Repeat interventions[a]

| *Leak group* | *Time to leak (days)* | *Leak type* | *Initial size (cm)* | *Device* | *Reinterventions* |
|---|---|---|---|---|---|
| Antiplatelet | | | | | |
| 1 | 9 | I | 7.4 | AneuRx[b] | Extension cuff |
| 2 | 28 | I | 5.1 | Excluder[c] | Extension cuff |
| 3 | 35 | III | 7.0 | AneuRx | Stent graft |
| 4 | 40 | I | 5.2 | Ancure[d] | Extension cuff |
| 5 | 72 | I | 7.0 | AneuRx | Extension cuff |
| 6 | 248 | I | 6.5 | AneuRx | Extension cuff |
| 7 | 1556 | Infected | 5.5 | Ancure | Explanted |
| 8 | 1677 | III | 7.5 | AneuRx | Explanted |
| Warfarin | | | | | |
| 1 | 38 | IV | 5.5 | Zenith[e] | Explanted |
| 2 | 38 | II | 5.3 | Excluder | IMA coiling |
| 3 | 99 | II | 5.5 | Excluder | IMA coiling |
| 4 | 321 | I | 5.5 | AneuRx | Extension cuff |
| 5 | 636 | II | 8.0 | AneuRx | Explanted |
| 6 | 728 | I | 5.9 | AneuRx | Explanted |

*IMA,* Inferior mesenteric artery.

[a]Most reinterventions were for complications of endoleak. Patient 7 was explanted for infected endograft and was not used in the analysis comparing reintervention due to endoleak between the anticoagulation groups. Patient 8 was diagnosed with a type III endoleak at the time of delayed rupture and emergency explantation.

[b]Medtronic, AVE, Santa Rosa, Calif.

[c]W. L. Gore and Assoc, Flagstaff, Ariz.

[d]Guidant Endovascular Solutions, Menlo Park, Calif.

[e]Cook Medical Inc, Bloomington, Ind.

dispose to clinically latent endoleaks by current CT protocols, or may result in persistent sac endotension that results in continued sac expansion.

This persistent sac expansion was not associated with an increased risk for delayed rupture in our study, however. This may be related to the small number of patients or to a more aggressive strategy for dealing with progressing endoleaks in these patients. We did not find a link between a specific EVAR device and persistent endoleak or continued sac expansion.

Despite equal follow-up intervals, the 5-year cumulative risk of endoleak nearly doubled when patients were receiving warfarin therapy. The overall incidence of endoleak in our study was somewhat higher than the incidence in previously published studies, although other groups have reported similar rates, with 3-year reintervention rates as high as 35%.[19] This may be partly due to an aggressive CT screening protocol and a low threshold for reintervention. Warfarin therapy appeared to be associated with de novo late endoleak formation as well as with a higher incidence of early endoleak identification.

Of all endoleaks, type II was the most common. Interestingly, the resolution rate of early type II endoleaks did not appear to be significantly affected (44% warfarin, 47% antiplatelet), although an absolute increase in the numbers of type II endoleak was observed. This observation may be due to anticoagulant-driven failure of initial thrombosis of collaterals or subsequent transient supratherapeutic levels of anticoagulation leading to persistently patent collateral vessels and reversible endotension. These collateral vessels then feed the sac and prevent shrinkage, consistent with the mean increase in sac volumes observed by CTA volumetric analyses seen in our study.

These results suggest patients who require warfarin therapy after EVAR should be closely monitored for endoleak and that they are at risk for both early and late endoleaks. Discontinuation of warfarin therapy, if clinically possible, should be considered. When warfarin therapy cannot be discontinued, patients should undergo monitoring for persistent sac expansion. Alternatively, open repair may be considered for patients who require life-long warfarin anticoagulation to reduce cumulative radiation exposure, subsequent repeat interventions, and the need long-term surveillance.

## CONCLUSIONS

Anticoagulation with warfarin appears to be linked to an increased risk for the development of endoleak after EVAR, specifically, type II endoleaks. Warfarin therapy also contributed to persistent aneurysm sac expansion by volumetric analysis; however, this was not associated with an increased risk of delayed sac rupture in our results. No single risk factor, other than warfarin therapy, could be identified that predisposed to endoleak formation. Our study also suggested that those patients requiring warfarin anticoagulation for other indications are at increased risk for secondary interventions and are at risk for persistent sac expansion. Close monitoring of AAA patients who require warfarin therapy for other indications after EVAR is advised.

Anticoagulation with warfarin in the AAA patients who have undergone EVAR is associated with an increase in the incidence of type II endoleaks, aneurysm sac expansion, and the rate of subsequent reintervention. Further study with larger patient populations is warranted and necessary.

## AUTHOR CONTRIBUTIONS

Conception and design: JB, GT
Analysis and interpretation: JB, GL
Data collection: JB, JH, GT
Writing the article: JB, JH, GT
Critical revision of the article: JH, GT
Final approval of the article: JB, JH, GT
Statistical analysis: JB, GL
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