ELSEVIER

Effect of Anticoagulation and Antiplatelet Therapy on Incidence of Endoleaks and Sac Size Expansions after Endovascular Aneurysm Repair

John B. Wild, Nikesh Dattani, Phillip Stather, Matthew J. Bown, Robert D. Sayers, and Edward Choke, Leicester, United Kingdom

Background: The effects of anticoagulation or antiplatelet therapy on the incidence of endoleak and aneurysm sac size after endovascular aneurysm repair (EVAR) are unclear. This study aims to determine whether these therapies affect the incidence of endoleaks or sac size expansions after EVAR.

Methods: The case notes of 407 patients (367 men and 40 women, mean age 74.7 years) who underwent elective EVAR between January 2006 and November 2011 were reviewed for medication history and EVAR-related outcomes.

Results: The median follow-up period was 18 months. There were 45 (11.1%) patients on warfarin (WA), 292 (71.7%) on antiplatelet therapy (AT) (aspirin, clopidogrel, or dipyridamole modified release), and 70 (17.2%) on no anticoagulation or antiplatelet therapy (NA). During the study period, 51 (12.5%) endoleaks were documented, 8 type I (AT = 6, NA = 0, and WA = 2) and 42 type II (AT = 31, NA = 9, and WA = 2). Medication did not significantly affect the incidence of type I ($P = 0.24$) (based on chi-squared analysis), type II ($P = 0.33$), or type III ($P = 0.82$) endoleaks, or sac expansions ($P = 0.95$).

Conclusions: Warfarin and antiplatelet therapies are not associated with increased incidence of postoperative endoleaks or aneurysm sac expansion after EVAR. The data in this study support safe use of anticoagulant and antiplatelet medications in patients undergoing EVAR.

INTRODUCTION

Endovascular aneurysm repair (EVAR) compares favorably with open abdominal aortic aneurysm (AAA) repair in terms of 30-day mortality and duration of intensive care stay.[1] In comparison to open AAA repair, The benefits of EVAR, such as reduced perioperative time, decreased pain, reduced number of blood transfusions, and decreased length of stay in intensive care units, are in contrast to an increased rate of postoperative intervention.[2] This increased intervention rate is, in part, due to endoleak formation, which can result in significant patient morbidity and mortality.[3] Endoleaks represent the persistence of blood flow within the aneurysm sac, which can lead to an increase in intrasac pressure and potentially result in aneurysm rupture, even after exclusion.[4] Endoleaks are subdivided by type: type I endoleaks arise from proximal graft leakage; type II endoleaks are from a retrograde accessory branch (lumbar, mesenteric, and testicular arteries); type III endoleaks are from bleeding between different portions of a graft; and type IV endoleaks occur through the graft.[3,5]

Vascular Surgery Research Group, University of Leicester, Leicester Royal Infirmary, Leicester, UK.

Correspondence to: John B. Wild, MBChB, Vascular Surgery Research Group, Robert Kilpatrick Clinical Sciences Building, University of Leicester, Leicester Royal Infirmary, Leicester LE2 7LX, UK; E-mail: bw69@le.ac.uk

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Although treatment is required for type I and III endoleaks, type II are often left to observation, with intervention rare.[6] The presence of an endoleak has been associated with postprocedure aneurysm ruptures, particularly with proximal endoleaks.[7] Rupture has also been observed in patients with type II endoleak, although this is much less common.[8] It has been demonstrated that there does not need to be an increase in the diameter of the sac preceding rupture.[9] In contrast, the formation of effective thrombus can lead to the cessation of blood flow within the aneurysm sac, although thrombus formation and adherence is dependent on a multitude of factors. Patients undergoing long-term anticoagulation or antiplatelet therapy may represent a group of patients at increased risk of endoleak formation due to their inability to form effective thrombus. This form of pharmacotherapy is quite common among patients with vascular disease, as both are known to positively modify cardiovascular risk, and hence further investigation into endoleak rates and morbidity for patients undergoing EVAR on anticoagulant or antiplatelet therapy is important.[10]

Previous studies examining the effect of anticoagulation and antiplatelet therapy on the incidence of endoleaks and sac expansions after EVAR have been limited by their small sample size, and have consequently yielded conflicting results.[11–13] This study aims to address this controversy by performing a retrospective analysis of a large cohort of patients undergoing EVAR from a single vascular center.

METHODS

Data Collection

Data were prospectively collected from patients who underwent EVAR at a single institution between January 2006 and November 2011 and were analyzed retrospectively. Collection included data from each consultant with no exclusions. Preoperatively all patients underwent computerized tomography (CT) imaging. Medication details were identified through the electronic medical record, iSOFT Patient Management Centre (version 4.1.1107).

A standardized proforma was used to collect data prospectively, which was a modification of the National Vascular Database AAA proforma (Vascular Society of Great Britain and Ireland). Data on demographics, lifestyle, comorbidity, procedural details, perioperative outcomes, and other variables were all prospectively entered into a database, which was retrieved from the electronic patient database.

Baseline characteristics included age and gender, and lifestyle variables included smoking (past and present). Comorbidities studied included ischemic heart disease (history of angina, any previous myocardial infarct, prior percutaneous coronary intervention, and coronary artery bypass surgery), hypertension, cerebrovascular disease (based on clinical events: previous transient ischemic attack or stroke), atrial fibrillation (AF), deep vein thrombosis or pulmonary embolus (DVT/PE), hypercholesterolemia, and diabetes mellitus (diet- or tablet-controlled or insulin-dependent types).

All patients who undergo EVAR at our center initially receive a postoperative plain abdominal radiograph and duplex ultrasound prior to discharge; this followed by repeat imaging at 1, 3, and 6 months, and then at 6-month intervals thereafter. CT angiography scans were performed as necessary (on all type I and III endoleaks and any type II endoleaks that demonstrated sac expansion) to confirm or clarify complications detected on duplex ultrasound scan surveillance. Sac expansion was defined as a persistent increase of >5 mm of the initial postoperative size at any point during follow-up. A specific protocol is utilized in the duplex surveillance of EVAR patients to ensure homogeneity of measurement. Duplex ultrasonography was performed by technologists accredited by the Society for Vascular Technology (SVT) in pairs to ensure accurate measurement. Data regarding outcomes at follow-up scans are prospectively entered into the database, and were complete for all patients.

The outcome measures were in accordance with the reporting standards for EVAR, and included type I endoleak, type II endoleak, type III endoleak, sac expansion, stent migration, thrombotic events, and reinterventions.

Statistical Analysis

Statistical analysis was performed using SPSS software (PASW Statistics, release version 20.0; SPSS, Inc., Chicago, IL, USA). Analysis between groups was done using the chi-squared test and unpaired analysis of variance (ANOVA) for categorical and continuous variables, respectively. $\alpha \leq 0.05$ was used to indicate statistical significance.

RESULTS

Patients' Demographics

Five hundred twenty-four EVAR grafts were performed at our center over the 5-year study period. Of these, 407 cases (median age 74.7 [range 39.9–91.7] years, 367 men and 40 women, median period