Abstracts

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Rate and Predictability of Graft Rupture After Endovascular and Open Abdominal Aortic Aneurysm Repair: Data From the EVAR Trials

Wyss TR, Brown LC, Powell JT, et al. Ann Surg 2010;252:805-12.

Conclusion: There are no ruptures after open repair for abdominal aortic aneurysm (AAA) and a low rate of rupture after endovascular aneurysm repair (EVAR). Mortality after graft rupture is high. Few ruptures after EVAR are spontaneous, with most having had complications identified during surveillance.

Summary: Aneurysm rupture after EVAR is well recognized, and endograft rupture has high mortality (Schlosser FJV, Eur J Vas Endovasc Surg 2009;37:15-22). Graft-related complications after EVAR are frequent. Certain types of endograft complications (endoleak types I and III, stent graft disintegration and migration) increase the risk of aneurysm rupture (Fransen GA, Eur J Vas Endovasc Surg 2003;26:487-93). The authors analyzed the patients in the United Kingdom (UK) EVAR trials to assess the rate and factors associated with rupture after EVAR or open repair of AAA.

Up to July 2009, 848 elective EVARS and 594 elective open AAA repairs had been performed in the UK EVAR Trials 1 and 2. Patients were followed-up for complications, reinterventions, and rupture. Incidence of rupture was analyzed with respect to baseline anatomy and subsequent complications in a Cox regression analysis model.

No ruptures occurred in patients treated with open repair, but 27 ruptures occurred after EVAR during a mean follow-up of 4.8 years, for a crude rate of 0.7 ruptures/100 person-years (95% CI, 0.5-1.0). Of patients who developed endograft rupture, 67% died ≤30 days of rupture. Five ruptures occurred in the first 30 postoperative days and 22 after that; crude rates of rupture were 7.2 (95% CI, 3.0-17.4) and 0.6/100 person-years (95% CI, 0.4-0.9). Previous complications (endoleak type I, type II with sac expansion, type III, migration or kinking) increased the risk of rupture, with a hazard ratio of 8.83 (95% CI, 3.76-20.76; $P < .0001$).

Comment: EVAR is a form of management of AAA and not a cure for AAA. As such, patients must be continually monitored for complications associated with potential rupture of an endograft-managed AAA. Patients with rupture fall into roughly three groups. Those who have a rupture in the perioperative EVAR period likely reflect inadequate initial isolation of the aneurysm. A small group of patients rupture with reasonable compliance with recommended surveillance. This suggests complications associated with EVAR that occur after a negative surveillance study can rapidly lead to rupture. Most ruptures, however, occur in patients with an identified problem with their endograft. Sac expansion was documented in 15 of 17 in this group. Rupture after EVAR can be minimized by ensuring that before leaving the hospital, the patients have had technically satisfactory procedures and that sac expansion is evaluated, diagnosed, and aggressively treated.

Aspirin for Prevention of Cardiovascular Events in a General Population Screened for a Low Ankle Brachial Index: A Randomized Controlled Trial

Fowkes FG, Price JF, Stewart MCW, and the Aspirin for Asymptomatic Atherosclerosis Trialists. JAMA 2010;303:841-8.

Conclusion: Patients with asymptomatic vascular disease identified by a low ankle-brachial index (ABI) do not have a reduction in vascular events with daily aspirin administration.

Summary: A low ABI is a marker of systemic atherosclerosis and is associated with coexisting cerebral vascular and coronary disease. In men with a low ABI (<0.9), there is a 27% risk over 10 years of a major coronary event vs 9% in those with ABIs between 1.11 and 1.4; for women, the figures are 19% and 9% (Fowkes FG, JAMA 2008;300:197-208). It is controversial whether screening ABIs should be performed in asymptomatic patients because there is minimal evidence for an effective intervention in asymptomatic individuals with a low ABI (US Preventative Services Task Force, AHRQ, Publication No. 05-0583-A-EF. http//www.ahrg.gov/clinic.uspstf05/pad/padrs.htm). The authors sought to determine the effectiveness of aspirin in preventing cardiovascular events in otherwise asymptomatic individuals found to have a low ABI. This was an intention-to-treat, double-blind, randomized, controlled trial conducted between April 1998 and October 2008 involving 28,980 men and women aged 50 to 75 years. All were free of clinically evident vascular disease. Patients wre recruited from a community health registry in Scotland and had ABI screening tests, and 3350 were identified with a low ABI (≤0.095) and entered the trial. Patients were randomized to daily 100 mg enteric-coated aspirin or placebo. The primary end point was a composite end point of an initial fatal or nonfatal coronary event, a revascularization procedure, or stroke. Secondary end points were a composite of all primary end point events plus new intermittent claudication or transient ischemic attack. An additional secondary end point was all cause mortality.

Mean follow-up was 8.2 ± 1.6 years. During follow-up, 357 of the 3350 study participants had a primary end point event (13.5/1,000 person-years; 95% CI, 12.2-15.0). There was no statistical difference between the incidence of the primary end point between groups, consisting of 13.7 events/1000 person years in the aspirin group vs 13.3 in the placebo group (HR, 1.03; 95% CI, 0.84-1.7). The secondary end point of any vascular event occurred in 578 participants (22.8/1,000 person years; 95% CI, 21.0-24.8). There was no statistical difference between the aspirin (22.8 events/1000 person-years) and placebo (22.9 events/1000 person years) groups (HR, 1.0; 95% CI, 0.85-1.17). Similarly there was no difference in all cause mortality between groups (176 vs 186 deaths, respectively; HR, 0.95; 95% CI, 0.77-1.16). Major hemorrhage requiring admission to the hospital occurred in 34 subjects (2.5/1,000 person years in the aspirin group and 1.5/1,000 person years in the placebo group; HR, 1.71; 95% CI, 0.99-2.97).

Comment: The trial suggested ABI screening in individuals free of clinical cardiovascular disease is unlikely to be beneficial if the intervention for a low ABI is prophylactic low-dose aspirin. It does not rule out the possibility that other therapies, such as statins or perhaps different antiplatelet agents with less hemorrhagic risk, might confer benefit. In practical terms, the results indicate that somewhere between 500 and 600 people would need to be screened with ABI studies and treated with aspirin to prevent one major cardiovascular event over 8 years. It seems difficult to justify the resources required for such massive screening with such little return.

Association Between Aneurysm Shoulder Stress and Abdominal Aortic Aneurysm Expansion: A Longitudinal Follow-Up Study

Li Z-Y, Sadat U, U-King-Im J, et al. Circulation 2010;122:1815-22.

Conclusion: High shoulder stress is associated with rapid expansion of abdominal aortic aneurysm (AAA).

Summary: AAA rupture can be seen as an example of structural failure when mechanical stresses acting on a weakened aortic wall exceed local mechanical failure strength. Wall stress may be due to an influence of several concomitant factors, including aneurysm shape, biochemical composition of the aneurysm wall, characteristic and shape of intraluminal thrombus, eccentricity of the aneurysm, and interaction between solid domains and fluid. Changes in wall stresses may be useful in identifying AAA stability. The authors sought to evaluate the association of wall stress and expansion rate of AAAs in a longitudinal follow-up study. The study included 44 patients with an infrarenal AAA monitored in one university vascular center. Axial and 3-dimensional computed tomography reconstruction images were used to assess maximum diameter of the aneurysm and location of the aneurysm shoulder at baseline and just before surgery. The shoulder of the aneurysm was defined as the junction of the neck and the aneurysm sac. Maximum diameter was defined as the maximum distance between the outer walls of the aorta in the aneurysm sac. Distances of the two locations were measured from the renal artery. Patients were divided into two groups: those with stable AAAs, defined as an expansion rate of <0.4 cm/year, and those with rapidly expanding AAAs, defined as an expansion rate of >0.4 cm/year. Structural analysis was performed to calculate wall stresses of AAAs at baseline and follow-up visits. A nonlinear large-strain finite element method was used to compute wall stress distribution.

Slowly and rapidly expanding AAAs had comparable baseline maximum diameters (median 4.35 cm [interquartile range, 4.12-5.0 cm] vs 4.6 cm [interquartile range, 4.2-5.0 cm]; $P = .32$). Rapidly expanding AAAs had higher shoulder stresses than slowly expanding AAAs (median, 300 kPa [interquartile range, 280-320 kPa] vs 225 kPa [interquartile range, 211-249 kPa]; $P = .0001$). There was a significant correlation of 0.71 between baseline shoulder stress and expansion rate ($P = .0001$).

Comment: The Society for Vascular Surgery uses AAA diameter as the primary criteria for determining follow-up intervals for unoperated AAAs. It is known, however, that some small AAAs will rupture unpredictably. Extending aneurysm surveillance to include measurement of shoulder stress as well as aneurysm diameter may eventually allow the determination of which AAAs should be repaired at relatively smaller diameters and which small AAAs are more likely to exhibit more rapid expansion and should therefore be monitored with closer follow-up intervals than currently recommended.