Type II Endoleaks after Endovascular Aneurysm Repair Are Not so Innocuous After All

NOTES

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Type II endoleak is increasingly seen as a benign, frequently self-healing event and its significance remains the subject of conflicting reports. Several anecdotal reports of post-endovascular aneurysm repair (EVAR) ruptures caused by isolated active type II endoleaks justify further scrutiny on this subject. Our aim in this study was to evaluate the impact of isolated type II endoleaks on post-EVAR aneurysm sac diameter evolution.

Pre-and postoperative computed tomography (CT) scans of patients who underwent endovascular abdominal aortic aneurysm repair (EVAR) between November 4, 1997, and May 15, 2003, at the Mayo Clinic were reviewed. Type II endoleak assessment was based on all postoperative contrasted CT and duplex ultrasound scans, supplemented with angiography in selected cases. Patients with type I or III endoleaks were excluded, leaving 198 patients. Maximum aneurysm diameter was evaluated using the minor axis. Changes in aneurysm diameter above or equal to 5 mm were considered significant. The rates of postoperative aneurysm diameter increase and decrease (? 5 mm) were calculated, and the effect of type II endoleaks on these two outcomes was evaluated. The cumulative incidence rates of all postoperative events (aneurysm diameter increase, aneurysm diameter decrease, spontaneous endoleak resolution, and endoleak-related reintervention) were calculated using the Kaplan-Meier method. Impact of different vessels involvement (lumbar artery, inferior mesenteric artery) was also evaluated.

Forty-two patients (21.2%) had a type II endoleak observable at some point during their follow-up period. Thirty-three endoleaks (16.7%) were still observable after the thirtieth-day postoperatively. Direct relation to one or more patent lumbar arteries was established in 10 cases (23.8%), the inferior mesenteric artery was involved in 10 cases (23.8%), and 11 patients had both kinds of vessels open (26.2%). In 11 (26.2%) cases the feeding vessel of the endoleak could not be identified with certainty. Spontaneous endoleak resolution rate was 44.9% at 1 year. There was a trend toward lower rates of resolution in endoleaks involving the IMA (61.5% versus 16.7% at 20 months; p = .054). The overall type II endoleak–related reintervention rate at 2 years was 9.6%.

The overall proportion of patients who experienced aneurysm diameter reduction (? 5mm) was 63.1% at 2 years. The median time to regression was 561 days. Patients with postoperative type II endoleaks had lower rates of aneurysm shrinkage at 1.5 years (17.8% versus 56.2%, p = .017). The presence of a type II endoleak was also associated with higher rates of aneurysm expansion at 1.5 years (14.3% versus 0.6%, p =.011). Among patients with a postoperative II endoleak, aneurysm expansion occurred exclusively in patients with a patent inferior mesenteric artery (14.1% vs 0% at 1 year; p = .052). Aneurysm expansion occurred in four patients, for an estimated probability of expansion of 3.9% (SE = 2.3%) at 2 years. With the exception of one case, all expansion events occurred in patients with a postoperative type II endoleak involving the IMA.

The presence of a postoperative type II endoleak may imply more than the apparently innocuous absence of aneurysm shrinkage. Our study showed an increased risk not only of non-regression among post-EVAR patients with type II endoleaks, but also of aneurysm expansion and more so when the inferior mesenteric artery is the implicated feeding vessel. Such patients should be monitored with an increased degree of suspicion in respect to their aneurysm sac evolution.

References

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