Chapter 9
In Patients with Type 2 Endoleaks Does Intervention Reduce Aneurysm Related Morbidity and Mortality Compared to Observation?

Lisa Kang and Brian Funaki

Abstract Type II endoleaks are the most common complication of endovascular aneurysm repair (EVAR) of abdominal aortic aneurysms (AAA). These endoleaks are the result of retrograde blood flow in arteries arising from the excluded portion of the aneurysm sac. The natural history of untreated type II endoleaks is not fully understood. This is confounded by the fact that imaging classification of type II endoleaks is not always accurate. However, it is clear that a subset of type II endoleaks are associated with aneurysm growth and rupture. Familiarity with the risk factors, prophylactic measures for prevention and imaging methods to identify type II endoleaks is essential for optimal management of this complication.

Keywords Type II endoleak • type 2 endoleak • Abdominal aortic aneurysm • Endovascular aneurysm repair • EVAR • Intervention

Introduction

Endovascular aneurysm repair (EVAR) of abdominal aortic aneurysms (AAA) was first performed by Parodi in 1991 [1]. As a minimally invasive option, EVAR has become the treatment of choice for many with infrarenal AAAs [2]. EVAR has advantages of lower peri-operative morbidity and mortality [3–5]. and comparable long-term survival rates [6]. However, data suggest that EVAR is best performed in patients who are younger than 70 years of age and likely to be compliant with the

L. Kang, MD
Assistant Professor, Department of Radiology, University of Texas Southwestern Medical Center, 5323 Harry Hines Blvd., Dallas, 75390 TX, USA

B. Funaki, MD (✉)
Department of Radiology, University of Chicago, Chicago, IL, USA
e-mail: bfunaki@radiology.bsd.uchicago.edu

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necessary follow-up. This is contrary to a common assumption that EVAR would be best for frail and elderly patients unfit for surgery. In these medically unfit patients, optimization of medical management appears to be the best approach [6, 7].

Endoleaks are characterized by persistent blood flow into the excluded portion of the aneurysm sac after EVAR [8, 9]. They complicate 3–44 % of EVAR for AAAs [9–14] and are categorized into 5 types. Type II endoleaks are the most common type [2, 9, 15] and result from retrograde blood flow in arteries which arise from the aneurysm sac. Most commonly, type II endoleaks occur via the inferior mesenteric artery (IMA) and lumbar arteries [2, 12, 14–16]. The internal iliac, sacral, gonadal and accessory renal arteries area less common culprits [17].

Recommendations for management of type II endoleaks have varied widely, ranging from an aggressive approach with intervention on all [16, 18] to labeling these endoleaks "benign" as a group and warranting no intervention [19]. Currently, the consensus is that type II endoleaks are a heterogeneous and exist along a spectrum of clinical significance [20–22].

Search Strategy

A search of the English literature was used to identify published data on type II endoleaks after EVAR of AAAs in human subjects using the PICO outline (Table 9.1). Pubmed and Cochrane Evidence Based Medicine databases were queried. Terms used in this search were "type 2 endoleak" OR "type II endoleak" AND "abdominal aortic aneurysm" Articles were excluded if they did not specifically address type 2 endoleaks after EVAR of AAAs. Furthermore, these articles were analyzed only if their main subject matter consisted of outcome measures related to strategies for prophylaxis of type II endoleaks or management of type II endoleaks. In regards to prophylaxis of type II endoleaks, 8 cohort studies, 2 case control studies, 11 case series and 1 review article were identified for analysis. 10 cohort studies, 2 case control studies, 15 case series, 4 case reports, 3 meta-analyses and 1 review article pertaining to management of type II endoleaks diagnosed after EVAR were included. The search for literature addressing treatment of type II endoleaks also yielded several cohort studies and numerous case reports, although these were not analyzed in depth. Upon review of the references of the included articles identified via the search, an additional 8 cohorts, 2 case control studies, 6

Table 9.1 PICO table for intervention for type II endoleak

P (patients)	I (intervention)	C (comparator group)	O (outcomes measured)
Patients with abdominal aortic aneurysms status post EVAR with type II endoleaks	Elimination of collateral blood flow supplying aneurysm sacs (ex. embolization)	Imaging surveillance only	Morbidity and mortality without and with intervention

case series and 2 reviews were identified and included. Data were classified using the GRADE system. Additional articles cited were for historic and background information.

Results

Clinical Relevance of Type II Endoleaks After EVAR

Published data describe variable outcomes in patients with type II endoleaks, and the natural history remains uncertain [23]. Spontaneous resolution is seen in many type II endoleaks by 6 months after EVAR, with reported resolution rates ranging from 33 to 80 % [11, 21, 24–27]. and most reports show resolution rates >60 %. These endoleaks are considered transient type II endoleaks. Persistent endoleaks, defined as those which remain after 6 months, are much less likely to resolve spontaneously, with reported incidence of predominantly <10 % [21, 25, 28]. Persistent type II endoleaks are associated with increased morbidity including conversion to open repair but not with increased mortality [21]. Earlier reports failed to demonstrate significant association between type II endoleaks and aneurysm rupture [19, 29, 30], probably due to viewing all type II endoleaks as a uniform group. However, the risk for aneurysm sac expansion and rupture is now well documented [21, 22, 26, 30–37]. When all type II endoleaks are considered, sac expansion occurs in 4–35 % [26, 28, 38, 39] and the risk of aneurysm rupture is 1 % or less [40, 41]. When only persistent endoleaks are considered, the risks are greater, with sac expansion occurring in 14–41 % [27, 36, 38, 42, 43] and aneurysm rupture occurring in up to 24 % [44].

Risk and Prevention Strategies

Various clinical factors have been associated with type II endoleaks, some of which are also associated with failure of aneurysm sac regression. These include increased age, hypertension and antithrombotic therapy [45–47]. Smoking and decreased ankle-brachial index are negatively associated with type II endoleak [48].

Morphologic risk factors predictive of type II endoleak include the presence of patent arteries arising from the aneurysm [12, 17, 26, 37, 49–53], a relative lack of mural thrombus within the aneurysm [26, 52, 54–57], and longer aneurysm neck length [58]. Fan et al. showed that 0–3 patent lumbar arteries was associated with a 13 % type II endoleak rate while ≥6 patent lumbar arteries was associated with a 50 % type II endoleak rate [12]. More recently, Brountzos et al. showed that the risk of persistent type II endoleak was increased by a factor of 12 in the presence of a patent IMA and further increased about four to six times by each additional patent branch arising from the aneurysm sac. A minority have shown no correlation between patency of branch vessels and development of type II endoleak [37].

During EVAR, vessels arising from the aneurysm sac and the sac itself may be embolized in attempt to prevent the type II endoleaks from occurring. Prophylactic embolization of the IMA and lumbar arteries is technically feasible with short-term success rates ranging from 83 to 100 % [42, 59–61]. However, the efficacy of these procedures is debated. Alerci et al. reported a significant decreased incidence of type II endoleak in patients who underwent collateral artery occlusion (3.6 %) during EVAR compared to those who did not (47.8 %) in a long-term study [62]. Gould et al. reported no change in the incidence of type II endoleak with prophylactic embolization of AAA branches [43]. However, not all branches were embolized in this study and aneurysm sac enlargement was observed only in the nonembolized group.

An alternative approach to endoleak prophylaxis is to induce thrombosis of the excluded aneurysm sac at the time of EVAR. Early attempts of direct sac embolization successfully prevented type II endoleaks at the expense of increased morbidity and mortality [63]. Subsequently, safe and effective methods of direct sac embolization have been demonstrated [64–67]. Zanchetta et al. reported a low incidence of type II endoleak and a high percentage of stable or decreasing aneurysm size (97 %) following injection of thrombin into the excluded sac at the time of EVAR [64]. Additionally, sac embolization may reduce health care costs relative to EVAR alone [65].

Identification

Multiphase CT with unenhanced, arterial phase contrast-enhanced and delayed images is the primary imaging test used to evaluate for endoleaks after EVAR [11, 20, 38, 68]. Although follow-up protocols vary, CT is frequently performed in the immediate postoperative period, at 6 months, at 12 months and then annually after EVAR [11, 21, 24, 29, 41]. Imaging surveillance is generally lifelong as new endoleaks may develop over time and late sac expansion and rupture can occur [6, 69–72].

Although multiphase CT is the current standard for diagnosis and evaluation of endoleaks, it is not immune to error. In one series, 36 % of type I and type III endoleaks were misclassified as type II endoleaks on CT with recognition on diagnostic angiography or follow-up CT after IMA embolization [73]. In another series, concomitant type I or type III endoleaks which were not identified on CT were observed in 21 % of patients undergoing angiographic evaluation of type II endoleaks [74]. It is important to recognize that all of these misclassifications occurred in patients with aneurysm growth. Therefore, it is clear that a subset of type II endoleaks which are purportedly associated with aneurysm growth actually represent misclassified type I or III endoleaks.

Some advocate using sonography as the first line imaging modality for EVAR follow-up, reserving CT for instances when sonography is equivocal or demonstrates aneurysm growth [28, 41, 74–76]. Gray et al. have adopted a protocol using duplex sonography performed following 6 h of fasting and supplemented by radiography to evaluate for structural abnormalities of the endograft [77]. This group reports sensitivity of 100 % and specificity of 85 % of duplex sonography for detection of endoleaks.

Contrast enhanced ultrasound and contrast enhanced MR angiography appear to have equivalent if not increased sensitivity relative to traditional CT for detection

of endoleaks [78–80]. Gadofoveset, an intravascular gadolinium based contrast agent which binds to serum albumin *in vivo*, may have special utility in evaluation of endoleaks, allowing detection of low-flow type II endoleaks which are below the detection threshold of CTA and may account for some endoleaks classified as type V [79].

Further characterization of endoleaks may be the key in optimizing treatment. Several novel imaging techniques have shown potential utility in evaluating endoleaks. Measurement of endoleak cavity volume (ECV), the enhancing portion of the excluded aneurysm sac, is possible with post-processing of CT images. Increased ECV on delayed CT images is associated with aneurysm enlargement [81]. Four-dimensional dynamic volumetric CT angiography involves rapid axial imaging of a volume of tissue using a modern scanner with a high number of detector rows. Multiple images are obtained over a short interval following contrast injection, producing three-dimensional angiographic images and better demonstrating the anatomy and physiology of the endoleak [82].

Management: Imaging Surveillance Versus Intervention

When a type II endoleak is identified within the first 6 months after EVAR, conservative management is generally indicated as the majority will be transient [11, 20, 83]. Additionally, the vast majority of asymptomatic type II endoleaks with stable or regressing aneurysm sacs do not result in aneurysm rupture. These patients are also generally managed conservatively with ongoing imaging surveillance [20, 27, 69].

Regardless of when a type II endoleak is diagnosed, most agree intervention is warranted if the aneurysm is symptomatic or if there is growth of the excluded sac [17, 48, 83]. Published criteria for significant aneurysm growth vary, with 5 mm used most commonly and proposed threshold size changes ranging from 5 to 10 mm [6, 9, 14, 21, 26, 48, 83–86]. Smaller apparent changes in sac size may reflect the imprecision of CT (and especially ultrasound) measurements rather than true growth [87]. Other triggers for intervention on type II endoleaks include total sac diameter >5.5 cm >6 months after EVAR [22], the presence of persistent endoleak at 6–12 months [17], and sac pressures >20 % of systolic pressure [17]. Some investigators have proposed using measurements of the endoleak cavity, defined as the enhancing portion of the excluded aneurysm sac on CT, to guide the decision to intervene. However, this is not yet widely used in clinical practice.

Interventions

The two primary approaches in the treatment of type II endoleaks are transarterial embolization and translumbar puncture and embolization of the aneurysm sac [9, 21]. Other less common minimally invasive approaches have been described [88–90] and laparoscopic and open surgical techniques are employed by some [91, 92].

Minimally invasive treatment of type II endoleaks is safe, with most published series reporting mortality of <1 % [22, 71, 73, 74]. However, meticulous technique is required as there is potential for significant morbidity, most frequently due to non-target embolization which may result in ischemic lumbar or sciatic neuropathy, colonic necrosis and pulmonary embolus [6, 93–96]. When the transarterial approach is used, the branch artery which is the site of endoleak should be embolized at its origin from the sac to minimize the risk of ischemic complications [17, 41].

Published rates of success, defined as a post-procedural decrease in aneurysm size, vary widely for the transarterial approach and less so for the translumbar approach ranging from 9 to 100 % and 67 to 100 %, respectively [10, 26, 36, 41, 49, 97–100]. Variability in success of transarterial endoleak embolization appears to be largely due to endoleak recurrence and may also relate to technical difficulty of the procedure as all patent side branches may need to be embolized to achieve success [17, 101, 102]. Notably, up to 80 % of cases which initially appear to be technically successful are complicated by recurrent endoleak [10, 49, 80, 98]. Better results with transarterial embolization have been documented when the type II endoleak originates from the IMA compared to lumbar artery endoleaks [26]. As a sole means of management, the transarterial approach often fails to yield satisfactory results [16, 68].

Data showing inadequacy of the transarterial approach alone have resulted in some considering translumbar sac embolization to be superior. Baum et al. describe the excluded aneurysm sac as being analagous to the nidus of an arteriovenous malformation, dynamically recruiting collateral arteries in communication with the excluded aneurysm sac [98]. Therefore, direct translumbar sac puncture and embolization was this group's therapy of choice. However, despite better overall results with the translumbar approach, transarterial embolization of branch arteries may still be beneficial as a measure to prevent non-target embolization when treating the sac with a liquid embolization medium [103].

It is worth noting that the clinical significance of recurrent endoleak is not fully understood and that technical failure (i.e. recurrence of endoleak) may coexist with clinical success (i.e. stabilization or decrease in size of the sac) [40, 73]. Additionally, regression of the sac size may not be required for technical success with some series reporting a decreased rate of rupture following intervention despite a lack of decrease in aneurysm size [40, 58].

Currently, a combination strategy employing embolization of patent arteries arising from the sac and direct embolization of the excluded aneurysm sac appears to be the best approach [6, 68, 73]. As more information becomes available regarding the natural history of type II endoleaks without and with intervention, this treatment approach may be modified.

Successful treatment of a type II endoleak does not obviate the need for continued imaging surveillance, as success rates appear to diminish over time [6, 72]. One series reported that within 5 years of a successful embolization 20 % of patients required another procedure, 38 % exhibited aneurysm sac growth and 8 % required explant and open repair [6]. Delayed endoleaks, detected >1 year after EVAR, were

the most frequent type observed during a longitudinal study with a mean follow-up period of 53 months. Despite initial aneurysm sac shrinkage after EVAR, these delayed endoleaks were associated with subsequent aneurysm sac enlargement [72].

Recommendations

Most type II endoleaks do not need to be treated but should undergo careful surveillance using CTA. Conventional angiography with possible embolization should be performed for sac enlargement or if there is suspicion of pinhole type III endoleak.

A Personal View of the Data

Although published data suggest prophylactic embolization of the excluded aneurysm sac and side branches are reasonable in the prevention of type II endoleaks, these practices are not routinely performed at our institution.

After EVAR, imaging surveillance is necessary. We perform multiphase CT at 1 month, 6 months and 12 months after EVAR. In the absence of evidence of complication, patients are imaged annually thereafter. An increasing trend toward color duplex sonography for EVAR surveillance is recognized. However, sonography is highly operator dependent, limiting routine utilization as the first line imaging modality.

Most type II endoleaks detected within the first 6 months after EVAR resolve spontaneously. No intervention is recommended during this interval in the absence of symptoms or significant sac expansion.

At our institution, we do not intervene upon type II endoleaks which are asymptomatic and are not associated with sac expansion, regardless of persistence. However, if an endoleak is symptomatic or associated with sac expansion (i.e. an increase of >5 mm from pre-EVAR measurements or >5 mm over an interval of 6 months following an initial decrease in aneurysm size) intervention is warranted.

When intervention is indicated based on CT findings, we use a staged approach. Diagnostic angiography is performed initially. Transarterial coil embolization of the IMA is performed if this vessel is shown to a patent and a contributor to the endoleak. Triple phase CT imaging is then repeated in 1 month. If persistent sac perfusion is identified and a type II endoleak is excluded, transarterial embolization of communicating arteries, including the lumbar arteries or accessory renal arteries, is performed. Coils and/or liquid embolic agents are used. If negative, CT guided translumbar sac embolization with a liquid embolic is performed. After another month, diagnostic imaging is again performed. If continued perfusion of the sac is identified, translumbar sac embolization with a liquid embolic is performed. This approach has resulted in a 100 % clinical success rate [73] with a mean follow-up of 27.5 months.

We continue imaging surveillance on a lifelong basis in EVAR patients who have undergone successful endoleak management to detect late recurrence and sac expansion.

Recommendations

- Intervention in patients with type II endoleaks and growing sacs appears to reduce morbidity and mortality compared to observation
- In type II endoleaks with stable aneurysm sacs, we support use of imaging surveillance. No intervention is recommended in this group. (**evidence quality moderate; strong recommendation**)
- Intervention is recommended in patient diagnosed with type II endoleaks with growing aneurysm sacs. (**evidence quality moderate; strong recommendation**)
  - We consider significant aneurysm growth as >5 mm in diameter from the pre-EVAR diameter or >5 mm over an interval of 6 months following an initial decrease in aneurysm size.
  - It is critical to exclude a pinhole Type III endoleak which may be indistinguishable from a Type II endoleak on CT and difficult to visualize on angiography
- For unstable type II endoleaks, a staged approach in treatment is advocated (**evidence quality low; moderate recommendation**)
- Success of intervention should be measured by clinical success (i.e. aneurysm sac shrinkage or stabilization) rather than technical success (absence of endoleak).

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