# How to differentiate benign from malignant endoleak

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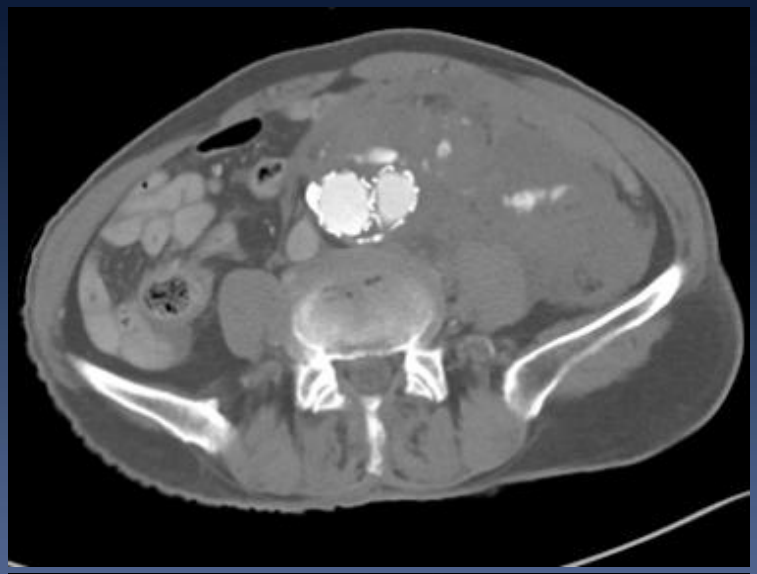
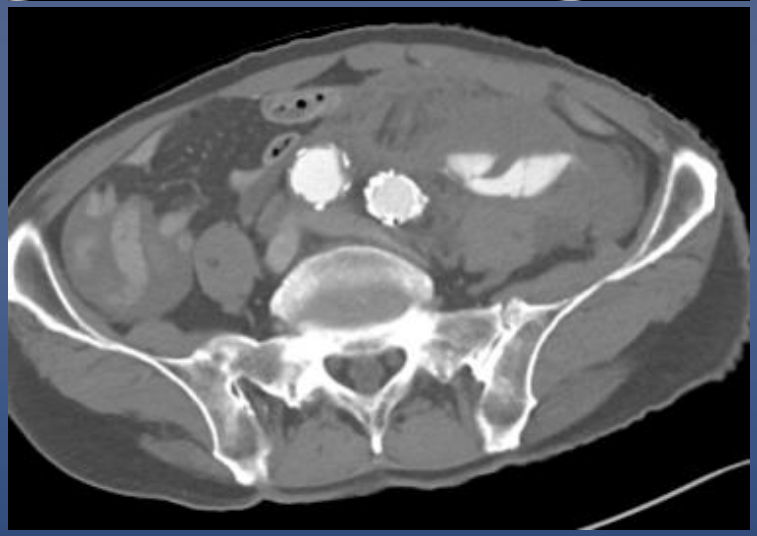
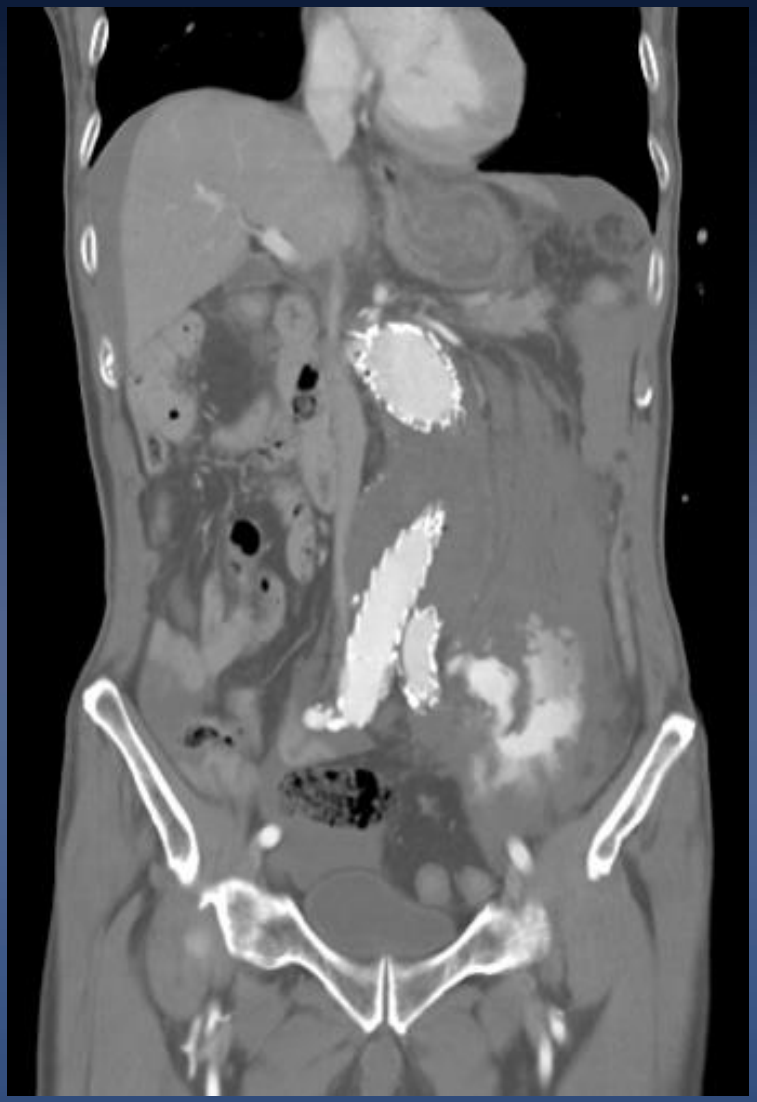
# M76,  follow-up loss (Post-EVAR 18 months)







Eur J Vasc Endovasc Surg (2009) **37**, 15—22

# Aneurysm Rupture after EVAR: Can the Ultimate Failure be Predicted?

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270 ruptures post-EVAR
<2-3 years postop.
160 due to endoleak
119 fatal

**KEYWORDS**
Aortic aneurysm;
Abdominal;
Aneurysm;
Ruptured;
Endovascular aortic aneurysm repair;
EVAR;
Prognosis

**Abstract** *Objectives:* To provide insight into the causes and timing of AAA rupture after EVAR.
*Design:* Original data regarding AAA ruptures following EVAR were collected from MEDLINE and EMBASE databases. Data were extracted systematically and patient and procedural characteristics were analyzed.
*Results:* 270 patients with AAA ruptures after EVAR were identified. Causes of rupture included endoleaks (in 160: type IA 57, type IB 31, type II 23, type III 26, type IV 0, endotension 9, unspecified 14), graft migration 41, graft disconnection 11 and infection 6. Most of the described AAA ruptures occurred within 2—3 years after EVAR. Mean initial AAA diameter was relatively large (65 mm). No abnormalities were present in 41 patients during follow-up before rupture. Structural graft failure was described in 96 and a fatal course in 119 patients.
*Conclusions:* Focus of surveillance on the first 2—3 years after EVAR may possibly reduce the AAA rupture rate, especially in patients with increased risk of early rupture (relatively large initial AAA diameter or presence of endoleak or graft migration). Better stent-graft durability and longevity is required to further reduce the AAA rupture risk after EVAR. Complete prevention will however remain challenging since AAA rupture may occur even if no predisposing abnormalities are present.

afssaps
Agence française de sécurité sanitaire des produits de santé

HAS
HAUTE AUTORITÉ DE SANTÉ

## 2009 guidelines of the French National Authority for Health (Haute Autorité de la santé [HAS])

### ÉVALUATION DES ENDOPROTHESES AORTIQUES ABDOMINALES UTILISEES POUR LE TRAITEMENT DES ANEVRISMES DE L'AORTE ABDOMINALE SOUS-RENALE

La surveillance du patient est obligatoire à long terme. En son absence le traitement ne peut pas être considéré comme complet. Cette surveillance est sous la responsabilité de l'implanteur selon un calendrier précis (cf. annexe I) dont le patient aura été informé (cf. annexe II).

**Without surveillance, treatment is incomplete!!**

# Significance of endoleaks after endovascular repair of abdominal aortic aneurysms: The EUROSTAR experience

**Corine van Marrewijk, MSc, Jacob Buth, MD, Peter L. Harris, MD, Lars Norgren, MD, André Nevelsteen, MD,** and **Michael G. Wyatt, MD,** *Eindhoven, The Netherlands*

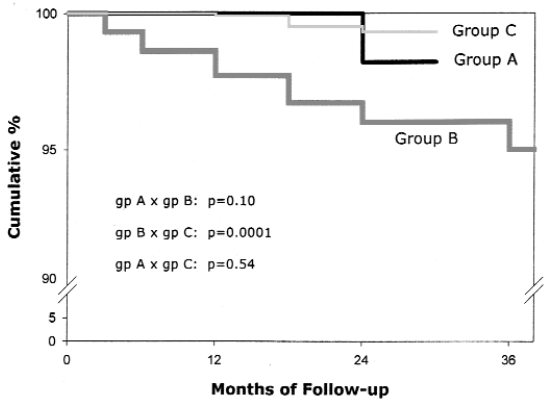
***Objective:*** **The purpose of this study was to assess the incidence, risk factors, and consequences of endoleaks after endovascular repair of abdominal aortic aneurysm.**

***Methods:*** **Data on 2463 patients were collected from 87 European centers and recorded in a cen data were compared for patients with collateral retrograde perfusion (type II) endoleak (grou related (type I and III) endoleaks (group B), and patients in whom no endoleak was detected ( observed after the first postoperative month of follow-up were taken into consideration. Re formed to investigate statistical relationships between the occurrence and type of endoleak an morphologic characteristics, operative details, type of device, and experience of the operating erative changes in aneurysmal morphology, the need for secondary interventions, conversion mal rupture, and mortality during follow-up were compared between these study groups.

***Results:*** **Patients in group A had a higher prevalence of a patent inferior mesenteric artery con out endoleak. Patients in group B were treated more frequently than patients in group C b experience of less than 30 procedures. The mean follow-up period was 15.4 months. Seco needed in 13% of the patients. Rupture of the aneurysm during follow-up occurred in 0.5 3.37% (10/297) in group B, and 0.25% (5/1975) in group C. Life table analysis compari demonstrated a significantly higher rate of rupture in group B than in group C ($P$ = .002). sion to open repair during follow-up was higher in group B than in the other two study gro related to the aneurysm or to endovascular repair of the aneurysm in 7% of patients. Se defined as absence of rupture and conversion, was significantly higher in group A and C con B ($P$ = .006 and $P$ = .0001, respectively).

***Conclusions:*** **The presence of device-related endoleaks correlated with a higher risk of aneury sion compared with patients without type I or III endoleaks. Type II endoleak was not associa events. Consequently, intervention in type II endoleak should only be performed in case of**

**(J Vasc Surg 2002;35:461-73.)**




| | 0 | 12 | 24 | 36 |
|---|---|---|---|---|
| **Group A** | | | | |
| Cumulative % | 100 | - | 98.2 | - |
| No at risk | 191 | - | 55 | - |
| No with rupture | 0 | - | 1 | - |
| **Group B** | | | | |
| Cumulative % | 100 | 97.7 | 96.0 | 95.0 |
| No at risk | 297 | 231 | 153 | 89 |
| No with rupture | 0 | 6 | 9 | 10 |
| **Group C** | | | | |
| Cumulative % | 100 | 99.9 | 99.3 | - |
| No at risk | 1975 | 1175 | 548 | - |
| No with rupture | 0 | 1 | 5 | - |

**Fig 4.** Freedom from aneurysm rupture in group A, group B, and group C.

# Type 2 Endoleak (T2EL)

- Incidence: overall 10 – 45%
  - 15-20% @1m
  - 8-12% @ 1y
- Collateral endoleak from lumbar arteries, IMA etc. (afferent and efferent collateral flow into nidus)
- Association with:
  - Age, gender
  - Diabetes, hypertension, hyperlipidemia, CRF…
  - Anticoagulation, antiplatelets
  - No. and size of branch vessels

JVIR 2017 H. Yu et al.

JVIR 2009 Stravopoulos

# Natural history of T2EL

**T2EL**

**Transient**

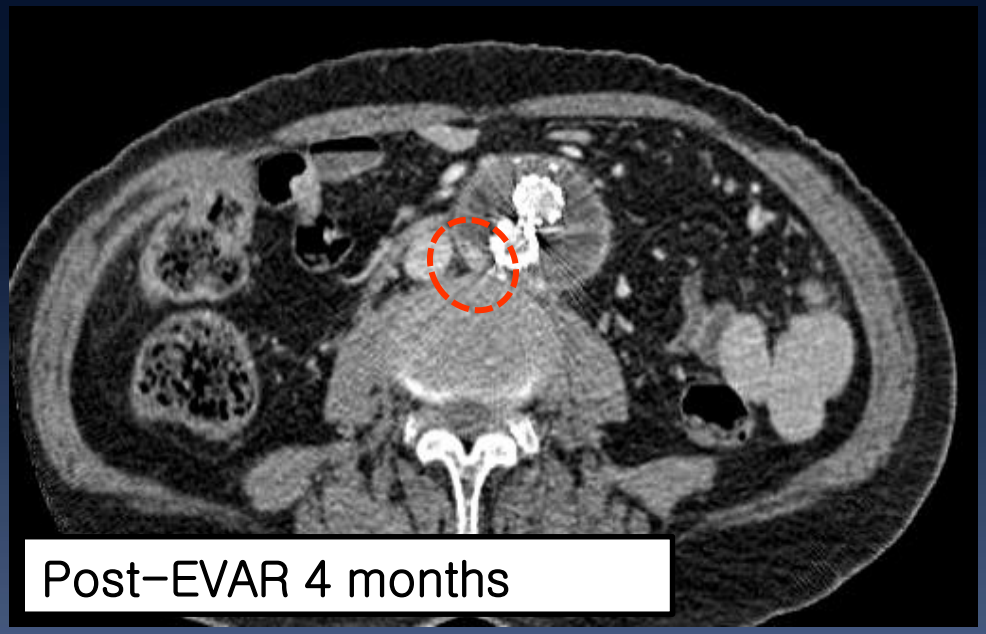
**<6 months**
**Resolves in 40-58%**
**(60% within 1 month)**

**Persistent**

**>6 months**
**Resolution rare**
**Sac enlargement in 5-6%**
**Rupture in 0.5%**

J Vasc Surg 2002;35:461
J Vasc Surg 2016;63:895-901
J Vasc Surg 2017;44:94-102

# F75, T2EL




# An 8-year experience with type II endoleaks: Natural history suggests selective intervention is a safe approach

**Daniel Silverberg, MD, Donald T. Baril, MD, Sharif H. Ellozy, MD, Alfio Carroccio, MD, Savannah E. Greyrose, MS, Robert A. Lookstein, MD,** and **Michael L. Marin, MD,** *New York, NY*

*Objective:* The treatment of type II endoleaks remains controversial because little is known about their long-term natural history and impact on changes in aneurysm morphology. This study reviews type II endoleaks occurring in patients after endovascular abdominal aortic aneurysm repair (EVAR) at a single-institution over an 8-year period.

*Methods:* All patients undergoing EVAR who had type II e
institution between January 1997 and March 2005 were rev
aneurysm size, device type, operative complications, and se
included the rate of spontaneous sealing, freedom from seco
conversion.

*Results:* Type II endoleaks were present in 154 of 965 patient
months (range, 1 to 72 months). Fifty-five patients (35.7%)
of 14.5 months. According to Kaplan-Meier analysis, approximately 75% of type II endoleaks sealed spontaneously within a 5-year period. Nineteen patients (12.3%) with type II endoleaks were treated at a mean time of 19.9 months at the operating surgeon's discretion, including 13 with sac enlargement >5 mm. Kaplan-Meier analysis estimated that approximately 65% of the patients remained free of intervention after a period of 4 years. Thirteen patients (8.4%) experienced aneurysm sac enlargement >5 mm. Kaplan-Meier analysis estimated that approximately 80% of patients with type II endoleaks remained free of sac enlargement >5 mm over a 4-year period. No patients with type II endoleaks experienced rupture or required conversion to open repair during their follow-up. Cox regression analysis showed that cancer, coronary artery disease, and chronic obstructive pulmonary disease were associated with earlier spontaneous closure of the type II endoleaks ($P < .05$).

*Conclusions:* We observed that type II endoleaks have a relatively benign course, and in the absence of sac expansion, can be followed for a prolonged course of time without the need for intervention. The rate of spontaneous seal continues to increase with time and, therefore, close follow-up of patients with type II endoleaks who show no signs of aneurysm expansion is a safe approach. For patients in whom the exact etiology of their endoleak is in question, dynamic imaging should be used to exclude the presence of a type I endoleak. (J Vasc Surg 2006;44:453-59.)

- 154/965 (16%) patients with T2EL
- Spontaneous resolution in 5Y is 75%
- Only 8.4% enlargement >5mm
- No ruptures

# Editor's Choice — Type II Endoleak: Conservative Management Is a Safe Strategy CME

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[b] NIHR Leicester Cardiovascular Biomedical Research Unit, University of Leicester, Leicester, UK

**WHAT THIS PAPER ADDS**

This study suggests that patients with isolated type II endoleak demonstrate equivalent aneurysm-related mortality and an improved all-cause survival. A conservative approach to the treatment of type II endoleak appears to be safe.

**Objective:** Type II endoleak is the most common complication after endovascula
repair (EVAR); however, its natural history is unclear. The aim of this study was
outcomes of type II endoleak, at a single institution after EVAR.
**Methods:** A total of 904 consecutive patients who underwent EVAR between Sep
single centre were entered onto a prospective database. All patients were follo
(DUSS). Patients who developed type II endoleak were compared for preoperativ
sac expansion.
**Results:** A total of 175(19%) patients developed type II endoleak over a median follow-up of 3.6 years (1.5—5.9 years); 54% of type II endoleaks spontaneously resolved within 6 months (0.25—1.2 years). No difference was found in preoperative demographics or choice of endograft between the two groups. Survival was significantly higher in the group with type II endoleak (94.1% vs. 85.6%; $p = .01$) and this effect was most pronounced in those with late type II endoleaks (97.7% vs. 85.6% $p = .004$). No difference was seen in aneurysm-related mortality or rate of type I endoleak between the two groups. Freedom from sac expansion (>5 mm from preoperative diameter) was significantly lower in the group of patients with type II endoleak (82.5% vs. 93.2%, $p = .0001$); however, at a threshold of >10 mm from preoperative diameter no difference was seen.
**Conclusions:** Patients with isolated type II endoleak demonstrate equivalent aneurysm-related mortality and an improved survival.



Article history: Received 17 February 2014, Accepted 3 June 2014, Available online 17 July 2014
**Keywords:** Endoleak, Type II, EVAR, Aneurysm, AAA

- 175/904 (19%) with T2EL
- 54% spontaneous resolution <6m
- Freedom from sac expansion lower in T2EL group than control

# Type II endoleaks after endovascular repair of abdominal aortic aneurysm are not always a benign condition

**Salma El Batti, MD,[a] Frédéric Cochennec, MD,[a] Françoise Roudot-Thoraval, MD,[b] and Jean-Pierre Becquemin, MD,[a]** *Créteil, France*

***Objective:*** **The aim of the study was to determine whether type II endoleak (T2E) after endovascular repair of abdominal aorta (EVAR) is a benign condition (ie, not associated with growth, reintervention, rupture, or death).**
***Methods:*** **Data from patients who underwent EVAR for atherosclerotic infrarenal aortic aneurysms between June 1995 and May 2010 in the Vascular Surgery Department of Henri Mondor Hospital were prospectively collected. Data from patients presenting with at least one T2E on computed tomography scan during their follow-up were compared with those with no T2E. Three subcategories of T2E were studied according to time of occurrence (early or late), persistence (persistent or transient), and recurrence (recurrent or not recurrent).**
***Results:*** **Seven hundred patients were included with follow-up ranging from 1 month to 15 years (median, 31.3 months; range, 12.4-61.4); 201 (28.9%) had at least one T2E. Patients with T2Es were significantly older ($P < .001$), female ($P = .015$), had larger aneurysms ($P = .019$), and patent lumbar arteries ($P = .003$). Patients without T2Es had a higher incidence of current smoking ($P < .001$) and chronic obstructive pulmonary disease ($P < .005$). Multivariate analysis showed risk of T2E was increased in older patients (odds ratio [OR], 1.04; confidence interval [CI], 95% 1.02-1.06; $P < .001$) and in those with patent lumbar arteries (OR, 1.70; CI, 95% 1.16-2.50; $P = .007$), and was reduced in active smokers (OR, 0.16 CI, 95% 0.04-0.71; $P = .015$) or patients with coronary artery disease (OR, 0.65; CI, 95% 0.45-0.92; $P = .016$). Patients with T2Es had more complications (death, rupture, reintervention, or conversion) ($P < .001$) and greater aneurysm sac enlargement (>5 mm upon follow-up) ($P < .001$). Multivariate analysis showed T2E was a risk factor for aneurysm diameter growth >5 mm; this risk wa[illegible] [HR], 3.16; CI, 95% 2.55-6.03; $P < .001$), was recurrent[illegible] a type I or III endoleak (HR, 1.96; CI, 95% 1.41-2.73; [illegible] reintervention ($P = .04$) and conversion to open surgery[illegible]**
***Conclusions:*** **Not all T2Es are benign. Recurrent as well [illegible]**
**(J Vasc Surg 2013;57:1291-8.)**

**Table IV.** Outcomes on patients with or without T2E

| | *T2E* | | *No T2E* | | |
|---|---|---|---|---|---|
| | *n* | *%* | *n* | *%* | P *value* |
| All complications | 92 | 45.8 | 118 | 23.6 | |
| Rupture | 4 | 2 | 5 | 1 | .290 |
| Conversion to open | 12 | 6 | 18 | 3.6 | .210 |
| Endovascular conversion | 7[a] | 3.5[a] | 10 | 2 | .280 |
| Aneurysm sac enlargement | 81 | 40.3 | 84 | 16.8 | <.001 |
| Reintervention[b] | 30 | 14.9 | 33 | 6.6 | .002 |

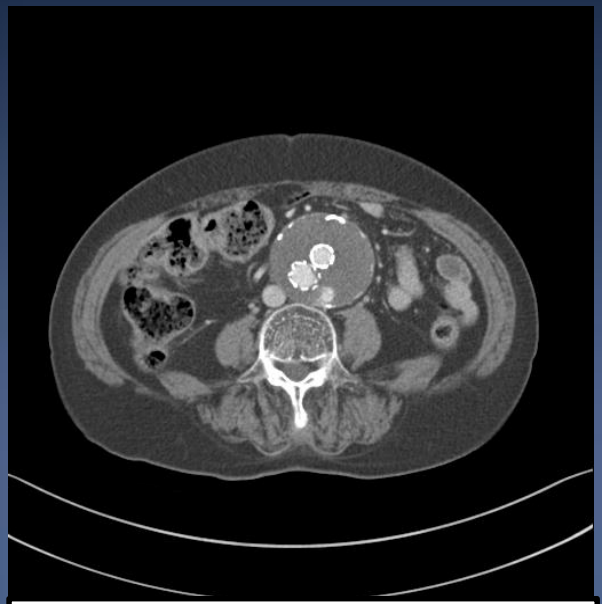
*T2E,* Type II endoleak.
[a]In cases of association of type I and/or III endoleak.
[b]Except for specific treatment of T2E.

**Table VII.** Literature review of incidence and outcomes of T2E

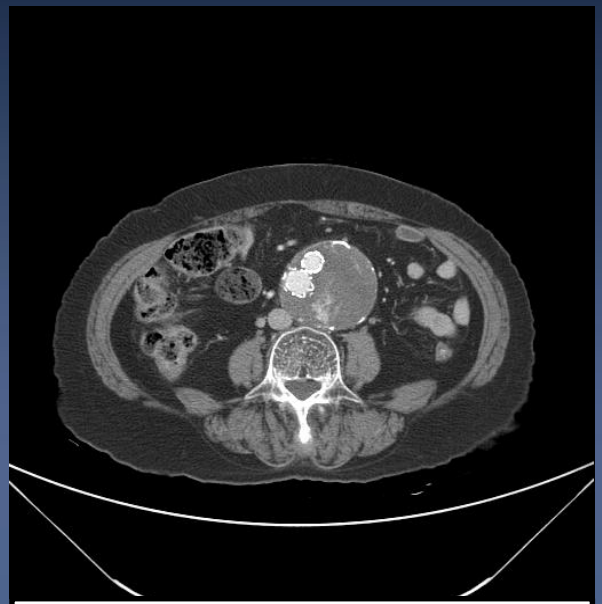
| *Authors* | *Year* | *n* | *Follow-up, months* | *T2E* | *Persistent T2E* | *Sac enlargement* | *T2E reintervention* | *Open conversion* | *Rupture* |
|---|---|---|---|---|---|---|---|---|---|
| | | | | *No. (%)* | | | | | |
| Timaran[11] | 2004 | 348 | 26.5 | 32 (9.2) | 32 (100) | 13 (40.6) | 16 (50) | 0 (0) | 1 (3.1) |
| Jones[15] | 2007 | 873 | 32.6 | 164 (18.8) | 33 (20.1) | 40 (24.4) | 16 (9.8) | 3 (1.8) | 4 (2.4) |
| Abularrage[16] | 2010 | 595 | 34.8 | 136 (22.8) | 89 (65.4) | 73 (53.7) | 39 (28.7) | 1 (0.7) | 2 (1.5) |
| Rayt[17] | 2009 | 369 | 50.4 | 39 (10.5) | 25 (64.1) | 6 (15.4) | 1 (2.6) | 0 (0) | 0 (0) |
| Steinmetz[18] | 2004 | 486 | 21.7 | 90 (18.5) | 35 (38.9) | 5 (5.6) | 5 (5.6) | 0 (0) | 0 (0) |
| Sylverberg[21] | 2006 | 965 | 22 | 154 (15.9) | N/A | 13 (8.4) | 19 (12.3) | 0 (0) | 0 (0) |
| AbuRahma[22] | 2011 | 266 | 22.1 | 56 (21) | 15 (26.8) | 5 (8.9) | 4 (7.1) | 0 (0) | 0 (0) |
| Van Marrewijk[24] | 2003 | 3595 | 15 | 320 (9) | N/A | 71 (22.2) | N/A | 5 (1.6) | 1 (0.3) |
| Our study | 2011 | 700 | 31.3 | 201 (28.7) | 44 (21.9) | 81 (40.3) | 40 (19.9) | 12 (6) | 4 (2) |

*T2E,* Type II endoleak.

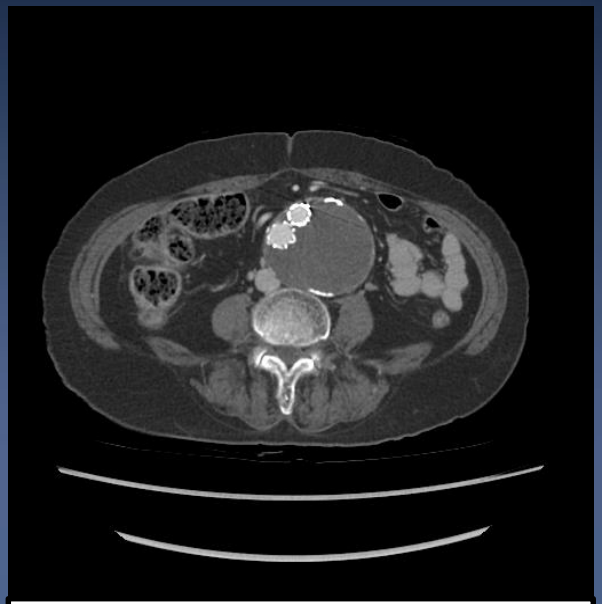
# F71, T2EL



Post−EVAR 12 months



+ 21 months



+ 12 months

Increased sac

# Aneurysm sac expansion is independently associated with late mortality in patients treated with endovascular aneurysm repair

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- Sac increase >5mm/year
- Independently associated with late mortality

## ABSTRACT

**Background:** Patients undergoing endovascular aneurysm repair (EV variations in sac behavior ranging from complete regression to expan 1-year follow-up on late survival.

**Methods:** We used the Vascular Study Group of New England (VSGNE) registry from 2003 to 2011 to identify EVAR patients with 1-year computed tomography follow-up. Aneurysm sac enlargement ≥5 mm (sac expansion) and decrease ≥5 mm (sac regression) were defined per Society for Vascular Surgery guidelines. Predictors of change in sac diameter and impact of sac behavior on long-term mortality were assessed by multivariable methods.

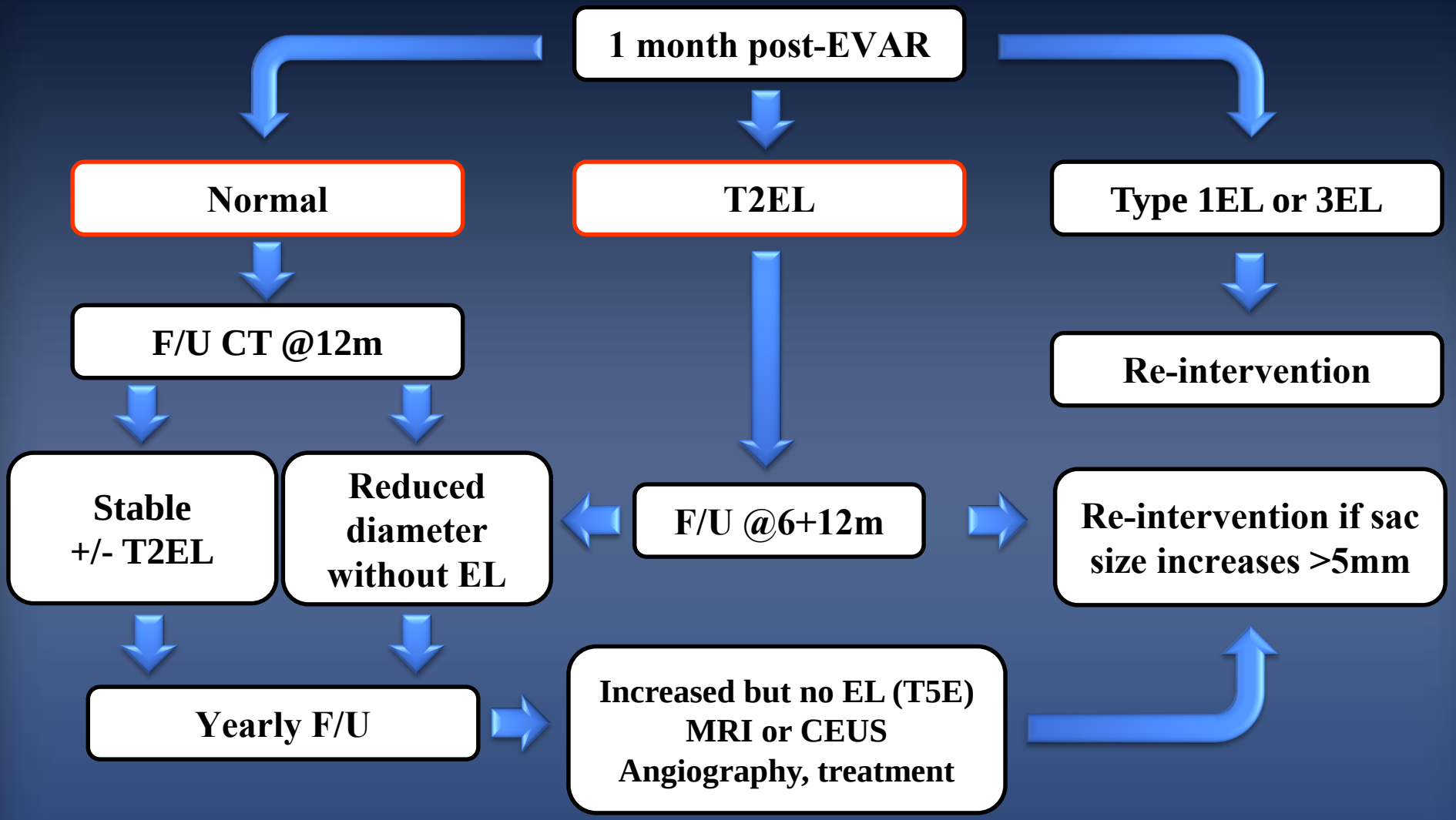
**Results:** Of 2437 patients who underwent EVAR, 1802 (74%) had complete 1-year follow-up data and were included in the study. At 1 year, 162 (9%) experienced sac expansion, 709 (39%) had a stable sac, and 931 (52%) experienced sac regression. Sac expansion was associated with preoperative renal insufficiency (odds ratio [OR], 3.4; 95% confidence interval [CI], 1.5-8.0; $P < .01$), urgent repair (OR, 2.7; 95% CI, 1.4-5.1; $P < .01$), hypogastric coverage (OR, 1.7; 95% CI, 1.1-2.7; $P = .02$), and type I/III (OR, 16.8; 95% CI, 7.3-39.0; $P < .001$) or type II (OR, 2.9; 95% CI, 2.0-4.3; $P < .001$) endoleak at follow-up, and sac expansion was inversely associated with smoking (OR, 0.6; 95% CI, 0.4-0.96; $P = .03$) and baseline aneurysm diameter (OR, 0.7; 95% CI, 0.6-0.9; $P < .001$). Sac regression (vs expansion or stable sac) was associated with female gender (OR, 1.8; 95% CI, 1.4-2.4; $P < .001$) and larger baseline aneurysm diameter (OR, 1.4; 95% CI, 1.2-1.5; $P < .001$) and inversely associated with type I/III (OR, 0.2; 95% CI, 0.1-0.5; $P < .01$) or type II endoleak at follow-up (OR, 0.2; 95% CI, 0.2-0.3; $P < .001$). After risk-adjusted Cox regression, sac expansion was independently associated with late mortality (hazard ratio, 1.5; 95% CI, 1.1-2.0; $P = .01$), even with adjustment for reinterventions and endoleak during follow-up. Sac regression was associated with lower late mortality (hazard ratio, 0.6; 95% CI, 0.5-0.7; $P < .001$). Long-term survival was lower (log-rank, $P < .001$) in patients with sac expansion (98% 1-year and 68% 5-year survival) compared with all others (99% 1-year and 83% 5-year survival).

**Conclusions:** These data suggest that an abdominal aortic aneurysm sac diameter increase of at least 5 mm at 1 year, although infrequent, is independently associated with late mortality regardless of the presence or absence of endoleak and warrants close observation and perhaps early intervention. (J Vasc Surg 2018;67:157-64.)

# Aim of surveillance

- Avoid aneurysm sac rupture
- Monitor sac diameter
- Monitor endoleak
  - Follow-up early endoleak
  - Detect new endoleak
  - Classify type of endoleak

# SFICV (2013) / N. American consensus



# Modality

- **Contrast-enhanced CT**
  - Non-enhanced and delay phase should be included
  - Information of sac, nidus, and surrounding structures
  - Does not show direction, velocity, or pressure

- **Duplex ultrasound**
  - No radiation, no contrast media
  - Comparable sensitivity to CT
  - Operator dependent
  - Does not show status of implanted device
  - Anatomy need to be fonfirmed with CT before re-intervention

# CT: attenuation of nidus

## Imaging-Based Predictors of Persistent Type II Endoleak After Endovascular Abdominal Aortic Aneurysm Repair

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Ichiro Sakamoto[1]
Hiroki Nagayama[1]
Eijun Sueyoshi[1]
Kazuyoshi Tanigawa[2]
Takashi Miura[2]
Masataka Uetani[1]

**OBJECTIVE.** The purpose of this study is to determine the imaging-based parameters associated with the occurrence of persistent type II endoleaks after endovascular abdominal aortic aneurysm repair.

**MATERIALS AND METHODS.** We reviewed the imaging and clinical data for 47 patients with early-onset type II endoleak after endovascular repair. Various predictors of persistent type II endoleaks were analyzed on the basis of preoperative CT findings. In addition, the appearance time of endoleak cavity on the operative angiogram and the relative attenuation of the endoleak cavity in the arterial phase image from the first postoperative CT study were analyzed.

**RESULTS.** The early-onset type II endoleak resolved spontaneously in 22 patients (i.e., the transient group), whereas it was identified on CT studies of the remaining 25 patients 6 months after endovascular repair (i.e., the persistent group). The appearance time of the endo-

**TABLE 3: Results of Univariate and Multivariate Analysis of Imaging-Based Predictors for Persistent Type II Endoleak**

| Variable | Persistent Endoleak | Transient Endoleak | *p* | |
|---|---|---|---|---|
| | | | Univariate Analysis | Multivariate Analysis |
| Abdominal aortic aneurysm size (mm) | 52.8 ± 1.8 | 51.0 ± 1.9 | 0.4996 | |
| No. of patent lumbar arteries | 6.4 ± 0.3 | 6.1 ± 0.4 | 0.5683 | |
| Maximum diameter of the lumbar arteries (mm) | 2.4 ± 0.1 | 2.1 ± 0.1 | 0.05[a] | 0.1121 |
| Patency status of inferior mesenteric artery (no. of arteries) | | | 0.2864 | |
| Occluded | 5 | 2 | | |
| Patent | 20 | 20 | | |
| Area of endoleak ($mm^2$) | 86.3 ± 73.0 | 57.0 ± 64.8 | 0.1366 | |
| No. of patent internal iliac arteries | 1.5 ± 0.14 | 1.6 ± 0.15 | 0.5543 | |
| Relative attenuation of endoleak cavity on first postoperative CT (HU) | 0.70 ± 0.03 | 0.30 ± 0.04 | < 0.0001[a] | 0.0186[a] |
| Appearance time of endoleak cavity on final operative angiogram (s) | 4.7 ± 0.3 | 8.8 ± 0.3 | < 0.0001[a] | 0.0039[a] |

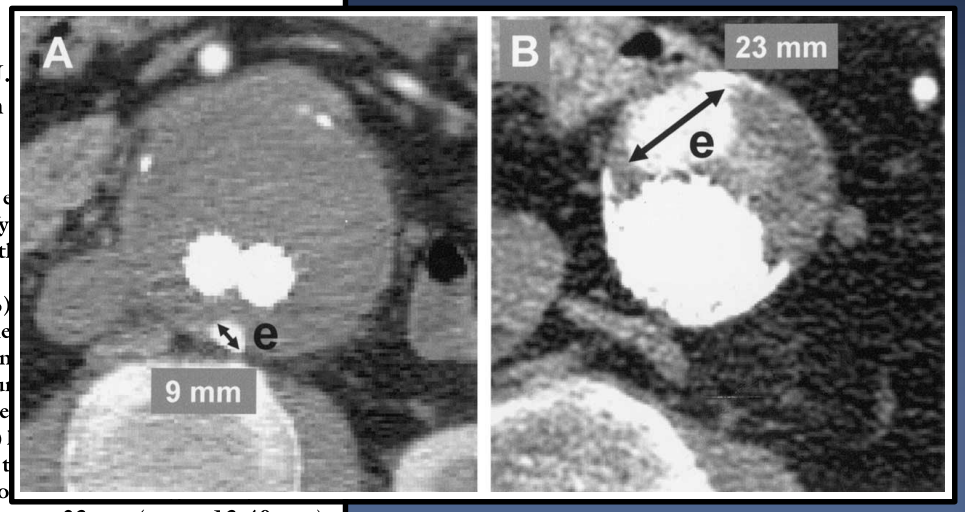
Note—Except where indicated otherwise, data are mean ± SD values.
[a]Statistically significant.

# CT: size of nidus

## Predicting aneurysm enlargement in patients with persistent type II endoleaks

Carlos H. Timaran, MD, Takao Ohki, MD, PhD, Soo J. Rhee, MD, Frank J. Gargiulo III, MD, Hisako Toriumi, MD, Mahmood B. Malas, MD, William Wain, MD, and Evan C. Lipsitz, MD, *New York, NY*

*Objective:* The clinical significance of type II endoleaks is not well understood. Some e some type II endoleaks might result in aneurysm enlargement and rupture. To identify aneurysm expansion, we analyzed the influence of several variables on aneurysm growt II endoleaks after endovascular aortic aneurysm repair (EVAR).
*Methods:* In a series of 348 EVARs performed during a 10-year period, 32 patients (9.2%) persisted for more than 6 months. Variables analyzed included those defined by the (SVS/AAVS) as well as other endoleak characteristics. Univariate, receiver operatin regression analyses were used to determine the association between variables and aneu
*Results:* The median follow-up period was 26.5 months (range, 6-88 months). Thirte enlargement by 5 mm or more (median increase in diameter, 10 mm), whereas 19 (59%) diameter. Univariate and Cox regression analyses identified the maximum diameter of t the nidus as defined on contrast computed tomography scan, as a significant predictor fo risk, 1.12; 95% confidence interval, 1.04-1.19; $P = .001$). The median size of the nidus was 23 mm (range, 13-40 mm) in patients with aneurysm enlargement and 8 mm (range, 5-25 mm) in those without expansion (Mann-Whitney $U$ test, $P < .001$). Moreover, receiver operating characteristic curve and Cox regression analyses showed that a maximum nidus diameter greater than 15 mm was particularly associated with an increased risk of aneurysm enlargement (relative risk, 11.1; 95% confidence interval, 1.4-85.8; $P = .02$). Other risk factors including gender, smoking history, hypertension, need of anticoagulation, aneurysm diameter, type of endograft used, and number or type of collateral vessels were not significant predictors of aneurysm enlargement.
*Conclusions:* In patients with persistent type II endoleaks after EVAR, the maximum diameter of the endoleak cavity or nidus is an important predictor of aneurysm growth and might indicate the need for more aggressive surveillance as well as earlier treatment. (J Vasc Surg 2004;39:1157-62.)



# Late increase in size

**Endovascular AAA Repair: Classification of Aneurysm Sac Volumetric Change Using Spiral Computed Tomographic Angiography**

John G. Pollock, FRCR, Simon J. Travis, FRCR, Simon C. Whitaker, FRCR, more... Show all authors

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Article information

## Abstract

### Purpose:

To classify and analyze the volumetric changes seen on spiral computed tomographic angiography (CTA) following endovascular abdominal aortic aneurysm (AAA) repair.

### Methods:

Fifty patients (46 men; mean age 71 years, range 51–83) with >1 year of imaging follow-up were retrospectively selected. The volume of the aneurysm sac was calculated on standard CT workstations to obtain plots of volume changes over time. For the purpose of this study, a 10% change in sac volume was considered significant.

### Results:

Over a mean 32-month follow-up, 256 CTA scans were [illegible] 259 mL and initial mean AAA diameter was 6.5 cm. Six distinct patterns of volume change were recognized: group Ia (28 patients, 56%): progressive reduction in aneurysm sac volume; group Ib (3 patients, 6%): transient initial increase then same as Ia; group II (4 patients, 8%): no significant change; group IIIa (5 patients, 10%): late increase in volume; group IIIb (8 patients, 16%): progressive increase in volume; and group IV (2 patients, 4%): late reduction in volume after secondary intervention. Group III changes were associated with endoleak types I and III ($p<0.0001$).

### Conclusions:

This classification system of spiral CTA volumetric changes features 6 patterns with recognized clinical significance and predictive value for endoleaks. Group I is the ideal outcome when the aneurysm sac shrinks and often completely disappears, while group III is associated with types I and type III endoleak and should prompt further investigation. Long-term volumetric analysis of all patients is advised.

- Sac reduction: ideal outcome
- Increased volume: associated with T1 and T3EL

# Late onset of endoleak

## Outcome and clinical significance of delayed endoleaks after endovascular aneurysm repair

Wei Zhou, MD,[a,b] Eddie Blay Jr, BA,[c] Vinit Varu, MD,[a] Syed Ali, BS,[d] Michelle Qiushuang J
Lisa Sun, MS,[f] and Jin Hyun Joh, MD, PhD,[g] *Stanford and Palo Alto, Calif; Durham, NC; Vall*
*Sunnyvale, Calif; and Seoul, Korea*

*Objective:* Endovascular aneurysm repair (EVAR) is considered the standard therapy for most patients wi
aortic aneurysm (AAA). Endoleak is a well-known EVAR-related complication that requires long-ter
However, patient follow-up is often challenging outside clinical trials. We sought to evaluate the incidence
of delayed endoleaks in a Veterans Administration (VA) health care system where long-term follow-up is
*Methods:* We retrospectively evaluated 213 consec
tration medical center. Age, aneurysm size, patenc
were recorded. Type of endoleak, date of detectio
follow-up were excluded. The $\chi^2$ test, Student *t*-t
analysis.
*Results:* The analysis included 183 patients with
patients (26%) had endoleaks, and 31 (17%) had a
endoleaks was 45 months (range, 3-127 months),
endoleak received prompt secondary intervention
An isolated type II endoleak was detected in 34 pa
41% of which were detected >1 year after EVAR.
aneurysm size at the latest computed tomography
whereas those with isolated type II endoleak had
preoperative size (5.8 vs 5.7 cm). Importantly,
enlargement (0.8 cm), and delayed type II endolea
endoleaks detected early. No significant correlatio
to AAA enlargement among the patients with a typ
II endoleak resulted in overall aneurysm stabiliza
*Conclusions:* This long-term outcome study de
contributed to most of the overall endoleaks an
underscores that type II endoleak is not benign a
2014;59:915-20.)

**Delayed onset**

- Less likely to resolve
- More likely to grow
- Higher re-intervention rate

## Late type II endoleaks after endovascular aneurysm repair require intervention more frequently than early type II endoleaks

Danielle M. Pineda, MD, Keith D. Calligaro, MD, Sam Tyagi, MD, Douglas A. Troutman, DO, *and* Matthew J. Dougherty, MD, *Philadelphia, Pa*

**ABSTRACT**

**Objective:** Type II endoleaks (T2ELs) are commonly observed after endovascular aneurysm repair (EVAR). We sought to determine whether time at onset of T2ELs correlated with the need to intervene based on sac expansion or rupture.

**Methods:** Between 1998 and 2015, 462 EVARs performed at our institution had duplex ultrasound surveillance in our accredited noninvasive vascular laboratory. Computed tomography and arteriography were reserved for abnormal duplex ultrasound findings. The need for intervention for T2ELs was classified according to time at onset after EVAR. Interventions for T2ELs were performed only for sac expansion >5 mm or rupture. We defined early-onset T2ELs as <1 year after EVAR and delayed or late onset as >1 year of follow-up.

**Results:** Of the 462 EVARs, 96 patients (21%) developed T2ELs after implantation. Of these, 65 (68%) had early and 31 (32%) had late onset (mean, 12 months; range, 1-112 months). Early T2ELs resolved without treatment in 75% (49/65) of cases compared with only 29% (9/31) of late T2ELs ($P < .0001$). Intervention was required for only 8% (5/65) of patients with early T2ELs (5 sac expansions, 0 ruptures) compared with 55% (17/31) for late T2ELs (16 sac expansions, 1 rupture; $P < .0001$). The remaining patients were observed for persistent T2ELs with no sac growth (17% [11/65] early vs 16% [5/31] late; $P = .922$).

**Conclusions:** Less than one-third (29%) of T2ELs that develop after 1 year will resolve spontaneously and about half (55%) will require intervention for sac growth or rupture. T2ELs that develop >1 year after EVAR should be followed up with a more frequent surveillance protocol and perhaps with a lower threshold to intervene. (J Vasc Surg 2018;67:449-52.)

# Sac pressure

## Aneurysm sac pressure measurements after endovascular repair of abdominal aortic aneurysms

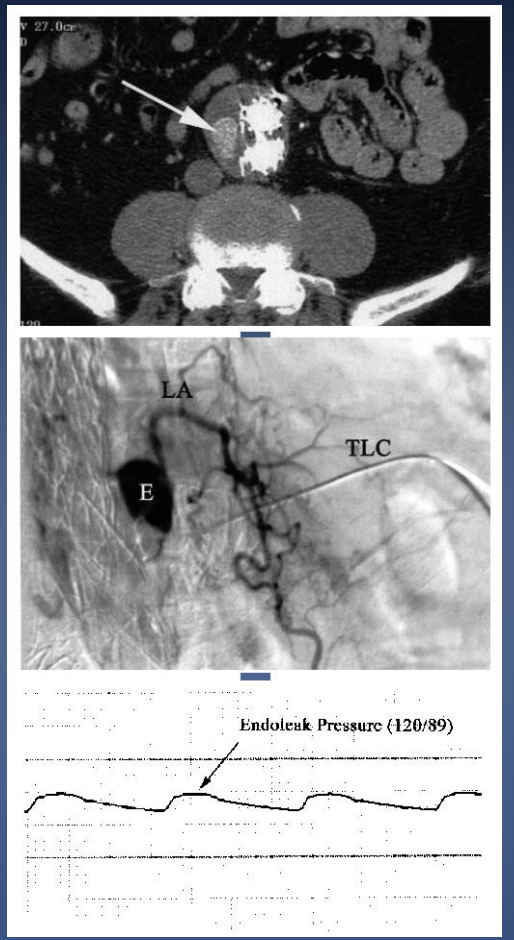
**Richard A. Baum, MD,[a] Jeffrey P. Carpenter, MD,[b] Constantin Cope, MD,[a] Michael A. Golden, MD,[b] Omaida C. Velazquez, MD,[b] David G. Neschis, MD,[b] Marc E. Mitchell, MD,[b] Clyde F. Barker, MD,[b]** and **Ronald M. Fairman, MD,[b]** *Philadelphia, Pa*

*Objectives:* The goal of endovascular grafting of abdominal aortic aneurysms (AAAs) is to exclude the aneurysm sac from systemic pressure and thereby decrease the risk of rupture. Unlike conventional open surgery, branch vessels in the sac (eg, lumbar artery and inferior mesenteric artery [IMA]) are not ligated and can potentially transmit pressure. The purpose of our investigation was to evaluate the feasibility of various interventional techniques for measuring pressure within the aneurysm sac in patients who had undergone endovascular repair of AAAs.

*Methods:* Sac pressure measurements were performed in 21 patients who had undergone stent graft repair of AAAs. Seventeen of 21 patients had endoleaks demonstrated on 30-day computed tomographic (CT) scans. Access to the aneurysm sac in these patients was through direct translumbar sac puncture (5 patients), through a patent IMA accessed via the superior mesenteric artery (SMA) (9 patients), or by direct cannulation around attachment sites (3 patients). Four patients had perioperative pressure measurements obtained through catheters positioned along side of the endovascular graft at the time of its deployment. Two of these catheters were left in position for 30 hours during which time CT and conventional angiography were performed. Pressures were determined with standard arterial-line pressure transduction techniques and compared with systemic pressure in each patient.

*Results:* Elevated sac pressure was found in all patients. The sac pressure in patients with endoleaks was found to be systemic (15 patients) or near systemic (2 patients) and all had pulsatile waveforms. Elevated sac pressures were also found in patients without CT or angiographic evidence of endoleak (2 patients). Injection of the sacs in two of these patients revealed a patent lumbar artery and an IMA.

*Conclusions:* It is possible to measure pressures from within the aneurysm sac in patients with stent grafts with a variety of techniques. Patients may continue to have pressurized AAA sacs despite endovascular AAA repair. Endoleaks transmit pulsatile pressure into the aneurysm sac regardless of the type. It is possible to have systemic sac pressures without evidence of endoleaks on CT or angiography. (J Vasc Surg 2001;33:32-41.)



# Intrasac flow

## Intrasac flow velocities predict sealing of type II endoleaks after endovascular abdominal aortic aneurysm repair

Frank R. Arko, MD, Konstantinos A. Filis, MD, PhD, Scott A. Siedel, MD, Bonnie L. Johnson, RVT, Angelia R. Drake, BSN, RN, Thomas J. Fogarty, MD, and Christopher K. Zarins, MD, *Stanford, Calif*

*Purpose:* The purpose of this study was to determine whether intrasac spectral Doppler flow velocities can predict whether or not a type II endoleak will spontaneously seal and to relate intrasac flow to preoperative branch vessel anatomy.

*Methods:* Between October 1996 and June 2002, 265 patients with abdominal aortic aneurysms underwent endovascular repair. Patients with less than 24 months of follow-up and type I endoleaks were excluded. Type II endoleaks were confirmed with duplex scan and computed tomographic angiography. Two groups were identified: 14 patients with sealed endoleaks (<6 months) without intervention and 16 patients with persistent endoleaks greater than 6 months and without resolution. Spectral Doppler flow velocities were recorded from endoleaks within the aneurysm sac.

*Results:*
29.9 ±
Dopple
78.8 cm
velociti

Intrasac flow velocities >100 cm/sec, washout <520 sec
→ persistent endoleak

mesenteric artery (5.6 ± 1.8 mm versus 7.2 ± 1.3 mm; $P < .01$), and fewer paired lumbar arteries (1.3 ± 0.8 versus 2.4 ± 0.6; $P < .0001$) compared with those with persistent endoleaks and high (>100 cm/s) intrasac flow velocities. Three patients with sealed endoleaks had Doppler velocities of 200 cm/s or greater. However, the diameter of the inferior mesenteric artery in these patients was 4 mm or less with no visualized lumbar arteries before surgery. Aneurysm diameter(−4.6 ± 5.6 mm) and volume (−0.9 ± 45.2 mL) decreased in patients with sealed endoleaks. Aneurysm diameter (1.8 ± 4.9 mm) and volume (18.5 ± 33.9 mL) increased slightly in patients with persistent endoleaks ($P < .05$). No ruptures or conversions occurred in any patient. Secondary interventions to treat type II endoleaks were unsuccessful in six of 16 patients (38%) with persistent endoleaks.

*Conclusion:* Intrasac Doppler velocities can be used to predict whether a type II endoleak will spontaneously seal. High-velocity type II endoleaks are related to preoperative large branch vessel diameter and number and are resistant to endovascular treatment. (J Vasc Surg 2003;37:8-15.)

**Arko et al. J Vasc Surg 2003;37:8**
**Bargellini et al. J Vasc Surg 2005;41:10**

# Must exclude T1EL and T3EL

## Evaluation and Treatment of Suspected Type II Endoleaks in Patients with Enlarging Abdominal Aortic Aneurysms

Brian Funaki, MD, Nour Birouti, MD, Steven M. Zangan, MD, Thuong G. Van Ha, MD, Jonathan M. Lorenz, MD, Rakesh Navuluri, MD, Christopher L. Skelly, MD, and Jeffrey A. Leef, MD

**ABSTRACT**

**Purpose:** To evaluate angiographic diagnosis and embolotherapy of patients with enla computed tomographic (CT) diagnosis of type II endoleak.

**Materials and Methods:** A retrospective review was performed of all patients referred radiology section from January 1, 2003, to June 1, 2011, with a diagnosis of enlarging aneu patients underwent 40 procedures between 12 and 82 months after endograft insertion (mea of endoleaks.

**Results:** Type II endoleaks were treated with cyanoacrylate, coils, and ethylene vinyl alco success rate was 88% (14 of 16 patients) and clinical success rate was 100% (16 of 16 pati all cases over a mean follow-up of 27.5 months (range, 6–88 mo). Endoleaks in nine patients I endoleaks and seven had type III endoleaks. Four of the nine patients (two type I endoleaks a classified after initial angiography. The other five type III endoleaks were correctly classifi inferior mesenteric artery. Direct embolization was performed via sac puncture with ethylen latter five patients and eliminated endoleaks in both.

**Conclusions:** Aneurysm growth caused by type II endoleaks was arrested by embolization. commonly; type III endoleaks purported to be type II endoleaks were found in 28% of pat

*J Vasc Interv Radiol 2012; 23:866–872*

## Occult type I or III endoleaks are a common cause of failure of type II endoleak treatment after endovascular aortic repair

Check for updates

Michael C. Madigan, MD, Michael J. Singh, MD, Rabih A. Chaer, MD, Georges E. Al-Khoury, MD, *and* Michel S. Makaroun, MD, *Pittsburgh, Pa*

**ABSTRACT**

**Objective:** Most type II endoleaks have a benign natural history, but 6% to 8% are associated with sac enlargement and respond poorly to treatment. Our aim was to evaluate whether these enlargements are associated with delayed or occult type I and III endoleaks.

**Methods:** Patients with interventions for endoleak after endovascular aortic repair from 2000 to 2016 were reviewed retrospectively. Patient demographics, comorbidities, endoleak type, secondary procedures, aortic sac growth (≥5 mm), and mortality were collected. Successful treatment was defined as endoleak resolution with no further aortic sac growth. Secondary procedures, ruptures, endograft explant, and death were captured.

**Results:** There were 130 patients diagnosed with a primary type II endoleak after endovascular aortic repair at a median of 1.3 months (interquartile range, 1.0-13.3 months). One hundred eighteen had their initial treatment for a primary type II. Twelve of the 130 were initially stable and observed, but were treated for a delayed type I or III endoleak. The 130 patients underwent 279 procedures for endoleaks (mean of 2.2 ± 1.3) over 6.9 ± 3.8 years of follow-up. Of the 118 patients treated for primary type II endoleaks, 26 (22.0%) later required interventions for delayed type I and III endoleaks. The mean time to intervention for a delayed type I or III endoleak was 5.4 ± 2.8 years. Overall, there were 16 type IA, 11 type IB, 2 type III, 7 combined type IA/IB, and 2 type IA/III delayed endoleaks. The odds of harboring a delayed type I or III endoleak was 22.0% before the first attempt at type II endoleak treatment, 35.1% before the second, 44.8% before the third, and 66.6% before the fourth attempts. Rapid aortic sac growth of ≥5 mm/y before initial endoleak treatment was associated with increased risk for delayed type I or III endoleak (47.8 vs 14.1%; $P = .003$). Patients with delayed type I or III endoleaks had a lower successful treatment rate (8.3% vs 52.3%; $P = .001$) than those with only type II endoleaks. Late rupture was increased with delayed type I or III endoleak ($P = .002$), whereas mortality ($P = .96$) and aortic-related mortality ($P = .46$) were similar. Graft explant ($P = .06$) trended toward an increase with a delayed type I or III endoleak, but was not statistically significant.

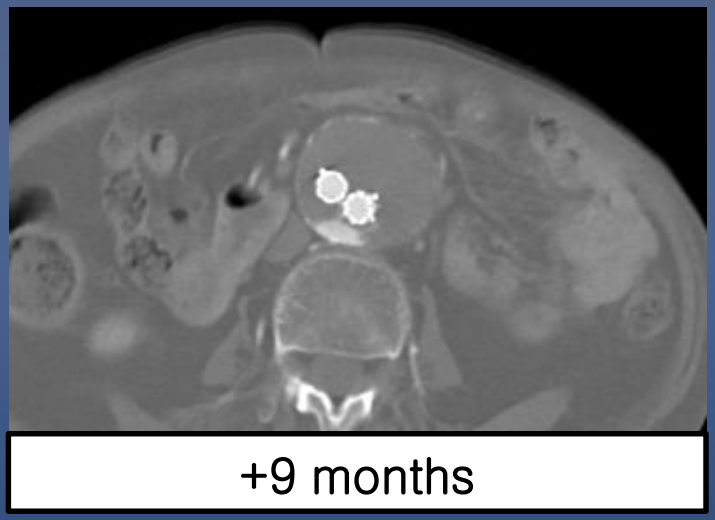
**Conclusions:** Failed attempts treating type II endoleaks and/or a rapid aortic sac growth of 5 mm/y or greater should raise the suspicion of a delayed or occult type I or III endoleak. Occult endoleaks are associated with decreased chance of endoleak resolution. (J Vasc Surg 2019;69:432-9.)

**Keywords:** Aorta; Aneurysm; Endovascular; Endoleak

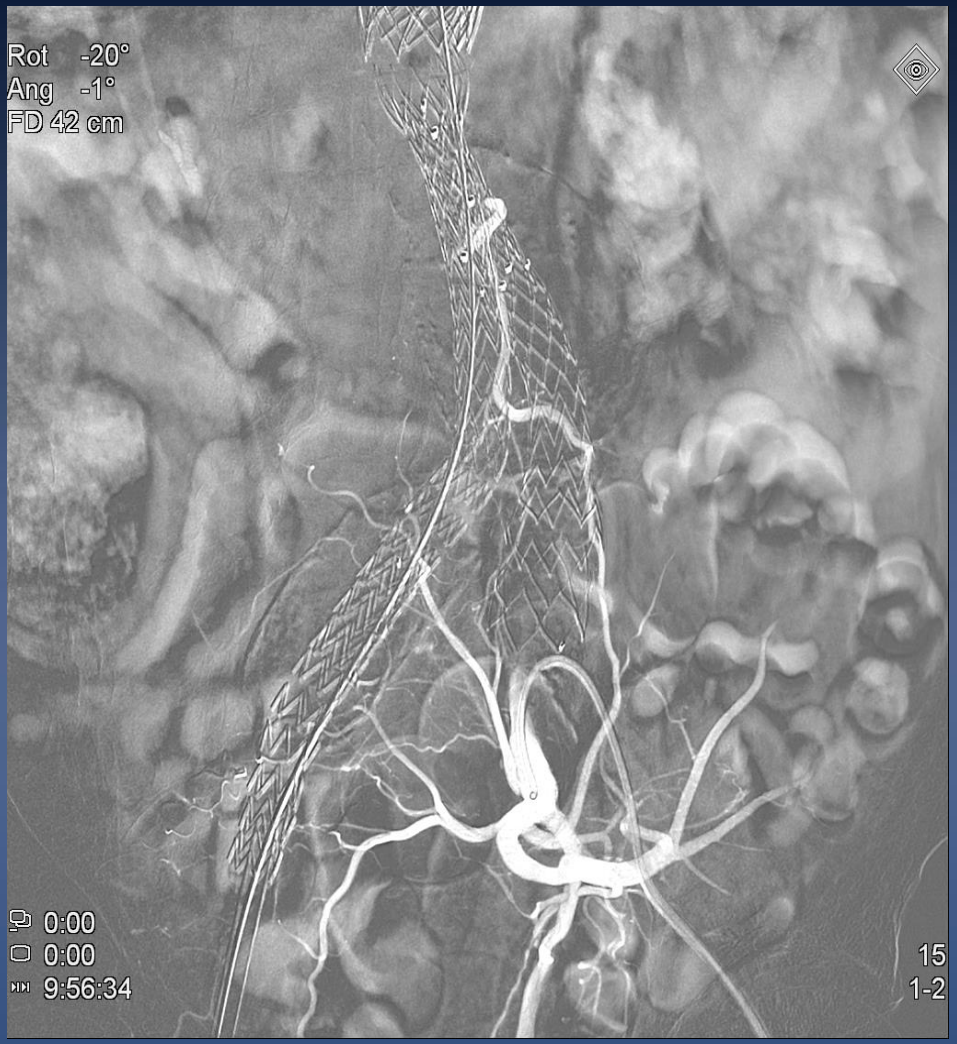
# F71, T2EL

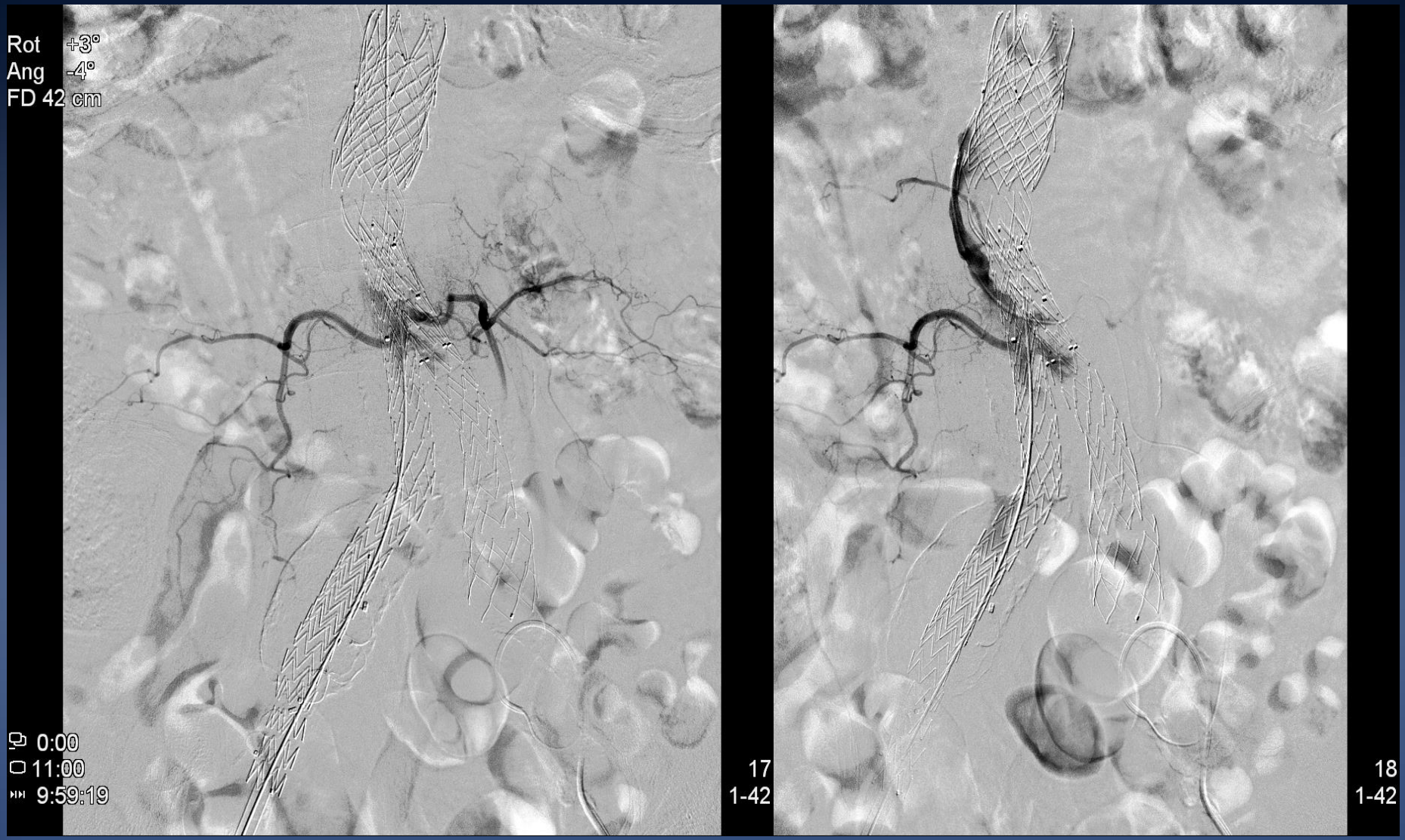
# 12M fu
# +9M f/u

Post-EVAR 12 months



+9 months

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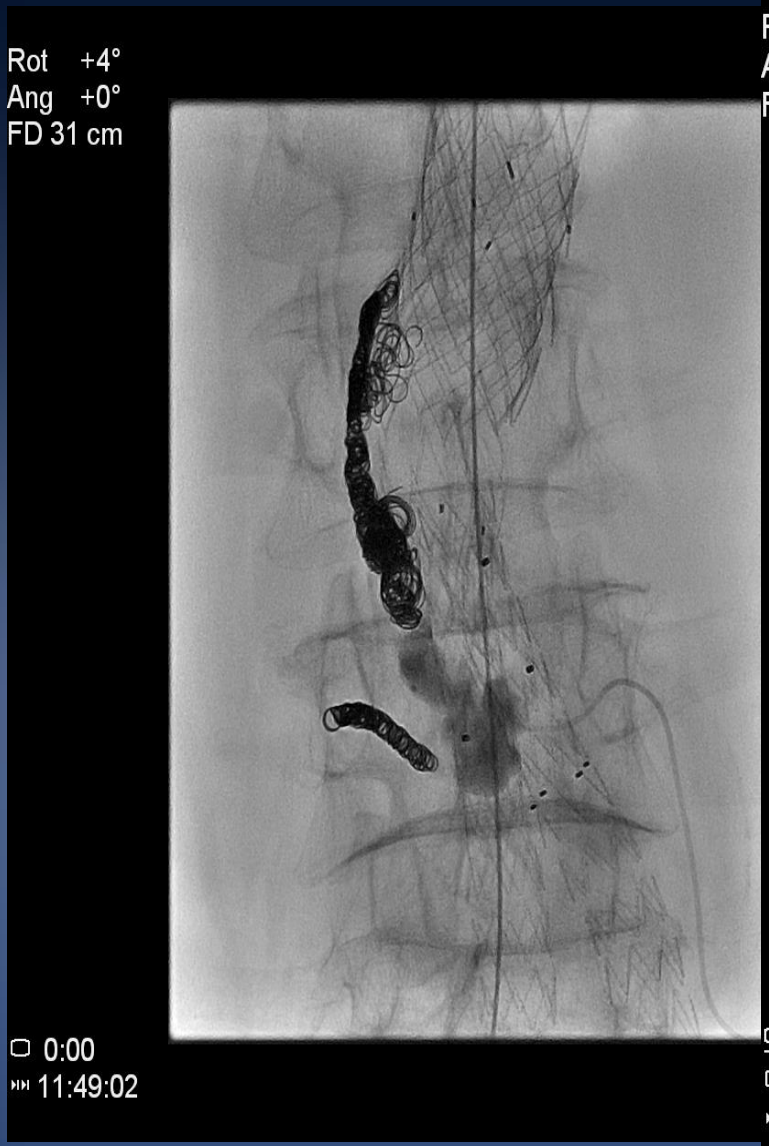
1-42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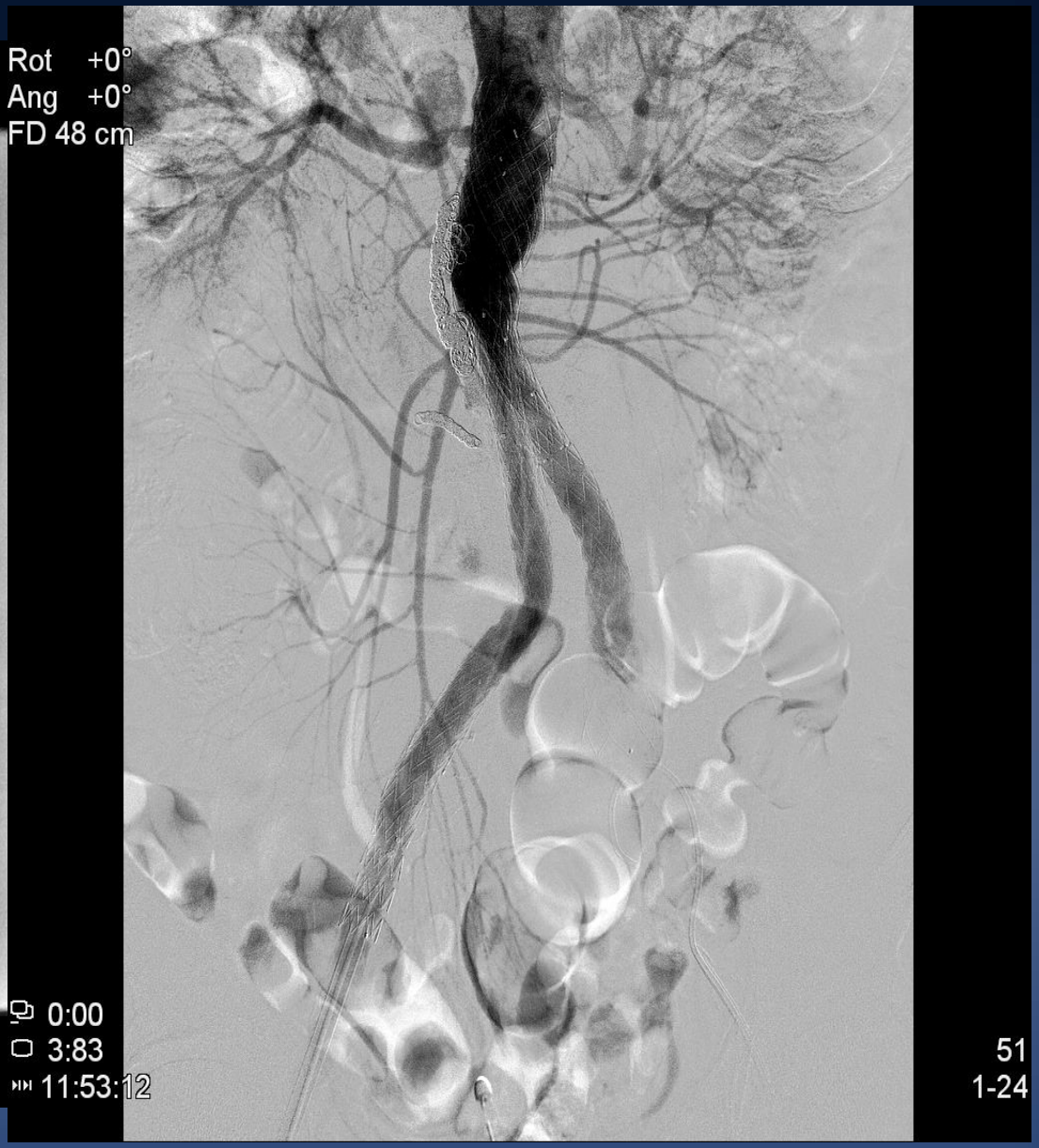
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51
1-24

# Conclusion/Take-home Message

- Surveillance is mandatory after all EVAR
- Must exclude T1EL and T3EL
- Lack of level I evidence on outcome and strategy for T2EL
- Treatment recommended if:
  - Persistent EL (>6 months)
  - Sac enlargement