

Type II lumbar endoleaks: Hemodynamic differentiation by contrast-enhanced ultrasound scanning and influence on aneurysm enlargement after endovascular aneurysm repair

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Objective: The objective of this study was to differentiate type II lumbar endoleaks on the basis of dynamic features identified by contrast-enhanced ultrasound scanning (CUS) and to evaluate the role of this differentiation in detecting abdominal aortic aneurysm (AAA) enlargement ≥ 1 mL/mo.

Methods: Eighteen male patients (mean age, 71.8 years) with type II lumbar endoleak suspected at CUS underwent computed tomography angiography (CTA) and digital subtraction angiography (DSA). On CTA, AAA volumes and endoleak visualization and volume were assessed. At CUS, performed after a bolus of 1.5 to 2.4 mL of a second generation blood pool contrast agent, the following parameters were evaluated: presence of contrast material within the aneurysmal sac (endoleak), delay of endoleak detection (wash-in) and disappearance (washout) from the beginning of contrast injection, visualization of inflow and outflow vessels, and presence of cavity filling. Statistical analysis was performed regarding endoleak features at CUS, endoleak detection at CTA, and rate of AAA enlargement.

Results: DSA confirmed all the endoleaks. Mean \pm standard deviation wash-in and washout times were 121.9 ± 132.6 and 337.2 ± 193.7 seconds, respectively; a significant relation was observed between these two parameters ($P < .01$, analysis of variance). By Youden plots, endoleaks were classified as hyperdynamic when wash-in was < 100 seconds ($n = 10$, 55.5%) and/or washout was < 520 seconds ($n = 13$, 72.2%). A slower washout was associated with nonvisualized outflow (66.7%) and/or inflow arteries (66.7%) ($P < .05$). Eight endoleaks (44.4%) were missed at CTA; it occurred in hypodynamic endoleaks, absence of detectable inflow or outflow vessels, and absence of cavity filling at CUS ($P < .05$). Overall mean AAA volume increase rate was 1.1 ± 1.7 mL/mo. By multiple logistic regression model, the washout time ≥ 520 seconds was the only independent predictor of AAA volume increase ≥ 1 mL/mo (8 patients, 44.4%).

Conclusion: Type II lumbar endoleaks show different hemodynamic features at CUS, which might influence the rate of aneurysm enlargement, addressing the need for treatment. (J Vasc Surg 2005;41:10-8.)

Endoleaks are defined as persistent blood flow into the aneurysm sac and outside the graft lumen and represent the most frequent complication after endovascular aneurysm repair (EVAR). They might be associated with aneurysm enlargement and possible rupture.¹ The reported incidence of endoleaks ranges from 10% to 45%, and lifelong surveillance is required for their early detection and management.²⁻⁵

Computed tomography angiography (CTA) represents the actual gold standard in the detection of endoleaks, in particular when both the arterial and the venous phases are acquired,^{6,7} although the role of magnetic resonance angiography (MRA), duplex ultrasound scanning (US), and, more recently, contrast-enhanced US (CUS) has been investigated.⁸⁻¹³

Endoleaks are classified according to their origin, which can be directly related to stent-graft complications (types I, III, and IV) or to retrograde flow from aortic collateral vessels (type II).^{14,15} The presence of aneurysm enlargement without any evident complication has also been described and defined as endotension or type V endoleak.^{16,17}

Type II endoleaks have been reported to be the most common type of leakage.² Recent studies have demonstrated that they might present different hemodynamic features, and that differentiation might have a role in their management and treatment.^{11,18}

The purpose of our study was to differentiate type II lumbar endoleaks on the basis of specific dynamic features identified by CUS and to evaluate the capability of this differentiation in identifying abdominal aortic aneurysm (AAA) enlargement ≥ 1 mL/mo.

MATERIALS AND METHODS

Patients and overall study design. Our study was approved by our Institutional Review Board, and informed written consent was obtained in all cases.

From February 2002 to March 2004, 39 consecutive, randomly selected patients with abdominal aortic stent-

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Competition of interest: none.

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graft underwent CUS (Technos; Esaote Biomedica, Genoa, Italy) at completion of a duplex US examination. Among this series, 18 male patients (mean age, 71.8 ± 7.8 years; range, 52 to 83 years) exhibited CUS findings suggestive for a type II lumbar endoleak and therefore entered the study. Several types of stent-graft were implanted in this series (Table I), and in 8 of 18 cases preprocedural hypogastric embolization was performed to allow deployment of an external iliac cuff.

After endovascular aneurysm repair (EVAR), all patients underwent CTA (LightSpeedPlus; GE Medical Systems, Milwaukee, Wis) at 7-day, 6- and 12-month follow-up, and annually thereafter. In patients with detected endoleak, examinations were performed every 3 to 6 months. After CUS, all patients underwent digital subtraction angiography (DSA) (Multistar; Siemens, Erlangen, Germany).

CTA protocol, post-processing and evaluation. CTA was performed from the celiac artery to the common femoral arteries, both before and after intravenous contrast administration (Visipaque 320; Nycomed, Oslo, Norway) at the dose of 120 mL with a flow-rate of 3 mL/s by using the following parameters: HighSpeed mode capability; gantry rotation time, 0.5 to 0.6 second; table speed, 7.5 mm per rotation; collimation, 2.5 mm; and reconstruction section thickness, 1.2 mm. Both the arterial phase and a delayed phase were acquired. Scan delay ranged between 20 and 40 seconds, according to the patient circulation time, determined by an automated bolus time test (SmartPrep; GE Medical Systems) by using 25 mL of iodinated contrast medium. Scans obtained during the venous phase were acquired with the same parameters, 80 seconds after contrast material injection.

Images were processed with a dedicated software package at an independent workstation (Advantage Windows 4.1; Sun Microsystems, Mountain View, Calif) to generate multiplanar reformations (MPRs), maximum intensity projections (MIPs), and volume renderings (VRs).

On CTA, volumes were obtained through a semiautomatic segmentation process by using the summation of area technique. The region-of-interest was drawn manually at the interface of the outer aortic wall and the extra-aortic tissues at 5 to 10 different levels throughout the aorta from the lowest renal artery to the aortic bifurcation. Then the system automatically determined the boundaries around a class of similar voxel intensity values in the remaining sections, and aneurysm sac volume was calculated.¹⁹ Presence and origin of endoleaks were recorded, and endoleak volume was calculated on the venous acquisitions, with the same modality described for AAA volume. Post-processing and volume assessment was retrospectively performed by a single expert radiologist (I.B., 5 years of experience in CTA and post-processing) blinded to the CUS findings. Each measurement was performed twice (coefficient of variation for AAA volumes, 1.74%), and the mean value of the two measures was obtained and used for the study.

In addition, CTA images were reviewed by consensus by one of several authors (R.C., P.P., C.V., 10 to 15 years

Table I. Patients' demographic and procedural data

Patient no.	Age (y)	EF (%)	Initial diameter (mm)	Stent-graft	Endoleak diagnosis	Hypogastric embolization
1	78	55	45	Excluder	Early	No
2	73	52.7	56	AneuRx	Early	Yes
3	70	58.8	75	Excluder	Late	Yes
4	77	29.4	59	AneuRx	Early	Yes
5	66	50	74	AneuRx	Late	No
6	61	55.7	52	Excluder	Early	Yes
7	75	60	46	Excluder	Early	Yes
8	78	55	47	Talent	Late	No
9	66	52	63	Excluder	Late	No
10	78	55	47	Talent	Late	No
11	61	38.8	60	AneuRx	Late	Yes
12	83	48.6	88	Zenith	Late	No
13	74	54	45	AneuRx	Late	No
14	52	56	55	Endologix	Early	No
15	73	52.7	56	AneuRx	Early	Yes
16	74	60	47	Endologix	Late	No
17	78	55	47	Talent	Late	No
18	75	52.5	58	Talent	Late	Yes

EF, Cardiac ejection fraction.

of experience in CTA) blinded to the volume measurements to identify presence of endoleak (defined by contrast enhancement within the sac and outside the stent-graft lumen) and of other complications (such as stent-graft migration or disconnection).

Contrast-enhanced US and evaluation. CUS was performed by one radiologist (V.N., 4 years of experience with CUS) blinded to CTA results, after a bolus injection of a second generation blood pool contrast agent (Sonovue; Bracco, Milan, Italy), consisting in stabilized microbubbles of sulfur hexafluoride gas, administered into an antecubital vein at the dose of 1.5 to 2.4 mL, followed by a flush of 5-mL saline solution. At the time this study was carried out, no published data were available regarding the optimal dose of contrast material for vascular examinations with CUS, particularly in the follow-up of stent-grafts. At the beginning of our experience, we performed CUS with a bolus of 1.5 mL; afterwards, the bolus was raised up to 2.4 mL, as package insert recommended, to ensure endoleak visualization in all patients. In the present series, the 2.4-mL bolus was injected in the last six patients. The contrast-enhanced sonographic study was performed with a continuous low Mechanical Index (0.01 to 0.04) real-time tissue harmonic imaging (Contrast Tuned Imaging; Esaote Biomedica). The entire aorta was scanned in the longitudinal and transverse planes from the diaphragm to below the iliac limb attachment sites. Scanning was maintained for 10 minutes after contrast administration, according to the pharmaceutical indications, setting at 11 minutes the maximum circulation time of the contrast agent. The entire examination was tape-recorded to allow later review.

The following parameters were assessed: presence of endoleak (defined as contrast enhancement within the aneurysm sac), endoleak wash-in (time between beginning of contrast material injection and contrast visualization within

the sac), endoleak washout (time between beginning of contrast material injection and disappearance of all contrast from the sac), and visualization of inflow and outflow vessels. Moreover, the contrast enhancement was described either as "cavity filling" (defined as contrast concentration into a pseudocavity within the sac) or as simple spreading of contrast within the sac.

DSA and evaluation. DSA was performed after CUS and CTA in the angiographic suite by four experienced interventional radiologists (I.B., P.P., R.C., C.V., 5 to 25 years of experience with angiography) aware of the findings of previous examinations. A transfemoral arterial percutaneous access was used under local anesthesia. Aortography was obtained by using a 5F pigtail catheter, positioned above the proximal stent-graft attachment, with a bolus of 40 mL iodinated contrast medium (Visipaque 320). Then selective angiographies of the internal iliac and superior mesenteric arteries were performed according to the aortographic findings by using the proper shaped catheter. Images were evaluated by consensus to assess presence of endoleak (visualized as spreading of contrast material outside the stent-graft) and to identify its origin (from the aortic branches or stent-graft disconnections).

Statistical analysis. Descriptive statistics (proportions, means, standard deviations [SDs], medians) were calculated for patient characteristics and numeric continuous data. The χ^2 , Pearson, and Fisher exact tests were used for categorical data, whereas continuous data were evaluated by bivariate fit, one-way analysis of variance, and Student *t* test. A *P* value $<.05$ was considered statistically significant.

Analysis was performed to identify possible factors related with (1) endoleak dynamic features at CUS, (2) endoleak visualization at CTA, and (3) rate of AAA volume enlargement per month.

Endoleak dynamic features were tested against the following parameters: patient age, cardiac ejection fraction, preprocedural hypogastric embolization, stent-graft type, preprocedural AAA maximum diameter (calculated on CTA on the plane perpendicular to the aortic centerline length), early or late endoleak (detected less or more than 30 days after EVAR), outflow arteries at DSA, endoleak features at CTA (visualization and volume), and amount of contrast material administered at CUS. CUS detection of inflow and outflow vessels and identification of cavity filling were tested against wash-in and washout times.

Endoleak detection at CTA was analyzed in relation with the following parameters: patient age, preprocedural hypogastric embolization, stent-graft type, preprocedural AAA maximum diameter, early or late endoleak, outflow artery at DSA, and endoleak features at CUS.

The rate of AAA increase was expressed in mL/mo, obtained by subtracting the AAA volumes calculated at the last two consecutive CTA examinations, divided by the number of months elapsing between examinations. A rate of enlargement ≥ 1 mL/mo was arbitrarily selected to define a significant volume increase and was tested against patient age, duration of follow-up after EVAR, preprocedural hypogastric embolization, stent-graft type, preproce-

dural AAA maximum diameter, early or late endoleak, outflow artery at DSA, and endoleak features at CTA and CUS.

By Youden plots (a graphic method able to determine a value to optimize specificity and sensitivity of the test measurement, it is similar to the Receiver Operating Characteristic (ROC) curve but not influenced by the prevalence of an event in the studied population, which makes it suitable for low number of patients), cutoff values of wash-in and washout times were set to identify rate of AAA enlargement ≥ 1 mL/mo, and their sensitivity, specificity, positive (PPV) and negative predictive values (NPV), and accuracy were calculated.

A multiple logistic regression analysis was performed to identify independent predictors of AAA enlargement ≥ 1 mL/mo.

RESULTS

Demographic data, CTA, DSA, and CUS findings are extensively reported in Tables I to III. No adverse reactions were observed after ultrasound contrast material administration. Endoleaks were detected 1 to 36 months after EVAR (mean \pm SD, 19 ± 16 months; median, 24 months); seven endoleaks were diagnosed within the first month of follow-up, whereas the remaining cases were late endoleaks. In all patients, DSA excluded the presence of a type I endoleak. A type II endoleak was clearly demonstrated in 14 cases, with visualization of the lumbar arterial inflow. In the remaining four cases (numbers 12, 13, 14, 18), DSA did not depict evident concentration of contrast material within the thrombus; however, it demonstrated complex hypogastric collateral pathways feeding multiple tiny lumbar arteries afferent to the aneurysm sac.

In 9 of 18 patients an outflow artery was demonstrated at DSA, represented by the inferior mesenteric artery in 4 cases and by lumbar arteries in the remaining 5 patients.

At CTA, only 10 of 18 endoleaks were detected. In the remaining 8 patients, the endoleak could not be visualized in the arterial or in the venous scans. Endoleak volume, calculated on venous scans, ranged from 0 mL (no evidence of endoleak) to 11.6 mL (mean \pm SD, 2.07 ± 3.3 mL).

Analysis of endoleak features at CUS. Mean \pm SD wash-in time was 122 ± 132 seconds (median, 84 seconds; range, 25 to 531 seconds); mean \pm SD washout time was 337 ± 194 seconds (median, 320.5 seconds; range, 52 to 600 seconds); these two parameters were directly associated ($P = .007$, analysis of variance). An inflow lumbar artery at CUS was detected in 6 patients; an outflow artery was visualized in 6 patients; in 4 of these patients both inflow and outflow arteries were visualized. Presence of cavity filling was delineated in 7 cases.

Longer wash-in times were identified in patients with lower cardiac ejection fractions; however, the difference was not statistically significant ($P = .08$). Faster washout time was significantly associated with the visualization of an outflow artery at CUS ($P = .01$, Student *t* test). Endoleaks with washout time ≥ 520 seconds were associated with the

Table II. CTA and DSA findings

Patient no.	Time between CTA exams (mo)	AAA volume (mL/mo)	AAA diameter (mm/mo)*	Endoleak volume (mL)	Endoleak detection at CTA	Outflow detection at DSA
1	8	-1.19	-0.54	1.6	Yes	IMA
2	4	2.1	0.82	6.6	Yes	IMA
3	6	0.23	0.22	0	No	No
4	4	2.62	0.07	8	Yes	No
5	8	0.36	-0.14	0.8	Yes	No
6	3	-1.37	-0.5	0.2	Yes	No
7	6	0.67	0.38	3.5	Yes	Lumbar
8	6	2.48	0.38	1.75	Yes	Lumbar
9	12	0.46	0.08	0	No	No
10	5	2.24	0.46	2.5	Yes	Lumbar
11	12	0.33	0.21	0	No	Lumbar
12	6	1.66667	0.17	0	No	No
13	12	0.64	0.13	0	No	No
14	12	1.58	0.36	0	No	No
15	7	-1.19	-0.54	11.6	Yes	IMA
16	12	0.04	-0.12	1.7	Yes	IMA
17	4	2.5	0.37	0	No	Lumbar
18	12	5.72	0.37	0	No	No

IMA, Inferior mesenteric artery.

*No relation between maximum diameter modifications and endoleak dynamic features at CUS.

Table III. Findings at CUS

Patient no.	Duration of follow-up (mo)*	Bolus (mL)	Wash-in (sec)	Dynamic wash-in	Washout (sec)	Dynamic washout	Inflow visualization	Outflow visualization	Cavity filling
1	21	1.5	40	Hyper	360	Hyper	No	No	Yes
2	7	1.5	25	Hyper	129	Hyper	Yes	Yes	Yes
3	18	1.5	50	Hyper	517	Hyper	No	No	No
4	28	1.5	110	Hypo	540	Hypo	No	No	Yes
5	46	1.5	36	Hyper	52	Hyper	No	No	No
6	3	1.5	113	Hypo	281	Hyper	Yes	No	No
7	24	1.5	28	Hyper	172	Hyper	Yes	Yes	Yes
8	43	1.5	67	Hyper	245	Hyper	Yes	Yes	Yes
9	31	1.5	40	Hyper	192	Hyper	No	No	No
10	45	1.5	104	Hypo	185	Hyper	Yes	Yes	Yes
11	36	1.5	220	Hypo	379	Hyper	No	Yes	No
12	12	1.5	113	Hypo	600	Hypo	No	No	No
13	41	2.4	34	Hyper	63	Hyper	No	No	No
14	26	2.4	142	Hypo	574	Hypo	No	No	No
15	24	2.4	73	Hyper	186	Hyper	No	Yes	No
16	36	2.4	95	Hyper	425	Hyper	Yes	No	Yes
17	55	2.4	531	Hypo	600	Hypo	No	No	No
18	37	2.4	373	Hypo	570	Hypo	No	No	No

Hyper, Hyperdynamic; Hypo, hypodynamic.

*Number of months between EVAR and CUS.

lack of visualization of inflow arteries at CUS and of outflow vessels at CUS and DSA ($P < .05$, χ^2 test).

The amount of contrast material injected did not affect the intra-arterial flow detection or any of the endoleak dynamic features.

Analysis of endoleak detection at CTA. Endoleaks missed at CTA showed longer wash-in and washout times (mean \pm SD, 187.9 ± 179.4 and 436.9 ± 206.8 seconds, respectively) compared with endoleaks visualized at CTA (mean \pm SD, 69.1 ± 35.1 and 257.5 ± 147.4 seconds,

respectively); the difference was statistically significant ($P = .05$, one-way analysis of variance and Student t test).

Presence of cavity filling at CUS seemed to allow endoleak visualization at CTA (Fig 1). In fact, all endoleaks with cavity filling at CUS ($n = 7$) were visualized at CTA, whereas only 3 of 11 leaks with no cavity filling at CUS could be depicted by CTA ($P = .0006$, χ^2 test).

The presence of a detectable inflow at CUS was significantly associated with endoleak depiction at CTA ($P = .002$, χ^2 test); all endoleaks with visualized inflow ($n = 6$)

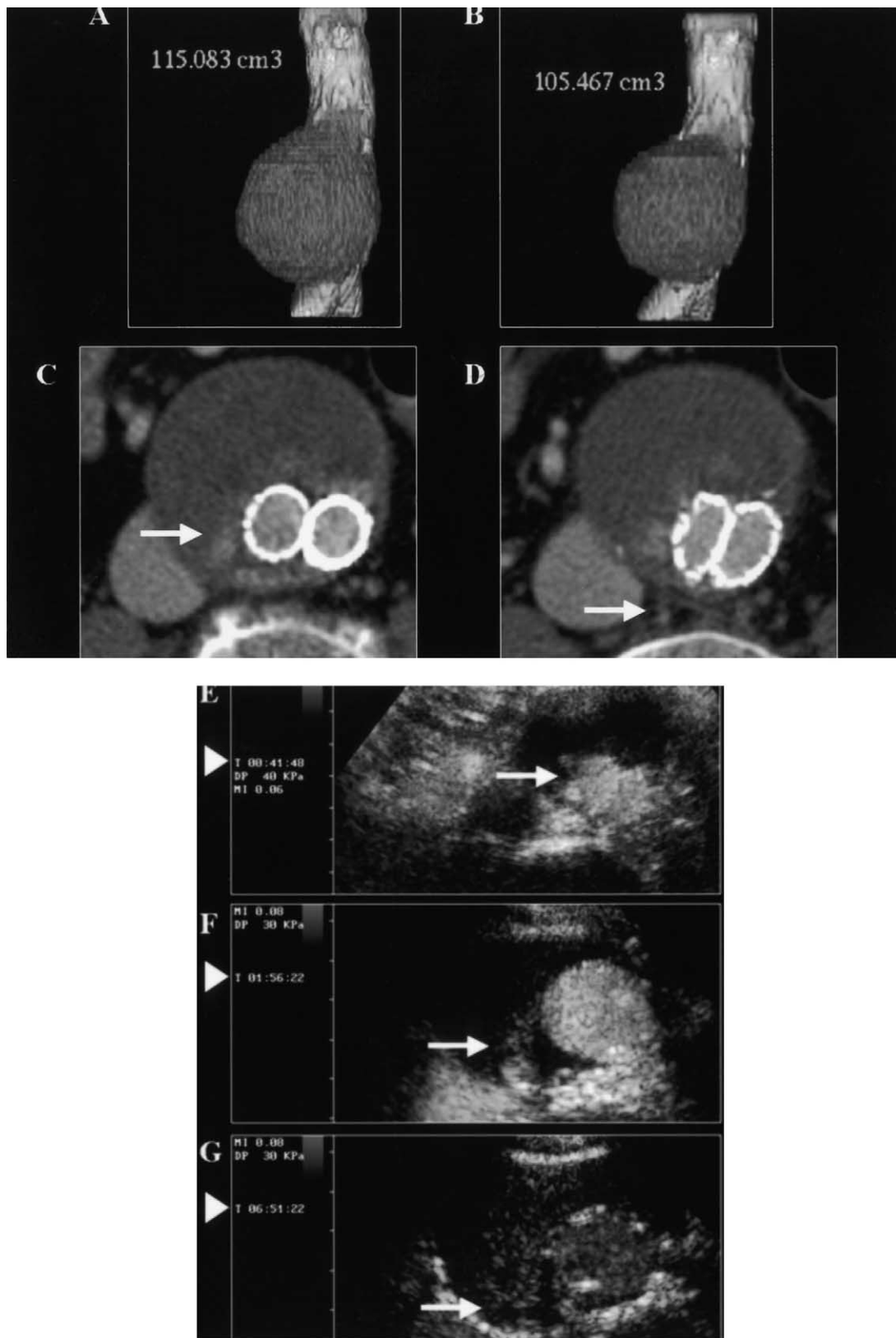


Fig 1. Patient 1, 78-year-old man with hyperdynamic endoleak. CTA shows a decrease in AAA volume at 20-month follow-up (**B**) compared to the 12-month follow-up control (**A**), despite the evidence of a type II endoleak visualized on the axial venous scans (**C**, *arrow*) fed by lumbar arteries (**D**, *arrow*). CUS shows the posterior endoleak (*arrows*) characterized by the wash-in at 40 seconds (**E**), progressive cavity filling (**F**), and washout at 360 seconds; the contrast material is no longer appreciated almost 10 minutes after contrast material administration. *Arrowheads* indicate the time (minutes and seconds) after injection of contrast material. At the *top left corner*, main parameters of CUS are indicated: mechanical index (*MI*) and derated pressure (*DP*).

were depicted, whereas only 4 of 12 endoleaks without inflow were visible at CTA.

Also the presence of an outflow at DSA seemed to increase endoleak detectability at CTA; all endoleaks with inferior mesenteric artery outflow and 3 of 4 cases with lumbar outflow were detected at CTA, whereas only 3 of 10 endoleaks without outflow were depicted at CTA ($P = .01$, χ^2 test).

Analysis of AAA volume increase. Mean AAA volume increase rate was 1.1 ± 1.73 mL/mo (median, 0.654 mL/mo; range, -1.37 to 5.72 mL/mo). An enlargement rate ≥ 1 mL/mo was observed in 8 patients, whereas the remaining 10 patients exhibited volume changes <1 mL/mo. In 3 patients the aneurysm showed a progressive shrinkage despite the presence of the endoleaks (Fig 1). The aneurysm size change was not influenced by the duration of follow-up.

AAA enlargement <1 mL/mo was significantly more frequent in AneuRx (Medtronic Inc, Minneapolis, Minn) (4 of 6) and Excluder (W.L. Gore & Associates, Inc, Newark, Del) (5 of 5) stent-grafts compared to the other types of devices ($P = .003$, likelihood test).

The mean endoleak volume calculated at CTA did not differ comparing patients with (2.6 ± 3.1 mL) and without (1.8 ± 3.6 mL) significant volume enlargement.

Mean wash-in and washout times were shorter in patients with AAA enlargement rate <1 mL/mo (72.9 ± 58.9 and 262.7 ± 155.5 seconds, respectively), compared to patients with AAA enlargement ≥ 1 mL/mo (183.1 ± 174.7 and 430.4 ± 205.3 seconds, respectively).

By Youden plots, the wash-in and washout cutoff values to identify significant AAA volume increase rate were 100 and 520 seconds, respectively (Figs 1 and 2). Times lower than these referral values were used to define the endoleak as hypodynamic.

According to the washout time, 5 endoleaks were defined as hypodynamic and the remaining 13 as hyperdynamic. Sensitivity, specificity, PPV, NPV, and accuracy in identifying significant enlarging AAAs were 62.5%, 100%, 100%, 76.9%, and 83.3%, respectively ($P = .001$). In fact, all hypodynamic endoleaks showed significant enlarging AAAs, together with three hyperdynamic endoleaks (patients 2, 8, and 10), whose washout times were 129, 245, and 185 seconds, respectively.

According to the wash-in time, 10 endoleaks were classified as hyperdynamic and 8 cases as hypodynamic; 3 cases showed fast washout with slow wash-in (patients 6, 10, and 11). Sensitivity, specificity, PPV, NPV, and accuracy in identifying enlarging AAAs were 75%, 80%, 75%, 80%, and 77.7%, respectively ($P = .01$); 2 hyperdynamic endoleaks were associated with significant increase, and 2 hypodynamic endoleaks showed no significant AAA increase.

According to the multiple logistic regression analysis, the only independent predictor of AAA volume enlargement rate ≥ 1 mL/mo was the washout time >520 seconds ($P = .009$).

DISCUSSION

Endoleaks represent the most frequent complication after EVAR, occurring in up to 45% of patients, and can determine aneurysm enlargement and increased pressurization of the aneurysm sac, which require treatment to reduce the risks of rupture.^{2,20} Endoleak classification has changed over the years and currently distinguishes between endoleaks resulting in direct antegrade perfusion of the aneurysm sac (types I, III, and IV) and endoleaks caused by retrograde flow from aortic collateral vessels (type II).^{14,15} In addition, type V endoleaks have been introduced to describe the continuous enlargement of an apparently excluded aneurysm, the so-called endotension.^{16,17,21,22}

Type II endoleaks are reported to be far more frequent than the other types² and might represent a challenge in terms of when and how to treat them.^{23,24} Recent studies have demonstrated that they can be differentiated on the basis of Doppler waveforms and flow velocities, affecting endoleak persistence and treatment outcome.^{2,11,18,25}

The purpose of our study was to investigate the ability of CUS in differentiating type II lumbar endoleaks according to specific CUS dynamic features and to identify a possible correlation between this differentiation and the rate of AAA enlargement. CUS has been proved to be a useful tool in endoleak detection.^{13,26} According to our experience, it provides the unique advantage of real-time imaging, thus representing a valid and relatively easy method in the analysis of endoleak dynamic behavior.

Our series included exclusively patients with CUS findings suggestive for type II lumbar endoleaks that were confirmed at DSA. By CUS, they showed different features in terms of contrast enhancement and washout. According to our data, these differences seem not to be related to the endoleak extension or to the patient's characteristics, such as age and cardiac ejection fraction, that can affect systemic cardiovascular conditions or to the amount of sonographic contrast material injected, although the series is still limited, and only two different volumes of contrast were tested. On the contrary, wash-in and washout times proved to be mainly associated with the visualization of inflow and outflow arteries. The absence of an outflow artery could explain enhancement persistence and, therefore, what we defined as a hypodynamic endoleak. Also, the lack of visualization of the inflow at CUS was related to slow contrast enhancement, probably because of the presence of tiny lumbar arteries not detectable by CUS.

Hypodynamic endoleaks were also significantly related to the lack of endoleak visualization at CTA. In fact, although CTA still represents the preferred imaging modality in the follow-up of aneurysm stent-grafts,^{6,7} several studies have pointed out the presence of leaks that are missed at CTA,² and alternative imaging tools have been proposed such as MRA,⁸ duplex US,^{27,28} and, lately, contrast-enhanced US.¹³ In a recent study on patients with AAA enlargement and no evidence of complications, CUS was able to identify the endoleak, thus arguing the existence of what has been defined as endotension.²⁶

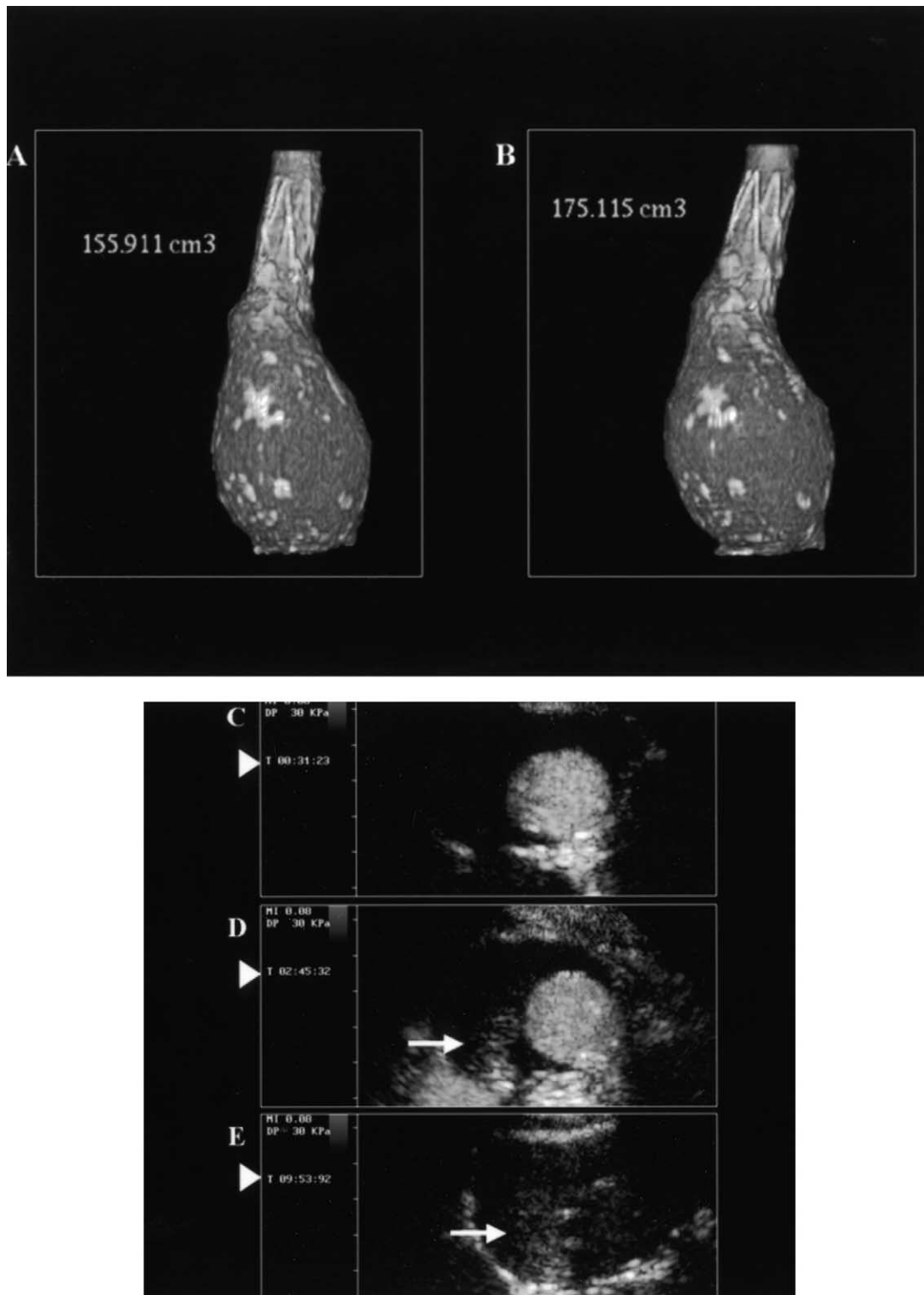


Fig 2. Patient 14, 52-year-old man with hypodynamic endoleak. Compared to the 1-month CT control (A), the 12-month follow-up AAA volume shows a significant increase (B). However, no endoleak could be demonstrated at CTA. CUS shows the absence of enhancement in the first minute scan after contrast material injection (C); a posterior enhancement is appreciated more than 2 minutes after injection of contrast material (D, *arrow*), persisting for almost 10 minutes (E, *arrow*). *Arrowheads* indicate the time (minutes and seconds) after injection of contrast material. At the *top left corner*, main parameters of CUS are indicated: mechanical index (MI) and derated pressure (DP).

In our series, all the endoleaks that were not detected at CTA were hypodynamic leaks. This association might confirm the hypothesis that the so-called endotension might be represented by a very slow flow endoleak, not depictable with standard imaging modalities.²¹ CTA visualization of the endoleak was also related to the absence of inflow arteries depicted at CUS (with slower wash-in time) and of an outflow artery at CUS and DSA.

Finally, all endoleaks without what we called cavity filling at CUS were missed at CTA. The spreading of the contrast medium within the thrombus, without concentration in a defined region of the sac, could reduce CTA detection capability; this limit could be overcome by MRA and CUS^{8,9,13} by using blood pool contrast agents, which could enhance flow detection.

To assess the role of dynamic features in the pressurization of the aneurysm, we calculated aneurysm volume defining a rate of increase per month. Volume assessment has been proved to be more reliable and useful than maximum transverse diameters in the evaluation and management of aneurysm enlargement.²⁹⁻³² In our series, only eight patients showed a rate of volume increase ≥ 1 mL/mo, and three patients showed no enlargement at all, despite the presence of the lumbar endoleak. This result confirms previous findings, demonstrating the different behavior of the leaks on aneurysm pressurization^{33,34} and challenging physicians on when and how to treat lumbar endoleaks,^{23,24} especially considering how difficult and disappointing their treatment can be.³⁵

Our data suggest that a volume increase ≥ 1 mL/mo might be associated with hypodynamic type II leaks, particularly when considering a washout time ≥ 520 seconds. Probably the hypodynamic endoleak is not able to create a way out of the sac, thus causing aneurysm pressurization and progressive enlargement,^{36,37} independently from other factors such as endoleak volume and extension. On the contrary, fast wash-in and washout could be interpreted as rapid flow going through the sac without significant effects on pressure and aneurysm increase; in these cases, treatment could be avoided. Nevertheless, our hypothesis is not supported by specific data, because intrasac pressures were not measured. However, in a recent study on in vitro aneurysm models, the absence of an outflow was associated with increased mean sac pressure, similar to the mean aortic pressure.³⁸ This finding supports our data demonstrating that the absence of an outflow, and therefore longer washout times, is associated with higher enlargement rate caused by higher intrasac pressure.

AAA enlargement rate was significantly different among different types of stent-grafts. Devices such as Excluder and AneuRx seemed to be associated with a lower enlargement rate, compared to the Talent stent-graft (Medtronic Inc, Minneapolis, Minn).³⁹ However, this result is biased by the limited number of patients and wide range of devices in this series.

CUS represents a noninvasive, fast, well-tolerated, reproducible, and apparently very sensitive imaging modality.^{12,13} In our experience the use of sonographic contrast

agent tends to increase the sensitivity of the ultrasound examination, overcoming some of its limitations and increasing detectability of blood flow within the vessels with no substantial need for complex maneuvers and no discomfort for the patient. However, the required equipment and the contrast agent are highly specific and relatively expensive, patient collaboration is needed, and the examination is still operator dependent. Moreover, CUS seems not to be appropriate for the evaluation of other parameters such as graft anchorage and integrity and aneurysm morphologic changes, for which CTA remains the first-option imaging modality.⁴⁰

The main limitation of our study is represented by the low number of patients included. Larger series would be required, possibly including a wider range of stent-grafts. Moreover, because follow-up was inhomogeneous, a rate of volume enlargement was used, and it was arbitrarily considered significant when ≥ 1 mL/mo, on the basis of the mean rate observed in our series (1.1 mL/mo); in fact, to our knowledge, there are no available data regarding this issue. However, we did not consider the interobserver variability in volume measurements,²⁹⁻³² which could bias our results.

Finally, we were not able to demonstrate the role of our findings in patient management, although we believe that further confirmation of our results might influence treatment planning of type II lumbar endoleak. In fact, because type II lumbar endoleaks differ in terms of flow hemodynamics and effects on the aneurysm sac, preprocedural systematic embolization of the lumbar arteries would not have a clinical rationale.⁴¹ Although the causes of these differences are still unknown, our results point out the usefulness of CUS to rapidly assess the type of lumbar endoleak, identifying the wash-in and washout times, recognizing patients who might benefit from endoleak treatment.

In conclusion, although larger series are required, type II lumbar endoleaks show different hemodynamic features at CUS, which might influence rate of aneurysm enlargement, addressing the need for treatment. Therefore, CUS might represent a valid tool in the decision making and treatment planning of selected patients after EVAR.

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