Treatment of type II endoleak with a transcatheter transcaval approach: Results at 1-year follow-up

Giancarlo Mansueto, MD,[a] **Daniela Cenzi, MD,**[a] **Alberto Scuro, MD,**[b] **Leonardo Gottin, MD,**[c] **Andrea Griso, MD,**[b] **Andrew A. Gumbs, MD,**[d] and **Roberto Pozzi Mucelli, MD,**[a] *Verona, Italy; and New York, NY*

Purpose: **This study assessed the feasibility and mid-term outcomes in the treatment of type II endoleak using transcatheter transcaval embolization (TTE).**

Methods: **During an 8-month period, 12 patients underwent TTE. After direct transcaval puncture of the aneurysm sac, embolization was performed by injecting thrombin and placing coils. Systemic and intrasac pressures were recorded throughout the entire procedure. Computed tomography (CT) scans were performed at 24 hours, 30 days, 6 months, and 1 year after TTE to evaluate endoleaks and changes in sac diameter. Technical success was defined as the feasibility of the procedure; clinical success was defined as no evidence of leaks during the follow-up evaluation.**

Results: **TTE was feasible in 11 of 12 patients (technical success 92%). The mean systemic pressure was 117 mm Hg. The mean intrasac pressure before embolization was 75 mm Hg (range, 39 to 125 mm Hg), 16.5 mm Hg (range, 7 to 40 mm Hg) in 10 patients after embolization, and it increased in one patient. CT scans at 24 hours showed stable contrast medium inside the sac in 10 patients. Only minor complications were observed during follow-up. At the 1-year follow-up, no recurrence of leaks was noted, and sac diameter was reduced in 10 of 11 patients. As a result, TTE clinical success was obtained in 10 (83%) of 12 patients.**

Conclusion: **TTE appears to be a feasible technique for the complete exclusion of type II endoleaks. Technical and clinical successes are comparable with other treatment strategies, and TTE should be considered an alternative to direct translumbar puncture of the aneurysm sac. (J Vasc Surg 2007;45:1120-7.)**

The endovascular repair of abdominal aortic aneurysms (EVAR) may be complicated by the incomplete exclusion of blood flow to the aneurysm sac. This complication has been defined as endoleak by White et al.[1] The incomplete sealing off of the graft to the native vascular system is defined as type I endoleak. Leaks related to fabric tears, graft disconnection, or disintegration are designated as type III.[2-4] Type I and III endoleaks lead to direct arterial flow into the aneurysm sac and are considered technical and clinical failures of EVAR treatment. Endovascular or surgical treatment is mandatory in these conditions.[3,4]

Notably, when an EVAR is successful, as defined as complete sealing at the attachment sites, blood flow into the aneurysm sac can still occur. The reperfusion is usually due to the patency of the collateral branches, such as from the inferior mesenteric artery (IMA) or the lumbar arteries, or both. This condition is known as a type II endoleak.[2-4]

The management of patients with type II endoleak is a source of continuing debate in the literature. Even though type II endoleak can lead to the late rupture of the aneurysm sac and resultant clinical failure of endovascular treatment, there is currently no consensus on the best treatment strategy.[5] Transcatheter transcaval embolization (TTE) was reported as a technique for the treatment of type II endoleaks, suggesting superior management of all steps in the embolization procedure compared with the direct translumbar approach.[6] In this prospective study, we evaluated the feasibility and mid-term results in 12 consecutive patients who underwent this approach.

From the Departments of Morphological and Biomedical Sciences—Radiology Institute,[a] Vascular Surgery,[b] and Anesthesiology and Surgery—Intensive Care Unit,[c] University of Verona; and Department of Surgery, New York Presbyterian, and the University Hospitals of Columbia and Cornell.[d]

Competition of interest: none.

Reprint requests: Giancarlo Mansueto, Department of Morphological and Biomedical Sciences–Institute of Radiology, University Hospital "GB Rossi," Piazza LA Scuro 10, 37134 Verona, Italy (e-mail: giancarlo.mansueto@univr.it).

0741-5214/$32.00

doi:10.1016/j.jvs.2007.01.063

MATERIAL AND METHODS

Patient group and study design. The Institutional Review Board approved our study, and all patients provided informed consent for the procedure. TTE was proposed instead of the direct translumbar approach as the initial treatment of type II endoleak and in cases where transarterial embolization failed. The indications for treatment were the same: evidence of type II endoleak at the 12-month follow-up or a significant increase in aneurysm sac diameter (>5 mm compared with the diameter highlighted at a previous examination) after at least 6 months of follow-up. From September 2004 to April 2005, 12 patients underwent TTE. They previously had undergone EVAR for infrarenal abdominal aortic aneurysms, and a follow-up computed tomography (CT) scan showed type II endoleak, or at least endotension.

The study population consisted of 11 men and 1 woman. The mean age ± standard deviation was 79 ± 5.3 years (range, 68 to 85 years). Patient clinical characteristics are presented in Table I. Two patients had been treated with Excluder stent grafts (W. L. Gore & Associates, Flagstaff, Ariz), three with AneuRx grafts (Medtronic, Minneapolis, Minn), and seven with Talent grafts (Medtronic). The mean time between EVAR and TTE was 47 months

Table I. Patient characteristics before transcatheter transcaval embolization

Characteristics	*Patients (n)*
Hypertension	9
Hyperlipidemia	2
Diabetes	2
Chronic renal failure	5
Prior MI/angina	2
Previous cancer surgery	6
Anticoagulant therapy	6
ASA class 1	—
ASA class 2	4
ASA class 3	5
ASA class 4	3

MI, Myocardial infarction; *ASA,* American Society of Anesthesiologists.

(range, 9 to 83 months). The mean maximal aneurysmal diameter before EVAR was 63 mm (range, 50 to 78 mm), whereas on repeat CT scan before TTE it was 71 ± 13 mm (range, 50 to 88 mm) with a mean increase in diameter of 8 mm (range, 0 to 30 mm).

Type II endoleak was clearly demonstrated on CT scan in 9 patients, eight of whom had a significant increase in diameter, and one patient was found to have a large endoleak without a significant increase in diameter for more than 12 months. In three patients, an increase in maximum aneurysm diameter (>5 mm compared with the diameter highlighted at a previous examination) was demonstrated on CT angiography, without evidence of blood flow inside the aneurysm sac. In these three patients, we hypothesized that a very slow flow reperfusion was present that imaging techniques could not visualize.

Before TTE, a selective digital subtraction angiography (DSA) was performed in nine of 12 patients to highlight the presence and source of endoleak. A type II endoleak was demonstrated in four patients: in two patients it was from the inferior mesenteric artery (IMA); whereas in the other two it came from the lumbar arteries. They underwent endovascular treatment consisting of embolizing the origin of the IMA or closing the afferent lumbar branches to the aneurysm sac. Immediate technical success was achieved in only two patients. One had a type II endoleak from the IMA, and the other had a type II endoleak coming from the lumbar arteries. In both of these patients, a recurrence of their leaks was seen on CT scan 30 days after the embolization.

In the first 24 hours after TTE, all patients were observed in the surgical department during which central venous blood pressure, arterial pressure, heart rate, and peripheral oxygen saturation were monitored. They underwent unenhanced CT scan at 24 hours. They were all discharged at 48 hours and were followed up with a clinical examination and CT scan at approximately 1 month, 6 months, and at 1 year. We evaluated the technical and clinical success of these patients. The technical success was defined as the feasibility of transcaval puncture of the aneurysm sac, and the clinical success was defined as the absence of type II endoleak on follow-up examination.

Preoperative imaging. Preoperative CT scan was performed on all patients on a Somatom Plus 4 CT scanner (Siemens, Erlangen, Germany). According to a standard triphasic protocol, helical images were obtained from the celiac artery to the common femoral arteries, both before and after intravenous administration of 120 mL of nonionic contrast medium with a flow rate of 3 mL/s. Before the injection of contrast medium, acquisition parameters for spiral CT were 8-mm collimation, 5-mm reconstruction, and 1.5 pitch. After contrast medium injection, acquisition parameters for spiral CT were 5-mm collimation, 3 mm reconstruction, and 1.5 pitch. Images were obtained in both the arterial and venous phases. The scanning delay for the arterial phase was usually set at 30 seconds and the venous/delayed phase was set at 120 seconds so that late endoleaks could be identified. An experienced radiologist reviewed the images and evaluated the maximum transverse aneurysm diameter and the presence and origin of endoleaks, which were defined as the evidence of contrast enhancement within the aneurysm sac in the postcontrast medium phases.

Preoperative DSA was performed in 9 of 12 patients on a Multistar angiography scanner (Siemens, Erlangen, Germany). After patients were given a local anesthetic (2% lidocaine) and transfemoral arterial percutaneous access was obtained, a 5F pigtail catheter was advanced into the abdominal aorta. Aortography in the anteroposterior projection was performed above the proximal end of the stent graft with a 20-mL bolus injection of contrast at 10 mL/s. A selective angiography of the superior mesenteric artery was then performed by using a properly shaped catheter with a 30-mL bolus injection at 5 mL/s. If no endoleak was demonstrated by selective catheterization, a Simmons 1 catheter (Terumo Europe NV, Leuven, Belgium) was then pulled down into the stent graft just above the flow divider and placed into the contralateral common iliac artery to perform pelvic angiographies in the left and right anterior oblique projections with a 16-mL bolus injection at 4 mL/s.

We concluded the diagnostic DSA by performing selective angiography of the ipsilateral internal iliac artery by using the Simmons 1 catheter and the same bolus injection used for the contralateral common iliac artery. Whenever a type II endoleak was identified, a superselective coil embolization of the afferent vessels was attempted.

Transcatheter transcaval embolization. The procedure was performed in the angiography suite (Multistar) with the patient in a supine position. A percutaneous transfemoral vein approach was chosen in the first seven patients, whereas in the next five patients we preferred percutaneous access via the right jugular vein. We followed a standard technique in almost all of the patients.

A 10F sheath is used to advance the introducer catheter assembly with the curved guiding cannula (William Cook Europe, Bjaeverskov, Denmark) into the inferior caval vein over a guidewire (Fig 1, *A*). The wire is removed and the system oriented to the stent graft. The system direction is verified in the two fluoroscopic orthogonal projections (Fig

Fig 1. Transcaval direct puncture of the aneurysm sac. **A**, A right transjugular percutaneous access is used to place a 10F sheath and a curved guiding cannula into the inferior vena cava. **B,** Anteroposterior projection of cavography and **(C)** lateral projections highlight the system's wedging to caval walls at the selected puncture site. **D,** The aneurysm sac is punctured with the flexible needle.

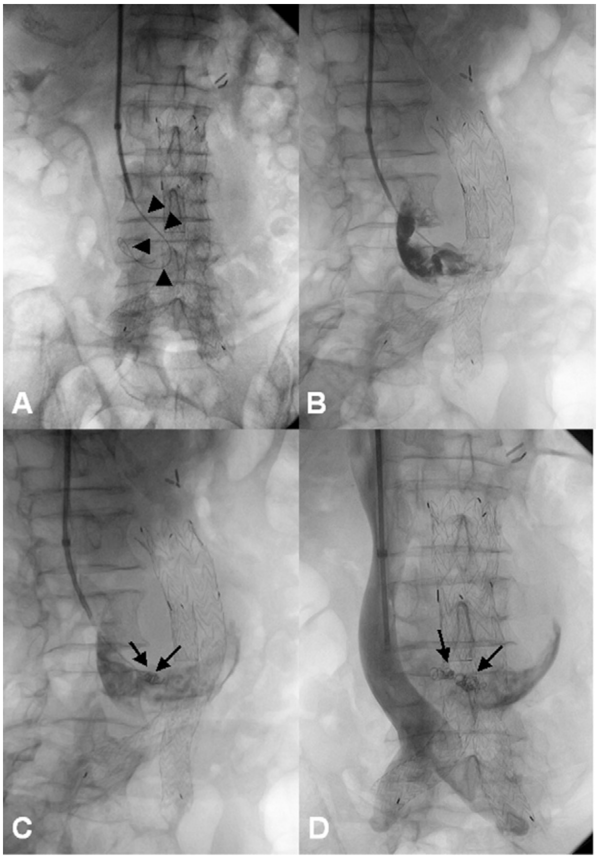

Fig 2. Transcaval transcatheter embolization of the aneurysm sac (same patient as in Fig 1). **A,** A hydrophilic wire is enrolled inside the sac *(arrowheads)*. **B,** A diagnostic angiography is performed, contrast medium fills the aneurysm sac, and the endoleak is highlighted. **C,** Embolization is realized under fluoroscopic guidance by placing coils and filling the sac with thrombin *(arrows)*. **D,** Cavography through the introducer sheath doe not demonstrate lesions at the site of puncture. Coils and stable contrast medium are highlighted inside the sac *(arrows)*.

1, *B* and *C*). The stent graft and wall calcifications significantly help the proper orienting of the aneurysm and the introducer assembly. The shunt set must be completely wedged along the caval wall at a site in which there is a tight adhesion between the caval and aneurysm walls. When there are no calcified walls, ultrasonographic guidance can be helpful.

After checking for the system's wedging against the caval wall with a contrast injection, we introduce the flexible puncture needle and 5F catheter assembly into the curved guiding cannula. The puncture was performed under fluoroscopic control in all cases (Fig 1, *D*) and with additional sonographic guidance in one case. After removing the flexible puncture needle, the success of the puncture is demonstrated by blood dripping from the 5F catheter or the intrasac pressure monitor, or both.

Once inside the sac, a 0.035-inch J hydrophilic guidewire (Terumo Corp, Tokyo, Japan) is advanced and enrolled into the sac (Fig 2, *A*). The placement of the needle/sheath system within the endoleak nidus is not always possible, but the thrombus can be broken into small clots by the guidewire as it enrolls inside the sac. To allow for the advancement of the 5F catheter over the wire into the aneurysm sac, a diagnostic angiography is performed with the catheter just inside the aneurysm. We call this *saccography*, and it is done to highlight the endoleak and to help visualize the feeding vessels (Fig 2, *B*).

Embolization is done regardless of where the type II endoleak nidus is by using thrombin (Tissucol Duo Immuno, Baxter, Rome, Italy) because it can spread inside the aneurysm sac and fill it. Metallic MReye Embolization Coils (William Cook, Bjaeverskov, Denmark) were used with thrombin in patients with large endoleaks, with the catheter tip placed directly inside the nidus. The embolization is performed under fluoroscopic guidance and with monitoring of the intrasac pressure (Fig 2, *C*).

Thrombin is injected at 1-mL boluses. After first bolus injection, we wait 3 minutes. Another bolus of thrombin is

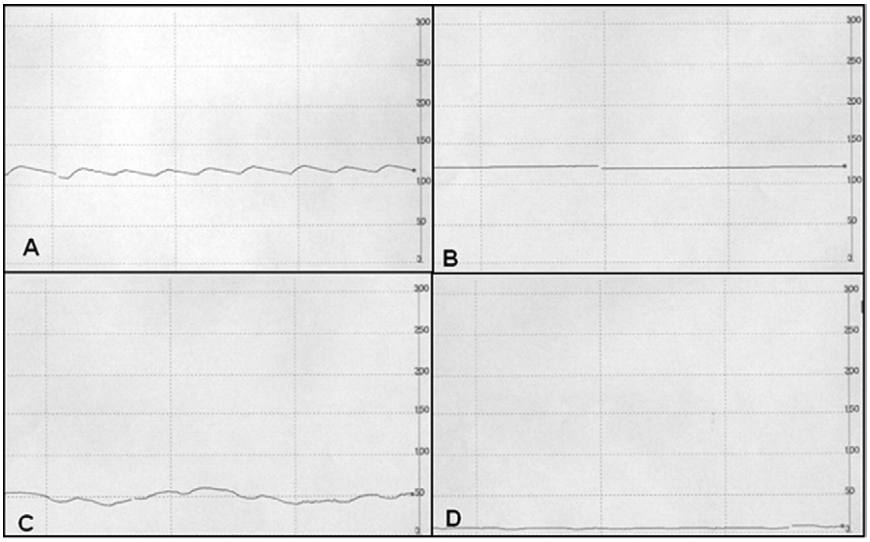

Fig 3. Intrasac pressure monitoring during transcatheter transcaval embolization. **A,** A systolic/diastolic wave is demonstrated inside the sac (122/118 mm Hg). **B,** After approximately 3 minutes from the injection of 1 mL thrombin, the waveform disappeared, with a stable high pressure (121 mm Hg). After another 2 mL of thrombin, waiting 3 minutes after every single injection before remeasuring pressure, a progressive decrease in pressure is observed. **C,** Pressure is about 48 mm Hg after 2 mL thrombin, and **(D)** 7 mm Hg after 3 mL thrombin.

injected if there is still evidence of blood dripping from the 5F catheter or if a systolic-diastolic pressure still exists in the aneurysm sac. We wait an additional 3 minutes and remeasure the intrasac pressure, after washing the 5F catheter with few milliliters of saline solution.

Intrasac embolization is stopped only when there is no more evidence of flow inside the aneurysm sac. This was defined as no evidence of blood dripping from the 5F catheter and that contrast is stable inside the sac. Stable intrasac pressure within an aneurysm that had a previous systolic-diastolic waveform or its reduction by a minimum of 50 mm Hg, or both, was considered as further evidence of successful embolization. At the end of all procedures, we check for any lesions at the site of caval puncture by performing a cavography through the sheath before removing the venous sheath (Fig 2, D).

Intrasac pressure measurement. Intrasac pressure measurements are performed by using a pressure transducer for invasive pressure monitoring (Edwards Lifesciences, Irvine, Calif) connected to a multiparametric monitor Datex Engtrom S/5 (Datex Ohmeda, Helsinki, Finland). After connection of the 5F catheter to the transducer and before thrombin injection, intrasac pressures was recorded in all of the patients, indicating that blood flow was present inside the sac. Intrasac pressure was recorded after the performance of each thrombin injection, usually 3 minutes after the injection (Fig 3). Both before and after embolization, pressure measurements was recorded in different places, even though the range of positions for the catheter tip was small. A stable intrasac pressure in an aneurysm with a previous systolic-diastolic waveform pressure or a reduction of at least 50 mm Hg, or both, were considered as signs of thrombosis of endoleak, signifying the abolition of arterial blood flow inside the endoleak itself.

Postoperative surveillance. Immediate imaging follow-up for all patients undergoing TTE consisted of an unenhanced CT scan at 24 hours to document the immediate success of embolization and to identify any evidence of complications related to the procedure. Noncontrast CT angiography was performed on a Somatom Plus 4 CT scanner. Acquisition parameters used were the same in preoperative unenhanced phase spiral CT scans: 8-mm collimation, 5-mm reconstruction, and 1.5 pitch. The evidence of stable contrast medium inside the aneurysm sac was defined as an immediate success of embolization.

After discharge, follow-up for all patients consisted of clinical examination and CT scans at approximately 1 month, 6 months, and at 1 year. Patients underwent triphasic CT imaging, with the same standard protocol followed as for the preoperative imaging. An experienced radiologist reviewed the images to evaluate maximum transverse diameter of the aneurysm and identify the presence and etiology of endoleaks. The clinical success was identified as the absence of evidence of recurrent endoleak during follow-up. The maximum aneurysm diameter was recorded prospectively and compared with the diameter before TTE in a dedicated workstation (Leonardo, Siemens, Erlangen, Germany) by comparing images at the same level using a multitask slice-by-slice method. Any decrease in diameter of >2 mm was considered significant, as was an initial decrease in diameter.

Table II. Intrasac pressure before and after transcatheter transcaval embolization

	Intrasac pressure	
Patients	*Before treatment*	*After treatment*
1	140/80	20
2	60	10
3	62	12
4	62	10
5	66	12
6	40/38	36
7	45/40	8
8	130/120	150
9	115/105	40
10	72/60	7
11	90	5

RESULTS

Before TTE, four patients had previously undergone transarterial embolization, but immediate technical success was achieved in only two patients. In both cases there was a recurrence of leak on CT scan at 30-days. The transcaval direct puncture of the aneurysm sac was feasible in 11 of 12 patients (technical success 92%). The transcaval puncture failed in only one case. In this patient, there was not a tight adhesion between the caval and aneurysm walls. Thus, the patient had to undergo direct translumbar embolization of the aneurysm sac under CT guidance, which was completed successfully. Before TTE, the mean arterial pressure was 117 mm Hg, and no significant changes were observed during and after the procedure.

Whenever the puncture of the aneurysm sac was feasible, intrasac pressures were recorded (Table II). No significant changes in pressure related to catheter position were observed before or after embolization. Before treatment, mean intrasac pressure was 75 ± 31.5 mm Hg (range, 39 to 125 mm Hg), with evidence of a systolic-diastolic wave in six of 11 patients. Afferent and efferent branches had been detected in these patients on preoperative imaging. In the other 5 patients, stable high pressure was recorded inside the aneurysm sac, with evidence of blood dripping from the 5F catheter. Three patients had a previous diagnosis of endotension, whereas in the other two, a type II endoleak had been demonstrated without any evidence of feeding collateral vessels.

After TTE, absence of a systolic-diastolic waveform and a significant reduction in intrasac pressure were observed in 10 of 11 patients, with a mean intrasac pressure of 16.5 ± 12.2 mm Hg (range, 7 to 40 mm) and a mean pressure reduction of 58.5 mm Hg. In one patient, the pressure inside the sac increased throughout the procedure. It was 150 mm Hg after the injection of 5 mL of thrombin, 20 mm Hg more than before starting TTE, therefore, the procedure was stopped.

We injected between 2 and 5 mL of thrombin to embolize the endoleak. The mean time to complete TTE was 30 minutes.

At unenhanced CT scan 24 hours after treatment, contrast medium was still seen inside the sac in 10 of 11 patients (Fig 4, *A*) with an immediate and stable reduction of intrasac pressure, whereas no stable contrast medium was demonstrated inside the sac in the patient with the increase in intrasac pressure after TTE.

During the follow-up period at 1 month, we observed complications in only one (8%) of 12 patients. A thrombophlebitis occurred in the common femoral vein that extended into the distal tract of the external iliac vein. This was related to the percutaneous puncture and was successfully managed with medical treatment (cefotaxime and heparin), without any sequelae.

At 1 and 6 months' follow-up, CT scan highlighted the recurrence of type II endoleak in only one patient successfully treated by TTE. At CT scan 1 year after TTE, no type II endoleak was found in 10 of 11 patients; furthermore, the maximum aneurysm diameter was reduced in 10 patients, with a mean diameter of 68 mm (range, 50 to 88 mm) and a mean reduction of 3 mm (range, 2 to 10 mm; Fig 4, *B*). As a result, clinical success of TTE was obtained in 10 (83%) of 12 patients.

DISCUSSION

The mechanism and behavior of type II endoleak development is unknown. Some collateral endoleaks have been shown to thrombose if left alone, but others will develop in patients with initially negative CT scans.[1,2,7,8] As a result, the management of type II endoleaks is still controversial. Some authors have suggested repair when type II endoleaks persist at 12 months after EVAR, despite a stable aneurysm sac diameter. This is because the collateral vessels transmit arterial pressure to the aneurysm sac, placing the patient at increased risk for aneurysm rupture.[5,9,10]

Others believe that treatment should be performed only when the aneurysm increases in size, even if there is no evidence of type II endoleak.[9,11,12] This condition is defined as endotension.[4,7,9,13] It may be due to very slow retrograde flow inside the sac or to the development of a hygroma. Because hygromas have become rare after the placement of the latest generation of stent grafts, we currently believe that endotension may be due to an unknown type II endoleak.

It is our view that type II endoleaks should be treated when their persistence is confirmed after 12 months of their development or when a significant increase in aneurysm sac diameter is observed after at least 6 months.

Different treatments have been proposed for embolization of type II endoleaks.[4,6,8,14-17] The more widespread treatments are transcatheter transarterial and percutaneous translumbar approaches rather than laparoscopic tying of collateral branches. The transcatheter transarterial approach with selective catheterization of the arch of Riolan and of the IMA permits embolization of the aneurysm sac and the endovascular exclusion of the origin of the IMA. This can be achieved by filling the aneurysm sac with embolization material. Selective embolization with coils

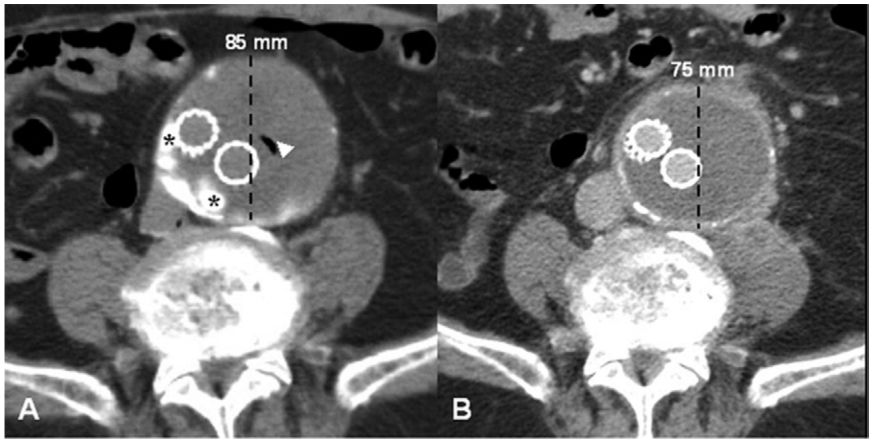

Fig 4. A computed tomography (CT) scan 24 hours after transcatheter transcaval embolization (TEE) and at 12 months. **A,** Unenhanced CT scan 24-hours after the treatment highlights stable contrast *(asterisks)* and gas bubbles *(arrowhead)* inside the aneurysm sac, whose main diameter is 85 mm. That confirms that the aneurysm sac was reached through the transcaval puncture and also that there was an immediate success of the embolization. **B,** At 12-months, an enhanced delayed-phase CT scan demonstrates no recurrence of endoleaks. Furthermore, the main diameter of the aneurysm sac is reduced to 75 mm. TTE clinical success is thus achieved. Of interest is that the sac walls are thicker and hyperdense. This finding may be related to the development of a stable thrombus and consequent fibrosis.

placed at the origin of the IMA, as well as the laparoscopic surgical tying of the IMA, may not be a sufficient treatment for type II endoleaks because some collateral branches may lead to reperfusion of the aneurysm.

As reported by Baum et al,[8] the embolization of a single feeding artery is ineffective because additional flow is recruited from nearby vessels, similar to what occurs with arterial malformations. Therefore, when a type II endoleak is due to multiple lumbar or hypogastric arteries, or both, transcatheter selective embolization can be a challenge.[18] In such cases, the direct translumbar puncture of the aneurysm sac under fluoroscopic or CT guidance can be attempted as an alternative treatment modality.[8,9,14,19] The placement of the needle/sheath system within the endoleak cavity can thrombose the endoleak by embolizing the central nidus.[8]

A comparison of transarterial and translumbar techniques demonstrates more recurrences of type II endoleaks after transcatheter embolization. As a result, direct translumbar embolization has become the best treatment option.[8] A translumbar transcaval approach with CT guidance for right-sided aneurysms[15] and a transabdominal approach with ultrasonographic guidance[16] have also been reported as alternatives to translumbar direct puncture of the aneurysm sac.

We proposed TTE for treatment of type II endoleaks. Compared with transcatheter transarterial embolization, it is less time consuming and can be performed in patients with lumbar endoleaks in which no direct lumbar collateral route exists and in cases of transarterial failure. This point is highlighted in our series: four patients underwent unsuccessful transarterial procedures before their TTE. TTE also offers additional advantages over translumbar embolization. The entire procedure is performed in the angiography suite in the supine position, under fluoroscopic control; as a result, patient compliance with the treatment is improved.

The inferior vena cava is only punctured in one site instead of in two different points, as occurs with the translumbar transcaval approach.[14,15] Therefore, a lower risk of retroperitoneal hemorrhage is expected. The percutaneous transfemoral/transjugular access led to a reduction in operator's radiant dose, and the entire procedure is possible after transcatheter embolization techniques (Fig 2).

Currently, we prefer transjugular percutaneous access because it allows a better orientation of the curved needle/sheath system to the site of puncture. As a result, the embolization is performed in conditions of safety, under complete sterility, and enables a choice of materials. In our opinion, thrombin is an optimal embolizing agent: when injected in 1-mL boluses, it can spread inside the sac lumen through the thrombus. Thrombus inside an endoleak sac is made of a somewhat gelatinous material and in close proximity to the leak it appears to be recently deposited and may be subject to ongoing remodeling with repeated cycles of resorption and deposition. When the endoleak is large or there is clear evidence of efferent vessels, or both, embolization should be done by positioning coils inside the aneurysm sac and then injecting thrombin.

The demonstration of stable contrast inside the sac on unenhanced CT scan at 24 hours is considered as technical success as well as proof of immediate clinical success. Intraprocedural evidence of technical success can be obtained by intrasac pressure monitoring by placing a 5F catheter directly inside the aneurysm sac. Few reports have been published about the relationship between intrasac pressure and endoleak changes.[17,20] Intrasac pressure monitoring should still be considered as a work in progress.

A correlation between imaging results and spectral Doppler ultrasound waveform analysis of type II endoleaks has been found and is thought to be predictive of endoleak behavior.[21-26] If flow velocities and resistances are associated with the dynamics of development and resolution of type II endoleaks, intrasac pressure changes seem to be an expression of their evolution. Therefore, the disappearance of the pressure waveforms and a reduction in intrasac pressure can be considered as a marker of successful embolization (Fig 3).

Baum et al[17] lowered endoleak-induced intra-aneurysmal sac pressure to <30 mm Hg after successful embolization of patent arterial branches. We demonstrated that in patients treated successfully, the disappearance of systolic-diastolic waveforms or the reduction of intrasac pressure had been observed during TTE. If these promising results are confirmed in a larger number of patients, intrasac pressure measurement might become an effective method to monitor the natural history of type II endoleaks.

Nevertheless, we have to keep in mind that measurement may be compromised by several pitfalls. The disappearance of the pressure waveform can be mainly due to the exclusion of the endoleak by blood flow but also by contact of the catheter tip with the aneurysmal wall, even in instances where there is persistent blood flow or the presence of thrombin inside the catheter. To reduce the impact of these two pitfalls on the reliability of the procedure, we rotated the catheter tip to record the presence of residual blood flow and we washed the catheter with saline after every thrombin injection.

The TTE is viable in almost all patients. As for translumbar embolization of the aneurysm sac, direct puncture is quite difficult when the aneurysm sac diameter is small and is associated with an increased risk of stent graft puncture. Furthermore, the transcatheter transcaval approach should be carefully evaluated when the aneurysm sac is completely left-sided to demonstrate a tight adhesion between the caval and aneurysmal wall. In fact, the asymmetrical increase of the aneurysm sac might be associated with a decrease of adhesion between the aneurysm and caval walls, so that TTE technical success might be compromised. In our experience, the puncture failed in only one patient because of the incomplete adhesion between the caval vein and the aneurysm sac. A careful re-evaluation of CT images highlighted that the aneurysm sac was almost completely left-sided and that there was a minimum distance of 3 mm between the caval vein and aneurysm sac walls. As a consequence, we think that in such cases direct translumbar puncture of the aneurysm sac should be considered as the first approach proposed for embolization of type II endoleaks.

The only complication we reported in our series was thrombophlebitis at the site of femoral percutaneous venous access. Other possible complications related to TTE are retroperitoneal hemorrhage and caval-aortic fistulas. If there is a tight adhesion between the caval walls and the aneurysm sac, the development of a retroperitoneal hemorrhage should rarely occur. It is also worth noticing that TTE requires a thin needle cannula and a 5F catheter, which are quite small in caliber and cause only small holes in the caval walls. Even though diameters of these devices are a little larger than the 18G to 20G needles used for direct translumbar puncture, the caval hole is expected to close immediately after the removal of the 5F catheter. Like Stavropoulos et al,[15] we did not report any caval-aortic fistulas in our study.

A cavography at the end of the procedure, as well as CT scan at 24 hours after treatment can check for both of these complications and treatment can be undertaken by placement of coils or a device like those to close inter-atrial cardiac shunts. According to our experience, TTE appears quite safe and less invasive than the translumbar transcaval approach;[15]; nevertheless, further studies are needed to confirm the small rate of clinical complications.

The current study has certain limitations. We have already mentioned some bias related to our technique of measuring intrasac pressure. The small number of patients treated cannot support a certain conclusion of success or complication rates. Nonetheless, our study population is comparable with other study groups reported in the literature of patients with persistent type II endoleak who required treatment.[8,9,14,15,18,27-29]

We did not prospectively analyze different approaches for embolization of a type II endoleak: we just enrolled all consecutive patients with a persistent endoleak who required treatment according to the criteria adopted at our center. We then compared technical and clinical success with the results reported both for transarterial transcatheter embolization and translumbar direct puncture of the aneurysm sac (Table III). Our aim was to consider if the TTE was at least as feasible and successful as other approaches in common clinical practice.

CONCLUSION

Currently, no treatment is considered the gold standard for type II endoleak management; as a result, a tailored treatment should be preferred. Different approaches have been proposed for embolization of type II endoleaks.[4,6,8,14-17] We evaluated TTE and it appears to be a promising and feasible technique for thrombosis of type II endoleaks, especially if a transjugular percutaneous access is used. According to the results of our study, TTE technical and clinical success rates are comparable with other treatment strategies (Table III), and it can be proposed as the first-line therapy in most patients or at least as an alternative to translumbar direct puncture of the aneurysm sac.

AUTHOR CONTRIBUTIONS

Conception and design: GM, DC
Analysis and interpretation: GM, DC, AAG, RP
Data collection: GM, DC, AS, AG
Writing the article: GM, DC, LG, AAG, RP
Critical revision of the article: GM, RP, LG, AAG, RP
Final approval of the article: GM, RP, AS, LG, RP
Statistical analysis: DC, AG, AAG
Obtained funding: GM, AS, RP

Table III. Comparison of technical and clinical outcomes of different treatments of type II endoleaks

First author (year)	*Patients*	*Embolization treatment*	*Success (%)*	
			Technical	*Clinical*
Baum,[10] (2000)	8	Transarterial	87.5	NS
Baum[14] (2001)	7	Translumbar	100	100*
Haulon[29] (2001)	18	Transarterial	94.4	89
Solis[27] (2002)	10	Transarterial	90	40
Baum[8] (2002)	20	Transarterial	90	20
	13	Translumbar	100	92
Kasirajan[28] (2003)	8	Transarterial	75	67
Stavropoulos[15] (2003)	9	Translumbar transcaval	100	67
Becquemin[18] (2004)	33	Transarterial	66	NS
This report (2006)	12	Transcatheter transcaval	92	83

NS, Not specified.
*A patient developed a new proximal attachment site leak.

Overall responsibility: GM, DC, AS, AAG, RP
GM and DC contributed equally to this work.

REFERENCES

1. White GH, Yu W, May J. Endoleak–a proposed new terminology to describe incomplete aneurysm exclusion by endoluminal graft. J Endovasc Surg 1996;3:124-5.
2. White GH, Yu W, May J, Chaufour X, Stephen MS. Endoleak as a complication of endoluminal grafting of abdominal aortic aneurysms: classification, incidence, diagnosis and management. J Endovasc Surg 1997; 4:152-68.
3. White GH, May J, Waugh RC, Chaufour X, Yu W. Type III and type IV endoleak: toward a complete definition of blood flow in the sac after endoluminal AAA repair. J Endovasc Surg 1998;5:305-9.
4. Veith FJ, Baum RA, Ohki T, Amor M, Adiseshiah M, Blankensteijn JD, et al. Nature and significance of endoleaks and endotension: summary of opinions expressed in an international conference. J Vasc Surg 2002;35:1029-35.
5. Harris PL, Vallabhaneni SR, Desgranges P, Becquemin JP, van Marrewijk C, Laheij RJ. Incidence and risk factors of late rupture, conversion, and death after endovascular repair of infrarenal aortic aneurysms: the EUROSTAR experience. J Vasc Surg 2000;32:739-49.
6. Mansueto G, Cenzi D, D'Onofrio M, Petrella E, Gumbs AA, Pozzi Mucelli R. Treatment of type II endoleaks after endovascular repair of abdominal aortic aneurysms: transcaval approach. Cardiov Inter Radiol 2005;28:641-5.
7. Gorham TJ, Taylor J, Raptis S. Endovascular treatment of abdominal aortic aneurysm. Br J Surg 2004;91:815-27.
8. Baum RA, Carpenter JP, Golden MA et al. Treatment of type 2 endoleaks after endovascular repair of abdominal aortic aneurysms: comparison of transarterial and translumbar techniques. J Vasc Surg 2002;35:23-9.
9. Baum RA, Stavropoulos SW, Fairman RM, Carpenter JP. Endoleaks after endovascular repair of abdominal aortic aneurysms. J Vasc Interv Radiol 2003;14:1111-7.
10. Baum RA, Carpenter JP, Tuite CM, Velazquez OC, Soulen MC, Barker CF, et al. Diagnosis and treatment of inferior mesenteric arterial endoleaks after endovascular repair of abdominal aortic aneurysms. Radiology 2000;215:409-13.
11. Steinmetz E, Rubin BG, Sanchez LA, Choi ET, Geraghty PJ, Baty J, et al. Type II endoleak after endovascular abdominal aortic aneurysm repair: a conservative approach with selective intervention is safe and cost-effective. J Vasc Surg 2004;39:306-13.
12. van Marrewijk CJ, Fransen G, Laheij RJ, Harris PL, Buth J; EUROSTAR Collaborators. Is a type II endoleak after EVAR a harbinger of risk? Causes and outcome of open conversion and aneurysm rupture during follow-up. Eur J Vasc Endovasc Surg 2004;27:128-37.
13. Gilling-Smith G, Brennan J, Harris P, Bakran A, Guols D McWilliams R. Endotension after endovascular aneurysm repair: definition, classification and strategies for surveillance and intervention. J Endovasc Surg 1999;6:305-7.
14. Baum RA, Cope C, Fairman RM, Carpenter JP. Translumbar embolization of type II endoleaks after endovascular repair of abdominal aortic aneurysms. J Vasc Interv Radiol 2001;12:111-6.
15. Stavropoulos SW, Carpenter JP, Fairman RM, Golden MA, Baum RA. Inferior vena cava traversal for translumbar endoleak enbolization after endovascular abdominal aortic aneurysm repair. J Vasc Interv Radiol 2003;14:1191-4.
16. Ellis PK, Kennedy PT, Collins AJ, Blair PH. The use of direct thrombin injection to treat a type II endoleak following endovascular repair of abdominal aortic aneurysm. Cardiovasc Intervent Radiol 2003;26:482-4.
17. Baum RA, Carpenter JP, Cope C, Golden MA, Velazquez OC, Neschis DG, et al. Aneurysm sac pressure measurements after endovascular repair of abdominal aortic aneurysms. J Vasc Surg 2001;33:32-41.
18. Becquemin JP, Kelley L, Zubilewicz T, Desgranges P, Lapeyre M, Kobeiter H. Outcomes of secondary interventions after abdominal aortic aneurysm endovascular repair. J Vasc Surg 2004;39:298-305.
19. Krueger K, Zaehringer M, Gawenda M, Brunkwall J, Lackner K. Successful treatment of a type II endoleak with percutaneous CT-guided thrombin injection in a patient after endovascular abdominal aortic aneurysm repair. Eur Radiol 2003;13:1748-9.
20. Mehta M, Veith FJ, Ohki T, Lipsitz EC, Cayne NS, Darling RC. Significance of endotension, endoleak and aneurysm pulsatility after endovascular repair. J Vasc Surg 2003;37:842-6.
21. Sato DT, Goff CD, Gregory RT, Robinson KD, Carter KA, Herts BR, et al. Endoleak after aortic stent graft repair: diagnosis by color duplex ultrasound scan versus computed tomography scan. J Vasc Surg 1998;28:657-63.
22. Carter KA, Nelms CR, Bloch PHS, Gregory RT, Parent N, DeMasi. RJ, et al. Doppler waveform assessment of endoleak following endovascular repair of abdominal aortic aneurysm; predictors of endoleak thrombosis. J Vasc Technol 2000;24:119-22.
23. Greenfield AL, Halpern EJ, Bonn J, Wechsler RJ, Kahn MB. Application of Duplex US for characterization of endoleaks in abdominal aortic stent-grafts: report of five cases. Radiology 2002;225:845-51.
24. Parent FN, Meier GH, Godziachvili V, LeSar CJ, Parker FM, Carter KA, et al. The incidence and natural history of type I and II endoleak: a 5 year follow up assessment with color Doppler ultrasound scan. J Vasc Surg 2002;35:474-81.
25. Arko FR, Filis KA, Siedel SA, Drake AR, Fogarty TJ, Zarins CK. Intrasac flow velocities predict sealing of type II endoleaks after endovascular abdominal aortic aneurysm repair. J Vasc Surg 2003;37:8-15.
26. Wright IA, Gordon MK, Buckenham TM. Spectral Doppler characterization of endoleaks following endoluminal abdominal aortic aneurysm repair. ANZ J Surg 2005;75:118-23.
27. Solis MM, Ayerdi J, Babcock GA, Parra JR, McLafferty RB, Gruneiro LA, et al. Mechanism of failure in the treatment of type II endoleak with percutaneous coil embolization. J Vasc Surg 2002;36:485-91.
28. Kasirajan K, Matteson B, Marek JM, Langsfeld M. Technique and results of transfemoral superselective coil embolization of type II lumbar endoleak. J Vasc Surg 2003;38:61-6.
29. Haulon S, Tyazi A, Willoteaux S, Koussa M, Lions C, Beregi JP. Embolization of type II endoleaks after aortic stent-graft implantation: technique and immediate results. J Vasc Surg 2001;34:600-5.

Submitted Oct 27, 2006; accepted Jan 25, 2007.