Trans-caval Endoleak Embolization (TCEE) of Type I and II Endoleaks Occurring after Endovascular Abdominal Aortic Aneurysm Repair (EVAR)

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<th>Poster No.:</th>
<th>C-0809</th>
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<tr>
<td>Congress:</td>
<td>ECR 2013</td>
</tr>
<tr>
<td>Type:</td>
<td>Scientific Exhibit</td>
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<tr>
<td>Authors:</td>
<td>R. Gandini, M. Chiocchi, D. Morosetti, A. Chiaravalloti, G. Loreni, G. Simonetti; Rome/IT</td>
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<tr>
<td>Keywords:</td>
<td>Arteries / Aorta, Interventional vascular, CT-Angiography, Fluoroscopy, Embolisation, Catheters, Stents, Aneurysms</td>
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<tr>
<td>DOI:</td>
<td>10.1594/ecr2013/C-0809</td>
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Purpose

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 (2,3). 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 several collateral branches, such as from the inferior mesenteric artery (IMA), the superior mesenteric artery, the sacral artery and the lumbar arteries. 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). A reasonable treatment strategy in Patient with type II endoleak may be to intervene in cases of increasing aneurysm size or if the endoleak does not resolve spontaneously within 6 months.

Translumbar endoleak embolization (TLEE) has been shown to be more effective than transarterial endoleak embolization (TAEE). An alternative embolization technique is transcaval endoleak embolization (TCEE), which has shown success rates comparable to translumbar and transarterial embolization (6-8). We present our results of treatment of type II endoleak in 26 Patients from January 2007 to April 2011.

Aim of this study is to describe treatment technique, to assess long-term results and to suggest, in accordance to our experience, management of type II endoleak, currently the most controversial in terms of methods of treatment.
Methods and Materials

From January 2007 to April 2011, 26 Patients underwent to a post-EVAR endoleak treatment. The Institutional Review Board at our institution gave full approval and waiver of informed consent for our retrospective study and approved our treatment protocol. Written patient informed consent was obtained from each patient prior to intervention.

Diagnosis of type II endoleak was performed with a follow-up computed tomography angiography (CTA) before the enrollment in the study. The indications for treatment were the same for all Patients: evidence of type II endoleak at 12-month follow up in large aneurysm (>5 cm) with no evidence of significant sac enlargement 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. TCEE was proposed as treatment of type II endoleak. The study population consisted of 18 men and 8 women. The mean age ± standard deviation was 73.6 ± 4.5 years old (range, 68 to 82 years). Time between initial AAA repair and endoleak treatment was 12.3 ± 4.2 months. The mean increase of aneurysm sac diameter was 5.7 ± 4.2 mm. All data for each patient, procedure, and follow up were analyzed retrospectively. Each of the 26 patients had concomitant comorbid medical condition (Table I).

PREOPERATIVE MANAGEMENT

Preoperative assessment included helical computed tomography angiography (CTA), performed by 64 -stations scanner ( LightSpeed VCT, General Electric Medical System, Milwaukee, WI, USA). 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, were used 5-mm collimation, 3 mm reconstruction and 1.5 pitch. Images were obtained in both 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, the presence and origin of endoleaks, defined as the evidence of contrast enhancement within the aneurysm sac, a tight adhesion between the aortic aneurysm and the caval walls in order to evaluate the transcaval approach feasibility, and the "safety space", defined as the area between the aortic aneurism and endoprostesis walls to avoid device damage (Fig.1 A,B).

The procedures were performed into angiographic suite (Allura Philips Healthcare, Best, the Netherlands). We used TCEE technique in 26 Patient (100%).

TECHNIQUE

All procedures were performed in a dedicated angiography suite with the patient in a supine position. A broad-spectrum antibiotic therapy was included in the immediate
preprocedural period, based on penicillin (2g/day) per os for one day, as prophylactic for infection to stentgraft or intrasac necrotic tissue. A percutaneous transfemoral vein approach was performed in all patients. The common femoral vein was punctured previous a local anesthesia (2% lidocaine) and a standard 0.035" J tipped 180 cm long hydrophilic guidewire (Radiofocus, Terumo, Tokyo, Japan) was advanced into the inferior vena cava (IVC). A 10F introducer sheath 40 cm long was placed and advanced into the IVC to give stability and allow a safe puncture. Transcaval puncture of the aneurysm sac was performed with the same technique for all patients, using a dedicated kit for trans-jugular porto-sistemic shunt (Angiodynamics, Queensbury NY, US). The angle of the metallic needle has been handmade modified from 40 degree to 60/70 degree to ensure the correct puncture of the abdominal aortic aneurysm and to reduce bleeding risk due to the more orthogonal access obtained on caval wall. The 8 French transjugular curved catheter and the 5F curved guiding cannula was advanced through the introducer sheath over the guidewire. The system direction is verified correlating the fluoroscopic images with the pre-procedural CT examination.

The stent graft, vertebrae and, when present, aortic wall calcifications help to orienting of the aneurysm and the introducer assembly. When there are no calcified walls, ultrasonographic guidance can be helpful. 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. After checking for the system's wedging against the caval wall with a contrast injection, the Colapinto needle assembly with the 5F curved guiding cannula was advanced through the system and finally the aneurysm sac was punctured according to the landmarks gathered from the pre-procedural CT scan. The 5 Fr cannula with its catheter was introduced under fluoroscopic guidance through the Colapinto needle. The flexible puncture needle was removed, and a 0.035" standard J tipped 180cm long hydrophilic guidewire was slowly advanced into the aneurysm sac until a low resistance path was engaged. This was considered as an indirect sign of the passage of the guidewire into an unthrombosed area, suggestive of the site of the endoleak. The 4 Fr Cobra catheter was then advanced over the guidewire into the area of the presumed endoleak. This diagnostic catheter allowed us to improve the guidewire control within the thrombus in the aneurysm sac. The backflow of blood through the catheter, after removal of the guidewire, confirmed its position within the endoleak. 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.

The contrast media was manually injected with low pressure to visualize the endoleak. It was performed a latero-lateral projection to highlight the lumbar arteries, a right oblique projection for the inferior mesenteric artery and a postero-anterior one for the sacral
artery. In this setting an optimal visualization of the feeding vessels was obtained to make the feeding arteries catheterization manageable. Intrasac pressure measurements were performed during procedure, by using a pressure transducer for invasive pressure monitoring the ACIST System. (Bracco, Milan, Italy).

After connection of the 5F catheter to the transducer and before glue injection, intrasac pressures was recorded in all patients, indicating that blood flow was present inside the sac. Intrasac pressure was recorded after the performance of each glue injection, usually 3 minutes after the injection. 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.

Initially, in nine cases (34.6 %), we performed an unselective embolization of aneurysm sac using a combination of 7-15 platinum fibered coils (diameter range: 6-9 mm), 1ml of Glubran 2 acrylic glue (GEM, Viareggio, Italy) mixed to Lipiodol (volume ratio1:3), in order to allow its fluoroscopic visualization, and thrombin. This procedure was considered in our study an unselective transcaval endoleak embolization (uTCEE).

In 20 cases (76.9 %), during the procedure, the embolization of the feeding branch with fibered coil was performed obtaining a selective occlusion of the in-flow and out-flow vessels, defined as selective TCEE (sTCEE) (Fig. 2 A-I). In only three patients embolization of the sac with acrylic glue mixed to Lipiodol and concomitant coils in the feeding vessel was performed due to the dimension of the aneurysm sac (Fig.4 A-I).

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. In the first 24 hours after endovascular treatment, all patients were observed in the our 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 24 hours from the procedure. A broad-spectrum antibiotic therapy, based on amoxicillin clavulanate (Augmentin; GlaxoSmithKline, Brentford, Middlesex, United Kingdom) at a dose of 2 g/day per os, was administered one day before the procedure as prophylactic for infection to stent-graft or intrasac necrotic tissue.

FOLLOW UP
Follow-up was performed by clinical examination and CTA evaluation at 1, 3, 6, 12 and each following year, by use the same standard acquisition protocol followed as for the preoperative Imaging, in case of no pathology recurrence (Fig. 3 A,B; Fig. 5 A-D).
Fig. 1: Pre-procedural CTA showing II type endoleak from lumbar arteries in axial (A) and sagittal views (B).

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Fig. 2: A right transfemoral percutaneous access is obtained in order to place a 12F sheath and a curved guiding cannula into the inferior vena cava. The Colapinto needle assembly with the 5F curved guiding cannula was advanced through the system until the puncture of the aneurysm sac (A,B,C). The flexible puncture needle was removed, and a 0.035” standard J tipped 180cm long hydrophilic guidewire was slowly advanced into the aneurysm sac following a low resistance path (C). The diagnostic angiography confirmed the presence of the type II endoleak (D, E, F). Embolization was performed under fluoroscopic guidance by placing multiple coils in each feeding collateral vessel (G, H). Postprocedural cavography showed absence of any complications related to caval wall perforation (I).

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**Fig. 3:** Fig. 3: Post-procedural CTA showing in axial view (A) and MPR reconstruction (B) the successful embolization.

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Fig. 4: Through the right transfemoral percutaneous access the Colapinto needle was advanced and used in order to puncture the aneurysm sac (A, B). A 0.035" standard J tipped 180cm long hydrophilic guidewire was gently advanced into the aneurysm sac (C) and a following diagnostic angiography was performed confirming the aneurysm sac and the collateral vessel which caused the type II endoleak (D, E). Embolization of the sac performed with acrylic glue mixed to Lipiodol inside the sac and with coils in the feeding vessel (E-F-G). Postprocedural angiography of the aneurysm sac reported stagnant contrast media inside the sac (H) The following cavography didn't show any complications in the inferior vena cava (I).
**Fig. 5:** Fig. 5: Post-procedural CTA showed, with MPR reconstruction before (A,C) and after contrast media injection (B, D), the successful embolization of the collateral vessel.
Table 1. Preoperative clinical data and risk factor

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<td>Mean Age (years)*</td>
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<td>Cardiac risk factor †</td>
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<tr>
<td>Female</td>
<td>8/26 (30.7%)</td>
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<tr>
<td>Hypertension</td>
<td>24/26 (92.3%)</td>
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<td>Dyslipidemia</td>
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<td>Smoke Habit</td>
<td>7/26 (26.9%)</td>
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<td>Diabetes mellitus</td>
<td>19/26 (73%)</td>
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<td>Coronary artery disease</td>
<td>16/26 (61.5%)</td>
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<td>Comorbidities ‡</td>
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<td>Chronic obstructive pulmonary disease</td>
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<tr>
<td>Chronic renal failure</td>
<td>4/26 (15.3%)</td>
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<td>Cerebrovascular disease</td>
<td>8/26 (30.7%)</td>
</tr>
<tr>
<td>Peripheral obliterating arteriopathy</td>
<td>16/26 (61.5%)</td>
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* Data are means ± standard deviation, with range interval in parentheses
† Data are ratio between characteristics and total population, with percentage in parentheses

Table 1: Table 1. Preoperative clinical data and risk factor
Table 2: Intraprocedural and follow-up results

<table>
<thead>
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<th>uTCEE 9 patients</th>
<th>sTCEE 17 patients</th>
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<tr>
<td>Female sex †</td>
<td>3/9 (30)</td>
<td>7/20 (35)</td>
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<td>Feeding artery embolization†</td>
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<td>2/20 (10)</td>
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<td>Lumbar arteries</td>
<td>4/9 (44.4)</td>
<td>10/20 (20)</td>
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<td>Superior mesenteric artery</td>
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<td>3/20 (15)</td>
<td>ns</td>
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<tr>
<td>Inferior mesenteric artery</td>
<td>1/9 (11.1)</td>
<td>5/20 (25)</td>
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<td>Mean Fluoroscopic time (min)*</td>
<td>15.4 ± 4.1</td>
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<tr>
<td>Embolization material †</td>
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<tr>
<td>Coils</td>
<td>8/9 (88.9)</td>
<td>15/20 (75)</td>
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<td>Coils associated with glue</td>
<td>1/9 (11.1)</td>
<td>5/20 (25)</td>
<td>ns</td>
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<tr>
<td>Technical success†</td>
<td>9/9 (100)</td>
<td>20 (100)</td>
<td>ns</td>
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<tr>
<td>Clinical success†</td>
<td>5/9 (55.5)</td>
<td>20 (100)</td>
<td>ns</td>
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<tr>
<td>Reintervention</td>
<td>4/9 (44.5)</td>
<td>0/20 (0)</td>
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<tr>
<td>Major complications†</td>
<td>1/9 (11.1)</td>
<td>0/20 (0)</td>
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<tr>
<td>Minor complications†</td>
<td>0/9 (0)</td>
<td>2/20 (10)</td>
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* Data are means ± standard deviation, with range interval in parentheses
† Data are ratio between characteristics and total population, with percentage in parentheses

Table 2: Table 2. Intraprocedural and follow-up results

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Results

Technical success of the procedure was defined as the successful deployment of embolization material to the endoleak cavity demonstrated by: - a stable intrasac invasive pressure within an aneurysm that had a previous systolic-diastolic waveform or its reduction by a minimum of 50 mm Hg, or both. - Presence of stable contrast inside the sac at the end of the procedure. Clinical success was defined as absence of endoleak without enlargement of the aneurysm sac on follow-up CTA. A change in aneurysm size ≥ 5 mm was considered significant at 1 year follow-up.

Bleeding that needed blood transfusion, dissections, aneurysm rupture, distal peripheral embolization and death were considered major complications. Minor complication were considered thrombophlebitis and haematoma at site puncture. Afferent and efferent branches had been detected in the whole cohort of patients on preoperative CT imaging.

All patients were matched as comorbidities, intraprocedural and follow-up results and are reported in table I and II (Table I, Table II).

The first nine patients were treated performing unselective transcaval endoleak embolization. Five of them were treated with this technique after a trans-arterial embolization attempt: two after a transarterial embolization failure and three were affected by type II endoleak recurrence. In four patients direct uTCEE was performed. In this cohort of patients the obtained technical success was 100%, however in four patients endoleak recurrence was pointed out at 9.75 ± 3.9 months of the follow-up period. In particular one patient at 3 months arrived at emergency department suffering from severe abdominal pain with a significant aneurysm enlargement and wall rupture diagnosed at CT examination and, therefore, underwent the surgical option with resection and replacing of the abdominal aortic aneurysm. The other three patients underwent a new treatment of uTCEE of the inflow and out-flow vessels, with no type 2 endoleak recurrence in the follow-up period. Mean intrasac pressure before and after uTCEE was respectively 58.6 ± 18.4 mmHg (range: 51-105) with evidence of a systolic-diastolic wave in 1 patient, and 6.5 ± 1.2 mmHg (range: 4-9) after the procedure. The average fluoroscopy time was 15.4 ± 4.1 min (range: 13-41 min). Mean follow-up period of the nine patients treated with the uTCEE was 25.9 ± 11.0 months. In the second period of our experience, we decided to perform directly the sTCEE attempt and this technique was performed and feasible in consecutive 17 cases. Mean intrasac pressure before and after sTCEE was respectively 63.6 ± 15.2 mmHg (range: 43-120) with evidence of a systolic-diastolic wave in 10 patients, and 7.8 ± 2.3 mmHg (range: 5-12) after the procedure. Mean fluoroscopy time was 18.4 ± 5.6 minutes (range: 10-35). Mean follow-up period of all patients treated with the sTCEE was 24.1 ± 7.2 months. No major complications were observed intra-and peri-procedurally. Mean follow-up period of all treated patients resulted 24.1 ± 8.8 months. Afferent and efferent branches had been detected in the whole cohort of patients on preoperative CT imaging.
Before TCEE, the mean arterial pressure was 113 mm Hg, and no significant changes were observed during and after the procedure. No significant changes in pressure related to catheter position were observed before or after embolization. At unenhanced CT scan 24 hours after treatment, contrast medium was still seen inside the sac in all patients. As a minor complication, 16 patients (61.5 %) reported a dull lumbar pain during transcaval puncture solved spontaneously.

During the follow-up period at 3 days, we observed minor complications in only two (7.7%) of 26 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.

The maximum aneurysm diameter was reduced in 22 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). Technical success was 100% in both uTCEE and sTCEE. Clinical success of uTCEE was obtained in 5 (55.6 %) of 9 Patients; clinical success of sTCEE resulted in all patients (100%) with no observed type II endoleak during the follow up period.
Type II endoleak is the most commonly encountered and also the most controversial type of endoleak.

Even though at low-flow, type II endoleaks may prevent thrombosis of the aortic sac and create a potential risk of continued aneurysm expansion and potential rupture. Type II endoleaks account for about 40% of all endoleaks and are reported in 10-25% of EVAR cases at 30 days (4,9).

The best indicator of hemodynamic significance of a type II endoleak is the associated change in the aneurysm sac: if the sac increases in size, higher pressure and a relatively higher risk of long-term rupture are implied. If the sac is stable or decreasing in size, the risk is likely to be less.

Many clinicians assume a "wait and see" approach with regular follow-up when there is no expansion of the aneurysm size, as up to 40% of type II endoleaks will eventually spontaneously thrombose (10,11,12). A more aggressive approach may be warranted in patients with persistent type II endoleak that has not resolved spontaneously within 6 months even in the absence of aneurysm enlargement. Authors indeed assume that collateral vessels can continue to transmit arterial pressure to the aneurysm sac and increase the risk of rupture, hence type II endoleaks should be treated if persisting after 6 months (13).

The strategy of pre-emptively occluding potential sources of collateral inflow has been widely accepted for some branch vessels, such as the hypogastric artery, but remains controversial for patent inferior mesenteric artery and lumbar arteries (14,15). The most common technique of type II endoleak management is TAAEE of the branch vessels with embolization using coils, glue, or thrombin. However, the unsuccessful embolization depended on the catheterization and complete embolization failure of the feeding arteries at the ostium of the aneurysm sac or to a partial embolization of the afferent branches, therefore this technique requires advanced endovascular skills and often is not feasible in all patients because of anatomic limitations. During follow-up, failure and recurrences have been reported in up to 80% of cases since endoleaks often appear again because of multiple communicating vessels or persistent flow through the coils. (7). In such cases, the TLEE under fluoroscopic or CT guidance can be attempted as primary treatment modality (7,16-19, 26-29). This technique, first described by Baum et al. (7), may be used as a primary treatment or after failure of transarterial approach. Baum et al. showed that translumbar embolization was durable with a success rate of > 90% after 8 months. A translumbar transcaval approach with CT guidance for right-sided aneurysms (20) and a transabdominal approach with ultrasonographic guidance(16) have also been reported as alternatives to translumbar direct puncture of the aneurysm sac (21,22).

TCEE represents an endovascular technique which offers several advantages. The entire procedure is performed in the angiography suite in the supine position, under fluoroscopic control; as a result, patient compliance during the treatment is improved. The inferior vena
cava is only punctured in one site instead of in two different points, as occurs with the Trans Lumbar approach (18,20). Therefore, a lower risk of retroperitoneal hemorrhage is expected.

An accurate pre-procedural CTA evaluation and the eventual presence of aortic wall calcifications are usually very useful to the correct orientation of the needle during sac puncture. Thereafter, the placement of the catheter directly inside the aneurysm sac permits intrasac pressure monitoring. The embolization results are followed with usual angiographic controls. The percutaneous venous access can be achieve either with a transfemoral or transjugular puncture. Currently, we prefer transfemoral percutaneous access because, in our opinion, 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.

TCEE was first described by Mansueto et al. using metal coils and/or thrombin injection under intrasac pressure monitoring (23). We believe that the real-time visualization of the distribution of the embolic agent during its injection is essential for determining its optimal required quantity. Thereby, in our series, we preferred the use of Glubran 2 acrylic glue mixed with Lipiodol instead of thrombin alone. Lipiodol enabled the fluoroscopic visualization of the glue. Moreover, we preferred use platinum coil instead metallic coils to reduce attenuation artifacts on follow up imaging studies.

The persistence of glue mixed with Lipiodol or of contrast material, and the absence of dynamic flow within the aneurysm sac indicated the complete resolution of the endoleak. 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 (24,25). The disappearance of the pressure waveforms and a reduction in intrasac pressure can be considered as a marker of successful embolization. We demonstrated that in patients successfully treated, the disappearance of systolic-diastolic waveforms or the reduction of intrasac pressure had been observed during TCEE, obtaining a stable or decrease of sac dimensions and an excellent clinical success rate. 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 glue/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 major limitation to TCEE is that it can be performed only in cases in which the aneurysm sac is adherent to the inferior vena cava and there is sufficient space between the aneurysm's wall and the endograft, condition present in most of the abdominal aortic aneurysms. This information is obtained with CTA which is thereby mandatory for an accurate pre-procedural planning.
In case of no CT evidence of a tight adhesion between cava and aortic wall, we think that direct translumbar/transcaval puncture of the aneurysm sac should be considered as the first approach proposed for embolization of type II endoleaks. The only two minor complications we reported in our series were thrombophlebitis at the site of femoral percutaneous venous access while a major complication, as aneurysm rupture, was reported as clinical result of uTCEE during the follow-up period.

Other possible complications related to TCEE 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. Moreover, in our opinion, we managed to avoid hemorrhage complication thanks to the angle modification of the needle Colapinto tip that allowed an orthogonal approach. It is also worth noticing that TCEE requires a thin needle cannula and a 5F catheter, which are quite small in caliber and causing only small holes in the caval walls. The caval-aortic fistulas represents a rare complication likely due to the significant decrease of the intrasac blood pressure after the embolization. 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. The current study has certain limitations. The small number of treated patients cannot support a certain conclusion of success or complication rates. We observed no significant differences of technical success between uTCEE and sTCEE. The selective procedure requires greater technical skill and a quite longer procedural time; it showed clinical success of 100% compared to the unselective that showed a recurrence rate of 44.4% in our cases.

We believe it is necessary to embolize all visible afferents to prevent new supplies to the sac. In addition, one patient treated with uTCEE showed rupture of the aneurism probably because the afferent vessels continued to transmit arterial pression to the sac, without visible endoleak.

Our aim was to consider if the TCEE as 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. We evaluated sTCEE as primary therapeutic option and it appears to be a promising and feasible technique for thrombosis of type II endoleaks. It does not require high operator skills and can be performed by almost all endovascular specialists in most clinical contexts using a simple C-arch. According to the results of our study, TCEE technical and clinical success rates are comparable with other treatment strategies, and it can be proposed as first line therapy in presence of type II endoleak caused by one or multiple feeding vessels.
References


