Rev Esp Cardiol. 2012;65(1):54–59

Original article

# Endovascular Treatment of Penetrating Aortic Ulcers: Mid-term Follow-up

Gaspar Mestres,* Rubén Rodríguez, Cesar García-Madrid, Xavier Montañà, Marta Burrel, Luis Fernando Cruz, Carlos Flores, and Vicente Riambau

*Sección de Cirugía Vascular, Servicio de Cirugía Cardiovascular, Instituto Clínico del Tórax, Hospital Clínic, Universidad de Barcelona, Barcelona, Spain*

*Article history:*
Received 6 May 2011
Accepted 27 July 2011
Available online 24 October 2011

*Keywords:*
Endovascular procedures
Aortic aneurysm
Aortic ulcer
Aortic syndrome
Thoracic aortic rupture

ABSTRACT

*Introduction and objectives:* The aim of this work is to describe the short- and mid-term results of endovascular treatment of penetrating ulcers in the thoracic aorta.
*Methods:* Between 1998 and 2010, 22 patients with penetrating ulcers in the thoracic aorta received endografts (mean age 69.8 years, 91% male); 50% were indicated for acute aortic syndrome (8 chest pain, 1 aortic rupture, 1 aortobronchial fistula, 1 distal embolization) and 50% for aortic or ulcer diameter. All preoperative, operative and follow-up data were recorded prospectively and met EUROSTAR criteria.
*Results:* Technical success was 100% with no intraoperative deaths or open conversions; 6 (27.3%) required preoperative supraaortic trunk debranching and 1.3 endografts were used per patient; 27.3% developed complications in-hospital and 9.1% required reintervention prior to discharge. Mortality at 30 days was 4.5%. After a mean 52.3 month follow-up (range 0.1-122), cumulative survival free from complications and reinterventions at 100 months was 61.7% and 79.5% respectively, with 95.5% cumulative survival free from aorta- or procedure-related death. We identified no factors significantly related to poor intra- or postoperative clinical course.
*Conclusions:* Endovascular treatment of penetrating aortic ulcers is both possible and effective despite high patient comorbidity. Although a substantial rate of complications and reinterventions can be expected—especially in-hospital—(38.3% and 20.5% respectively at 100 months), long-term mortality is low (4.5%).


## Tratamiento endovascular de las úlceras penetrantes de aorta torácica: seguimiento a medio plazo

*Palabras clave:*
Endovascular
Aneurisma de aorta torácica
Úlcera aórtica
Síndrome aórtico
Rotura aórtica

RESUMEN

*Introducción y objetivos:* Se presentan los resultados a corto y medio plazo del tratamiento endovascular de las úlceras penetrantes de aorta torácica.
*Métodos:* Entre 1998 y 2010, se trató a 22 pacientes con úlceras penetrantes de aorta torácica mediante endoprótesis (media de edad, 69,8 años; el 91% varones). La indicación se realizó por un síndrome aórtico agudo (el 50%: dolor torácico en 8, rotura en 1, fístula aortobronquial en 1, embolización en 1) o por el diámetro aórtico o de la úlcera (50%). Todos los datos preoperatorios, intraoperatorios y de seguimiento fueron analizados prospectivamente siguiendo el protocolo EUROSTAR.
*Resultados:* El éxito técnico fue del 100%, sin muertes ni conversiones intraoperatorias; 6 casos (27,3%) requirieron revascularización previa de troncos supraaórticos, y se utilizaron 1,3 dispositivos por paciente; el 27,3% tuvo complicaciones intrahospitalarias, y el 9,1% requirió reintervenciones antes del alta. La mortalidad acumulada a 30 días fue del 4,5%. Tras un seguimiento medio de 52,3 (0,1-122) meses, la supervivencia acumulada libre de complicaciones y reintervenciones a 100 meses fueron del 61,7 y el 79,5% respectivamente, con un 95,5% de supervivencia acumulada libre de mortalidad relacionada con la aorta o el procedimiento. No se han identificado factores pronósticos significativos de mala evolución clínica intraoperatoria o postoperatoria.
*Conclusiones:* El tratamiento endovascular de las úlceras penetrantes de la aorta torácica es posible y eficaz, a pesar de tratarse de pacientes con elevadas comorbilidades. Aunque se asocia a una considerable tasa de complicaciones y reintervenciones (el 38,3 y el 20,5% a 100 meses), sobre todo intrahospitalarias, la tasa de mortalidad relacionada a largo plazo es baja (4,5%).


* Corresponding author: Sección de Cirugía Vascular, Servicio de Cirugía Cardiovascular, Instituto Clínico del Tórax, Hospital Clínic, Villarroel 170, 08036 Barcelona, Spain.
*E-mail address:* gasparmestres@gmail.com (G. Mestres).

1885-5857/$ – see front matter 
doi:10.1016/j.rec.2011.07.019

## INTRODUCTION

First described by Shennan in 1934,[1] penetrating aortic ulcers are among the least frequent entities causing acute aortic syndrome although asymptomatic cases are increasingly identified in thoracic imaging studies. They are caused by the ulceration of a previous atherosclerotic plaque, penetrating the aortic lumen from the internal elastic lamina to the artery media.[2] Usually affecting elderly patients with previous atherosclerosis, penetrating aortic ulcers are located in the distal aortic arch and descending thoracic aorta (Standford type B)[3] and may be associated with intramural hematoma or other aortic syndromes.[4]

The natural history of aortic ulcers remains unclear and reports on their malignancy are contradictory.[5–8] However, especially in symptomatic patients (associated with acute aortic syndrome), a high risk of progression (to growth, pseudoanaeurysm formation, or aortic dissection) and of aortic rupture (early rupture in ≤38% of symptomatic patients) have been described,[5] associated with high mortality.

Endovascular treatment of penetrating aortic ulcers has shown promising results and mortality has been reduced by 0%-11%[9–11] in the treatment of selected cases–even in patients contraindicated for open surgery, which continues to be associated with high mortality (20%).[12,13] However, mean follow-up studies published to date are short-term –usually <3 years[9,10,13,14]– and offer little information on mid-term behavior. The objective of the present study is to present short- and mid-term results of endovascular treatment of penetrating aortic ulcers in the last 10 years.

**Table 1**
Preoperative Characteristics

| Characteristics of the sample | |
|---|---|
| *Age (years)* | 69.8 (54.6-85.7) |
| *Sex (men/women)* | 20 (90.9)/2 |
| *ASA classification* | |
| 2 | 3 (13.6) |
| 3 | 15 (68.2) |
| 4 | 4 (18.2) |
| *Chronic obstructive pulmonary disease* | 9 (40.9) |
| *Diabetes mellitus* | 9 (40.9) |
| *Tobacco use* | 19 (86.4) |
| *High blood pressure* | 22 (100) |
| *Dyslipidemia* | 13 (59.1) |
| *Kidney failure* | 6 (27.3) |
| *Carotid artery disease* | 1 (4.5) |
| *Coronary disease* | 11 (50) |
| *Previous myocardial infarction* | 4 (18.2) |
| *Previous coronary revascularization (surgical or endovascular)* | 2 (9.1) |
| Congestive heart failure | 0 |
| *Previous thoracotomy* | 2 (9.1) |
| *Previous thoracic aorta procedures* | 1 (4.5) |

ASA, American Society of Anesthesiologists' classification of physical fitness.
Described according to the EUROSTAR protocol[15] and the Society for Vascular Surgery/International Society for Cardiovascular Surgery clinical practice guidelines.[16]
Data are expressed as no. (%) or mean (range).

## METHODS

Initial treatment of aortic ulcers in our center (*Instituto Clínico del Tórax, Hospital Clínic*, Barcelona, Spain) is medical (monitoring, control of blood pressure, and aortic imaging). Only symptomatic patients (acute aortic syndrome) or asymptomatic patients associated with rapidly progressing aortic diameter >60 mm in the ulcerated zone, or ulcer diameter and depth >20 mm,[3] have been referred for surgical repair. Endovascular treatment has been preferred unless the aortic anatomy was unfavorable.

In this context, from June 1999 to August 2010 we treated 22 consecutive patients using aortic endografts for penetrating ulcers in the aortic arch and descending thoracic aorta. Surgery was indicated for acute aortic syndrome in 11 patients (50%): chest pain in 8 (36.4%), aortic rupture in 1 (4.5%), distal embolization with chest pain in 1 (4.5%), and aortobronchial fistula in 1 (4.5%). The remaining 50% were operated because they presented >60 mm aortic diameter in the ulcerated zone (5 patients) or >20 mm ulcer diameter and depth (6 patients). All preoperative, operative, and follow-up data were recorded prospectively and met EUROSTAR criteria.

Most patients were men (90.1%); mean age was 69.8 years with a high prevalence of comorbidity (Table 1). In all patients, diagnosis and aortic measurements were based on preoperative computed tomography angiographic imaging. Seven patients (31.8%) had associated intramural hematoma, usually small in extent and aortic ulcer-related.

In the sample, 27.3% (6 patients) required previous supraaortic trunk debranching to obtain an adequate proximal anchor zone in the aortic arch; 2 patients (9.1%) underwent left subclavian artery occlusion without previous revascularization.

All procedures (22) used general anesthesia, orotracheal intubation, and systemic heparinization in a hybrid theater with radiologic control using Siemens Axiom Artis equipment (Siemens, Tarrytown, United States) of recent vintage, and selective transesophageal echocardiography in patients with limited proximal neck (5 patients, although frequency of use is increasing). We used femoral surgical access to advance the device (except in 4 patients who required iliac approach), and 1 left humeral percutaneous or contralateral femoral approach, and advanced a pigtail catheter to the aortic arch or ascending aorta for intraoperative angiography. We implanted 1.3 devices per patient, requiring strict pharmacologic control of blood pressure at deployment (mean blood pressure <80 mmHg) in proximal anchorage above zones Z1 and Z2 (proximal to the left subclavian artery ostium) and more marked hypotension (mean blood pressure < 50 mmHg, with the aid of an occlusion balloon in inferior vena cava) in the Z0 anchor zones (proximal to the brachiocephalic trunk).[17] We used prophylactic control of intra- and postoperative cerebrospinal fluid pressure in 2 patients (9%). Details of intraoperative data are in Table 2.

### Statistical Analysis

We obtained descriptive pre- and in-hospital data (mean±SD, median [range]) and frequency (no. [%]) with SPSS 15.0. We used Kaplan-Meier survival curves to analyze the time until each event during follow-up (complications, reinterventions, and procedure- or aorta-related mortality); we calculated means and 95% confidence intervals and estimated survival at 100 months. Differences between groups were obtained by Kaplan-Meier survival analysis and evaluated with the Mantel-Cox log-rank test (factors possibly associated with poor prognosis: age, acute aortic syndrome, urgent presentation, presence of associated intramural hematoma, aortic diameter). We considered $P<.05$ statistically significant.

**Table 2**
Intraoperative Data

| Operative data | |
|---|---|
| *Previous supraaortic trunk bypass* | 6 (27.3) |
| Complete bypass (of ascending aorta to the brachiocephalic trunk and left carotid artery) | 1 (4.5) |
| Carotid-carotid plus left subclavian artery bypass | 1 (4.5) |
| Carotid-carotid bypass (and coverage without left subclavian artery revascularization) | 2 (9.1) |
| Carotid-left subclavian artery bypass | 2 (9.1) |
| *Left subclavian artery occlusion without revascularization* | 2 (9.1) |
| *Artery access type* | |
| Femoral | 18 (81.8) |
| Iliac conduit | 4 (18.2) |
| *Endograft type by patient* | |
| Relay (Bolton Medical, Sunrise, Florida, United States) | 9 (40.9) |
| Talent or Valiant (Medtronic AVE, Santa Rosa, California, United States) | 7 (31.8) |
| Excluder or TAG (W.L. Gore & Associates, Flagstaff, Arizona, United States) | 5 (22.7) |
| Endofit (Endomed, Phoenix, Arizona, United States) | 1 (4.5) |
| *Number of endografts* | 1.3 (1-3) |
| *Proximal oversizing, %* | 16.2 (6-48) |
| *Proximal anchor zone** | |
| Z0 | 1 (4.5) |
| Z1 | 3 (13.6) |
| Z2 | 4 (18.2) |
| Z3 | 5 (22.7) |
| Z4 | 9 (40.9) |
| *Procedure duration, min* | 82.9 (40-180) |
| *Technical success* | 22 (100) |
| *Intraoperative complications* | 0 |
| *Intraoperative conversion* | 0 |
| *Intraoperative mortality* | 0 |

Data are expressed as no. (%) or mean (range).
*Endograft proximal anchor zone in the aortic arch: Z0 (proximal to the brachiocephalic trunk ostium), Z1 (between the brachiocephalic trunk and the left primitive carotid artery), Z2 (between the left primitive carotid artery and the left subclavian artery), Z3 (aortic arch distal to the left subclavian artery), and Z4 (descending thoracic aorta).[17]

## RESULTS

All patients were treated successfully with no complications or intraoperative conversions (Figs. 1 and 2). The aortic ulcer was excluded in all cases and all patients with chest pain reported that this disappeared postoperatively. However, during hospitalization 27.3% of the sample presented postoperative complications, related to the procedure or to previous comorbidities (Table 3). Only 1 neurologic complication occurred (paraparesis) in a patient without preoperative cerebrospinal fluid drainage, and this was fully resolved spontaneously in-hospital.

Two in-hospital reinterventions were required: embolization of the left subclavian artery for early type II endoleak in a patient receiving a Talent endograft (Medtronic AVE, Santa Rosa, California, United States) with subclavian coverage without previous revascularization, and surgical repair of a humeral pseudoaneurysm following puncture. Both were successful. One in-hospital death occurred due to septic shock and acute

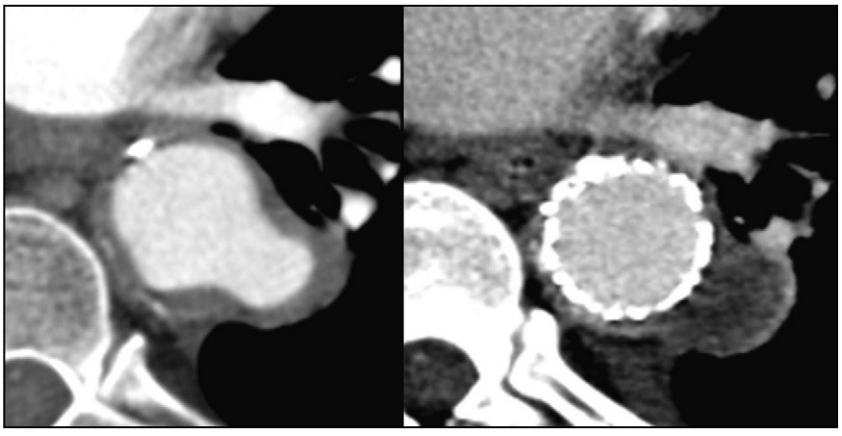

**Figure 1.** Exclusion of an aortic ulcer (pre- and postoperative computed tomographic angiography) in 2 different patients.

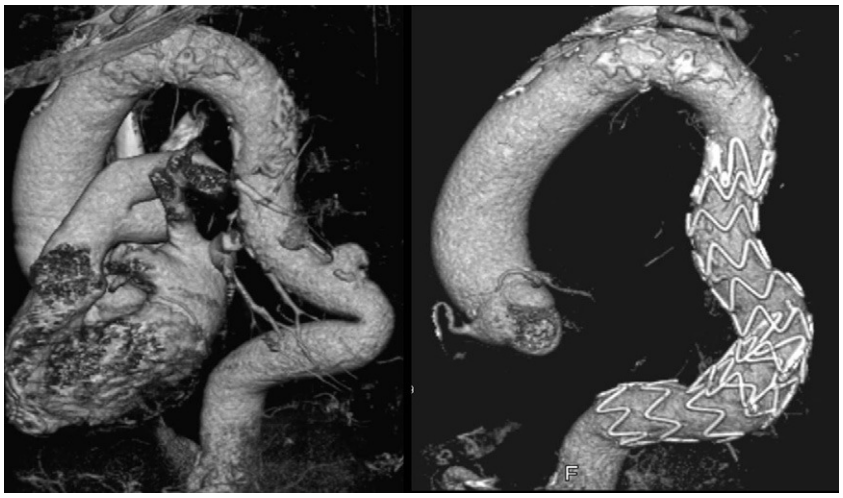

**Figure 2.** Exclusion of an aortic ulcer (pre- and postoperative computed tomographic angiography) in 2 different patients.

respiratory failure caused by postoperative bronchoaspiration. The remaining patients were discharged following a median 3 days hospitalization.

After a mean 52.3 (0.1-122) months follow-up, 2 patients presented new complications (for type I proximal leak and type III leak due to displacement of 2 previous endografts). Both required repeat endovascular reintervention (deployment of new endografts to seal the leak). No conversion to open surgery occurred nor were there any new procedure- or aorta infection-related deaths during follow-up. Two patients required endovascular repair of an abdominal aorta aneurysm and 1 required endovascular repair of a new aortic ulcer proximal to the previous repair (Table 4).

At 100 months follow-up, complication- and reintervention-free cumulative survival were 61.7% and 79.5%, respectively (Fig. 3). Procedure- or aorta-related cumulative mortality was 4.5% (only 1 in-hospital death), and total cumulative mortality from other causes was 38.8%.

We analyzed factors possibly associated with poor prognosis (complications, reinterventions, or procedure- or aorta-related mortality) following treatment of aortic ulcers: age, acute aortic syndrome, emergency presentation, presence of associated intramural hematoma,[4] and aortic diameter.[18] We found no statistically significant relationship in any case ($P>.05$).

## DISCUSSION

Penetrating ulcers of the thoracic aorta present in elderly patients with many comorbidities, and high blood pressure is the most frequent risk factor.[2,19] Advanced age and the frequency of comorbidities have particularly encouraged many groups to treat these lesions by using endovascular surgery, which provides better results, both in early morbidity and mortality, than does open surgery.[9,10,12,13] In fact, the only death in our series was a consequence of bronchoaspiration in an octogenarian, with no direct aortic complication.

Just how malignant the clinical course of penetrating aortic ulcers actually is remains controversial. While some publications report a benign clinical course and low risk of progress, aortic rupture or other serious complications,[6,19] several prospective series find high risk of progress and aortic rupture (≤38% early rupture, above all in symptomatic patients) and even ≤40% of urgent procedures for aortic rupture in patients receiving medical treatment.[4,5]

While initial treatment of aortic ulcers can be considered medical (monitoring, control of blood pressure, and aortic imaging), there is no consensus about indications for surgical or endovascular treatment. Our group indicates endovascular

**Table 3**
In-Hospital Data

| Postoperative morbidity and mortality | |
|---|---|
| *Hospitalization, days* | 3 (3-36) |
| *In-hospital complications* | 6 (27.3) |
| Arterial (pseudoaneurysm following puncture) | 1 (4.5) |
| Neurologic (paraparesis) | 1 (4.5) |
| Cardiac (arrest) | 1 (4.5) |
| Pulmonary | 2 (9.1) |
| Kidney failure | 1 (4.5) |
| Leaks | 1 (4.5) |
| Bleeding | 0 |
| Aortic rupture | 0 |
| Infection of wound | 0 |
| *In-hospital reintervention* | 2 (9.1) |
| *In-hospital mortality* | 1 (4.5) |
| *Mortality at 30 days* | 1 (4.5) |

Data are expressed as no. (%) or median (range).

**Table 4**
Short- and Mid-Term Follow-up Data

| Follow-up | |
|---|---|
| *Follow-up, months* | 52.3 (0.1-122) |
| *Complications* | 2 (9.1) |
| Leaks | 2 (9.1) |
| Migrations | 1 (4.5) |
| Penetrating aortic ulcer growth | 0 |
| Aortic rupture | 0 |
| Endograft collapse or torsion | 0 |
| *Aortic reintervention* | 2 (9.1) |
| Proximal extent | 1 (4.5) |
| Distal extent or interposition | 1 (4.5) |
| Conversion to open surgery | 0 |
| *Lost to follow-up* | 0 |
| *Procedure-related mortality** | 1 (4.5) |
| *Nonprocedure- and nonaorta-related mortality** | 6 (38.8) |

Data are expressed as no. (%) or mean (range).
*Cumulative survival at 100 months follow-up.

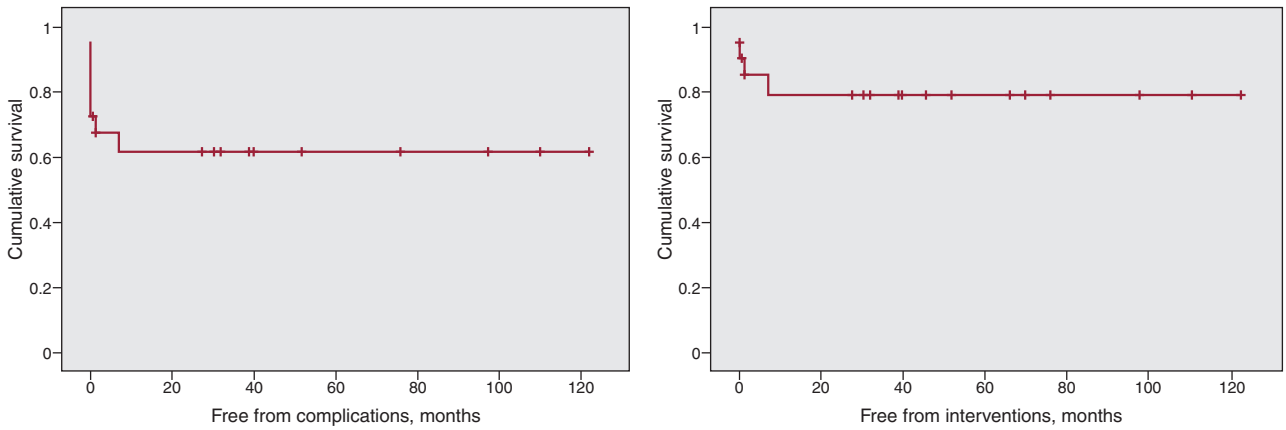


**Figure 3.** Kaplan-Meier survival curves: complication- and reintervention-free cumulative survival during follow-up (61.7% and 79.5%, respectively, at 100 months).

intervention in patients with penetrating aortic ulcer with infection of the aortic arch or descending thoracic aorta who present favorable anatomy and when associated with symptoms (acute aortic syndrome) or in asymptomatic patients associated with rapidly progressing >60 mm aortic diameter in the ulcerated zone, or with >20 mm ulcer diameter and depth.[3]

Results of endovascular treatment in this type of patient have been promising in several series that have achieved high rates of technical success (92%-95%). Moreover, given the small extent of the lesions and the good condition of the proximal and distal aorta, in >90% of cases only 1 device is used,[14,20] results that closely coincide with our series.

Given that many ulcers are located in or near the aortic arch, many previous supraaortic trunk debranching procedures were needed (27.3%), as reported elsewhere (5.3%-29.7%).[4,9] Differences in prevalence may be due to the more aggressive treatment of proximal lesions and the increasing use of carotid-left subclavian artery bypass before occlusion of the latter, in order to minimize the risk of ischemia of the upper extremity, stroke, or spinal chord ischemia.[15,21–23] In fact, our attitude on this issue has changed over time. Before 2006, we performed selective revascularization (dominant left vertebral artery, previous coronary bypass with internal mammary artery, left arm vascular access); since 2006, we have performed systematic revascularization. Moreover, by associating the bypass to ligation or embolization of the proximal subclavian artery, we eliminate the risk of type II leak described in the earlier cases.

As well as supraaortic trunk debranching, dorsal artery occlusion by the endograft can also increase risk of spinal chord ischemia, above all in patients with a deteriorated collateral network (occlusion of subclavian or internal iliac arteries, previous abdominal aortic or thoracic surgery)[15,24,25] and extensive aortic coverage. This can be minimized through intra- and postoperative control of cerebrospinal fluid drainage.[26] However, specifically in aortic ulcers—due to their limited extent and the use of single, usually short devices—we predicted low risk of spinal chord ischemia. Consequently, cerebrospinal fluid control was only used in patients with extensive aortic coverage or previous aortic surgery, 9% of cases.

In our series, the only instance of spinal chord ischemia was in a patient with no left subclavian artery coverage, who underwent short aortic coverage; this patient had not had preoperative cerebrospinal fluid drainage.

As well as low initial and 30-day mortality, several series report short-term mean follow-up (usually <3 years[9,10,13,20]), and larger registries are the exception.[3] Most authors coincide in describing low postoperative and in-hospital mortality (from 0%[13] to 14.6%[9,27]), usually in symptomatic, emergent patients with high comorbidity. Although complication rates during follow-up are significant (from 18%[4] to 30%[14]), these are usually resolved with endovascular techniques, achieving low or nil mortality related to the aorta or conversions to open surgery during follow-up.[9,10,27] The series we present confirms the continued achievement of the good mid-term results described to date. Even though it is not complication- and reintervention-free (38.3% and 20.5%, respectively, at 100 months follow-up), in the mid-term, endovascular surgery associates with high cumulative survival free from aorta- or procedure-related death (95.5% at 100 months).

## CONCLUSIONS

Endovascular treatment of penetrating ulcers of the aortic arch and descending thoracic aorta is possible and effective despite high patient comorbidity. It associates with a considerable rate of complications and reinterventions (38.3% and 20.5% respectively, at 100 months follow-up), above all in-hospital. Nonetheless, long-term aorta- or procedure-related mortality is low (4.5%).

## CONFLICTS OF INTEREST

Dr. Riambau receives consultancy fees from Aptus Endosystems Inc., Bolton Medical, Cordis Corporation, Medtronic Inc., TriVascular Inc., and W.L. Gore & Associates.

## REFERENCES

1. Shennan T. Dissecting aneurysms. Special report series, no. 193. London: Medical Research Council; 1934.
2. Stanson AW, Kazmier FJ, Hollier LH, Edwards WD, Pairolero PC, Sheedy PF, et al. Penetrating atherosclerotic ulcers of the thoracic aorta: natural history and clinicopathologic correlations. Ann Vasc Surg. 1986;1:15–23.
3. Ganaha F, Miller DC, Sugimoto K, Do YS, Minamiguchi H, Saito H, et al. Prognosis of aortic intramural hematoma with and without penetrating atherosclerotic ulcer: a clinical and radiological analysis. Circulation. 2002;106:342–8.
4. Patel HJ, Williams DM, Upchurch Jr GR, Dasika NL, Deeb GM. The challenge of associated intramural hematoma with endovascular repair for penetrating ulcers of the descending thoracic aorta. J Vasc Surg. 2010;51:829–35.
5. Tittle SL, Lynch RJ, Cole PE, Singh HS, Rizzo JA, Kopf GS, et al. Midterm follow-up of penetrating ulcer and intramural hematoma of the aorta. J Thorac Cardiovasc Surg. 2002;123:1051–9.
6. Quint LE, Williams DM, Francis IR, Monaghan HM, Sonnad SS, Patel S, et al. Ulcerlike lesions of the aorta: imaging features and natural history. Radiology. 2001;218:719–23.
7. Cho KR, Stanson AW, Potter DD, Cherry KJ, Schaff HV, Sundt 3rd TM. Penetrating atherosclerotic ulcer of the descending thoracic aorta and arch. J Thorac Cardiovasc Surg. 2004;127:1393–9.

8. Evangelista A, Padilla F, López-Ayerbe J, Calvo F, López-Pérez JM, Sánchez V, et al. Registro Español del Síndrome Aórtico Agudo (RESA). La mejora en el diagnóstico no se refleja en la reducción de la mortalidad. Rev Esp Cardiol. 2009;62:255–62.
9. Botta L, Buttazzi K, Russo V, Parlapiano M, Gostoli V, Di Bartolomeo R, et al. Endovascular repair for penetrating atherosclerotic ulcers of the descending thoracic aorta: early and mid-term results. Ann Thorac Surg. 2008;85:987–92.
10. Brinster DR, Wheatley 3rd GH, Williams J, Ramaiah VG, Diethrich EB, Rodriguez-Lopez JA. Are penetrating aortic ulcers best treated using an endovascular approach? Ann Thorac Surg. 2006;82:1688–91.
11. Gutiérrez-Martín MA, Araji OA, Velázquez CJ, Barquero JM. Tratamiento endovascular de la úlcera aterosclerótica penetrante sintomática de aorta. Rev Esp Cardiol. 2009;62:944–5.
12. Cooley DA. The history of surgery of the thoracic aorta. Cardiol Clin. 1999;17: 609–13.
13. Coselli JS, Conklin LD, LeMaire SA. Thoracoabdominal aortic aneurysm repair: review and update of current strategies. Ann Thorac Surg. 2002;74:S1881–4.
14. Demers P, Miller DC, Mitchell RS, Kee ST, Chagonjian L, Dake MD. Stent-graft repair of penetrating atherosclerotic ulcers in the descending thoracic aorta: mid-term results. Ann Thorac Surg. 2004;77:81–6.
15. Buth J, Harris PL, Hobo R, Van Eps R, Cuypers P, Duijm L, et al. Neurologic complications associated with endovascular repair of thoracic aortic pathology: Incidence and risk factors. A study from the European Collaborators on Stent/Graft Techniques for Aortic Aneurysm Repair (EUROSTAR) registry. J Vasc Surg. 2007;46:1103–10.
16. Rutherford RB, Baker JD, Ernst C, Johnston KW, Porter JM, Ahn S, et al. Recommended standards for reports dealing with lower extremity ischemia: revised version. J Vasc Surg. 1997;26:517–38.
17. Mitchell RS, Ishimaru S, Ehrlich MP, Iwase T, Lauterjung L, Shimono T. First International Summit on Thoracic Aortic Endografting: roundtable on thoracic aortic dissection as an indication for endografting. J Endovasc Ther. 2002;9: 98–105.
18. Evangelista A, Dominguez R, Sebastia C, Salas A, Permanyer-Miralda G, Avegliano G, et al. Prognostic value of clinical and morphologic findings in short-term evolution of aortic intramural haematoma. Therapeutic implications. Eur Heart J. 2004;25:81–7.
19. Harris JA, Bis KG, Glover JL, Bendick PJ, Shetty A, Brown OW. Penetrating atherosclerotic ulcers of the aorta. J Vasc Surg. 1994;19:90–8.
20. Eggebrecht H, Herold U, Schmermund A, Lind AY, Kunht O, Martini S, et al. Endovascular stent-graft treatment of prenetrating aortic ulcer: results over a median follow-up of 27 months. Am Heart J. 2006;151:530–6.
21. Svensson LG, Kouchoukos NT, Miller DC, Bavaria JE, Coselli JS, Curi MA, et al.; Society of Thoracic Surgeons Endovascular Surgery Task Force. Expert consensus document on the treatment of descending thoracic aortic disease using endovascular stent-grafts. Ann Thorac Surg. 2008;85:S1–41.
22. Rehman SM, Vecht JA, Perera R, Jalil R, Saso S, Kidher E, et al. How to manage the left subclavian artery during endovascular stenting for thoracic aortic dissection? An assessment of the evidence. Ann Vasc Surg. 2010;24: 956–65.
23. Da Rocha MF, Miranda S, Adriani D, Urgnani F, Riambau VA, Mulet J. Procedimientos híbridos para patología aórtica compleja. Experiencia inicial de un centro. Rev Esp Cardiol. 2009;62:896–902.
24. Eggebrecht H, Baumgart D, Schmermund A, Herold U, Hunold P, Jakob H, et al. Penetrating atherosclerotic ulcer of the aorta: treatment by endovascular stent-graft placement. Curr Opin Cardiol. 2003;18:431–5.
25. Chiesa R, Melissano G, Marrocco-Trischitta MM, Civilini E, Setacci F. Spinal cord ischemia after elective stent-graft repair of the thoracic aorta. J Vasc Surg. 2005;42:11–7.
26. Rodríguez-Caulo EA, Lucio R, Zarka ZA, Riambau V. Endoprótesis ramificada para el tratamiento endovascular completo de aneurisma de aorta toracoabdominal tipo II. Rev Esp Cardiol. 2010;63:1106–8.
27. Geisbüsch P, Kotelis D, Weber TF, Hyhlik-Dürr A, Kauczor HU, Böckler D. Early and midterm results after endovascular stent graft repair of penetrating aortic ulcers. J Vasc Surg. 2008;48:1361–8.