- 3. Khairy P, Seslar SP, Triedman JK, Cecchin F. Ablation of atrioventricular nodal reentrant tachycardia in tricuspid atresia. J Cardiovasc Electrophysiol. 2004;15: 719–22.
- Nehgme RA, Carboni MR, Care J, Murphy JD. Transthoracic percutaneous access for electroanatomic mapping and catheter ablation of atrial tachycardia in patients with a lateral tunnel Fontan. Heart Rhythm. 2006;3:37–43.
- 5. Dave AS, Aboulhosn J, Child JS, Shivkumar K. Transconduit puncture for catheter ablation of atrial tachycardia in a patient with extracardiac Fontan palliation. Heart Rhythm. 2010;7:413–6.

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Utility of Multidector Computed Tomography for Postprocedure Evaluation of Endovascular Aortic Stent-grafts

Utilidad de la tomografía computarizada multidetectores en el seguimiento de las endoprótesis aórticas

To the Editor,

Endovascular repair techniques for diseases affecting the descending thoracic aorta¹ are often used as an alternative to surgery, particularly in older patients and those with associated diseases, because of the lower morbidity and mortality of these procedures.² The reported long-term outcome of endovascular repair varies considerably among studies. The most commonly described complication is detection of endoleaks, which are defined as persistent arterial flow external to the stent-graft lumen. Although the diagnosis has been traditionally established by digital subtraction angiography, computed tomography, which tends to be the initial diagnostic technique used in these aortic diseases, is increasingly being employed for long-term follow-up of these patients.³

We carried out a study to determine the presence and types of late endoleaks in descending aorta stent-grafts by multidetector computed tomography.

This study included 36 patients (32 men) with a mean age of 63 (36-83) years, who underwent endovascular stent grafting of the descending aorta between January 2008 and January 2011, and had more than 1 year of multidetector computed tomography follow-up.

Endovascular treatment was performed for the following indications: aortic aneurysms (14 patients), type B aortic dissection (16 patients), intramural hematoma associated with a penetrating aortic ulcer (5 patients), and traumatic aortic rupture (1 patient).

A 64-detector computed tomography unit was used, and the study covered the entire thorax and abdomen in 2 acquisitions. The first was performed without contrast and the second was contrast-enhanced in the arterial phase to identify calcifications that could simulate an endoleak because of their high radiologic density. In addition to the usual axial, coronal, and oblique views, multiplanar recontruction (MPR), maximum intensity projection (MIP) and 3-dimensional virtual recontructions (3D-VR) were performed. In all patients, an advanced analysis of the vessel was carried out, calculating the area and diameters of the entire aorta in slices perpendicular to its longitudinal axis. When required, the vessel section was corrected manually (as in cases of thrombosed aneurysms) to include the area that had been excluded and to assess possible growth.

We analyzed the presence of 4 of the 5 types of endoleaks described:⁴ type I, deficient sealing of the stent-graft (proximal or distal neck); type II, retrograde flow through collateral vessels; type III, structural changes in the stent-graft (breakage or modular separation); and type V, endotension (expansion of a thrombosed aneurysm without evidence of endoleak formation). Type IV endoleak (porosity) was not included, being a rare, transient endoleak that is detected on invasive arteriography.

Endoleaks were detected in 9 (25%) of the 36 patients. In 8 patients, type I, II and III endoleaks were seen on the first multidetector computed tomography study following stent-graft implantation (1-6 months) and in 1 patient, a type V endoleak was demonstrated at the 1-year follow-up, with continuous expansion in successive studies.

Type 1 endoleaks were observed in 4 patients with aortic dissection; type II, in 1 patient with an aneurysm; type III, in 3 patients with an aneurysm; and type V, in 1 patient, also with an aneurysm (Figure).



Figure. Type I endoleak: Dissection of the descending aorta with defective sealing of the proximal portion of the stent-graft. Type II endograft: With repatency of the aneurysmal sac through the celiac trunk, which was occluded at its origin, with filling through collateral vessels from the superior mesenteric artery. Type III endoleak: 2 cases with contrast passage to the aneurysm due to a structural stent-graft alteration. Type V endoleak: Expansion of a thrombosed aneurysm at the follow-up examination, without evidence of a leak.

Endoleaks are the most common late complications of endovascular repair of the descending aorta, with a reported incidence reaching 30% and a mean incidence of 13%.⁵ The incidence in our study was 25%.

Type I endoleak tends to occur in patients whose underlying aortic disease is a dissection and the communication between the true and false lumen persists. Type II endoleak usually occurs in patients treated for an aortic aneurysm, and consists of repatency of the aneurysmal sac by collateral vessels. In our single case of this type of endoleak, the collateral originated from branches of the superior mesenteric artery that filled the proximal portion of the celiac trunk (occluded by the stent-graft, which covered the distal third of the descending thoracic aorta and the upper third of the abdominal aorta). Type III endoleak usually occurs in patients treated for an aortic aneurysm. In addition to structural failure of the stent-graft, the stress that the stent sustains due to aortic pulsatility or constriction of the aneurysmal sac can facilitate the development of endoleak. Type V endoleak consists of gradual expansion of the aneurysmal sac without an obvious endoleak.

In conclusion, endoleaks are common following endovascular treatment of the descending aorta, and their noninvasive follow-up with multidetector computed tomography study is a feasible approach.

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Role of Intravascular Ultrasound in Stent Thrombosis

Valor de la ecografía intracoronaria en la trombosis de stent

To the Editor.

Despite ongoing development in stent design and the greater efficacy of antiplatelet therapy, stent thrombosis (ST) continues to be a widely recognized and much dreaded adverse event, with an incidence of 1% to $5\%^{1,2}$ and a mortality that exceeds 10% in all series.^{3,4} In ST, the implantation of an additional stent has been related to an adverse outcome, higher risk of rethrombosis, and increased mortality.^{1,2} Intravascular ultrasound (IVUS) is an essential tool to identify the causal mechanism of ST; however, IVUS is rarely used to investigate ST in our setting.^{1,5}

We describe IVUS findings obtained in definitive STs referred between 2008 and 2011 to our hospital and compare the therapeutic management of patients who underwent IVUS to that of patients not examined with IVUS.

A total of 2028 patients with 3004 stents were treated and 45 definitive STs were reported, 18 (40%) of them investigated with IVUS (Table). IVUS was more likely to be used in acute and subacute ST than late or very late ST. In most cases, several STrelated mechanisms were identified: in patients with early ST (acute and subacute), underexpansion and lesion at the stent border were the most common echographic findings, whereas patients with late and very late thrombosis were most likely to show in-stent proliferation with severe stenosis and, in 1 case, malapposition due to positive vessel remodeling. The 4 ST mechanisms observed are shown in the Figure.

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REFERENCES

- 1. Therasse E, Soulez G, Giroux MF, Perreault P, Bouchard L, Blair JF, et al. Stent-graft placement for the treatment of thoracic aortic diseases. Radiographics. 2005; 25.157-73
- 2. Svensson LG, Kouchoukos NT, Miller DC, Bavaria JE, Coselli JS, Curi MA, et al. Expert consensus document on the treatment of descending thoracic aortic disease using endovascular stent-grafts. Ann Thorac Surg. 2008;85 Suppl 1:S1-41.
- 3. Bean MJ, Johnson PT, Roseborough GS, Black JH, Fishman EK. Thoracic aortic stent-grafts: utility of multidetector CT for pre- and postprocedure evaluation. Radiographics. 2008;28:1835-51.
- 4. Pua U, Tay KH, Tan BS, Htoo MM, Sebastian M, Sin K, et al. CT appearance of complications related to thoracic endovascular aortic repair (TEVAR): a pictorial essay. Eur Radiol. 2009;19:1062-8.
- 5. Jones L, Ak Yiku L, Wilson R. A systematic review of the recent evidence for the efficacy and safety relating to the use of endovascular stentgraft (ESG) placement in the treatment of thoracic aortic disease. London: NICE: 2005. Available at: www.nice.org.uk/ip001review

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In terms of therapeutic management, patients with late thrombosis most often required balloon predilation to advance the IVUS probe, which could overestimate the minimum stent area. In 17 patients, IVUS identified the definitive cause of thrombosis. The symptoms were related to discontinuation of dual antiplatelet therapy in only 1 patient with late thrombosis, and IVUS study revealed no pathologic findings. The use of glycoprotein IIb-IIIa inhibitors and thrombosis aspiration devices was more common in the group of patients assessed with IVUS. STs examined by IVUS were treated less often with implantation of a second stent. In fact, IVUS study made it possible to orient and optimize treatment in all patients. No significant differences were detected in angiographic outcome, mortality, or rethrombosis.

The IVUS findings of early and late ST in our series presented different profiles, which could indicate that these entities have different pathophysiologic mechanisms. The relationship between early thrombosis and mechanical factors during the implant procedure has already been reported in previous studies. Cheneau et al.⁶ found that subacute ST and inadequate outcome in the implantation procedure was related to significantly smaller stent areas and other echographic findings, such as dissection, residual thrombus, or tissue prolapse between the struts. In the largest published register, Amstrong et al.⁴ identified multiple clinical, angiographic, and prognostic factors based on the point in time of the STs, which would indicate that each entity must correspond to a different etiologic mechanism. Additionally, these authors observed a stronger tendency toward stent implantation in very late thrombosis than in early thrombosis.

Implantation of an additional stent in thrombosis conditions was identified as an independent predictive factor of mortality and