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 ST-related 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.

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

Table
Intravascular Ultrasound Findings in Enrolled Patients and Therapeutic Management

Patient	Age	Vessel	Stent	Time, d	Angiographic findings	Intravascular ultrasound findings	Therapeutic management
1	68	ADA	DES	0	Complete occlusion at proximal stent border	Underexpansion and adherent thrombus	Thrombus aspiration, abiximab, and postdilation
2	70	RCA	BMS	1	Complete occlusion at proximal stent border	Intimal flap at proximal stent border	Thrombus aspiration and implantation of new BMS
3	69	RCA	BMS	4	Image of organized stent thrombus, with TIMI 1 distal flow	Slight underexpansion. Severe lesion at distal stent border	Thrombus aspiration and implantation of new BMS
4	72	ADA	DES	4	Complete stent occlusion at overlap of tandem stents	Considerable underexpansion (360° calcium ring in vessel) (Figure B)	High-pressure postdilation with balloons of increasing size
5	63	ADA	DES	4	Complete occlusion at proximal stent border	Intimal flap at distal stent border, malapposition	Thrombus aspiration, abiximab, and implantation of a new DES
6	69	ADA	DES	4	Complete occlusion at proximal stent border	Stent underexpansion and malapposition	Thrombus aspiration, abiximab, and postdilation
7	47	RCA	DES	4	Complete stent occlusion (distal border)	Intimal flap at distal stent border with portal entrance distally visible but proximally trapped by stent; 60-90° arch	Thrombus aspiration, abiximab, and new DES
8	76	Cx	DES	5	Complete occlusion at proximal stent border	Severe underexpansion, malapposition	Thrombus aspiration, abiximab, and postdilation
9	72	RCA	BMS	5	V-shaped stents at bifurcation, with occlusion from stent origin in posterolateral artery	Underexpansion of posterolateral artery stent	Abciximab and postdilation
10	40	ADA	BMS	8	Complete occlusion anterior to stent with ruptured plaque image at proximal stent border	Intimal flap at proximal stent border. Adherent thrombus (Figure A)	Thrombus aspiration, abiximab, and new DES
11	83	Dx	BMS	9	Complete occlusion from stent origin in first Dx	Stent struts adjusted to Dx ostium, protruding into ADA; underexpansion	Thrombus aspiration toward ADA, abiximab, and postdilation
12	64	RCA	BMS	14	Stent thrombosis in middle RCA with TIMI 0 flow	Stent underexpansion	Thrombus aspiration, abiximab, and postdilation
13	71	GSV	DES	240	Critical focal in-stent restenosis at distal border of bridge (anastomosis toward OM) with TIMI 1 distal flow	Severe concentric hyperplasia	Thrombus aspiration, abiximab, and new DES
14	58	OM	DES	290	Image of stent thrombus with aneurysmal vessel dilatation at proximal stent border	Malapposition by positive vessel remodeling (Figure C)	Thrombus aspiration, abiximab, and postdilation; IVUS confirmation of outcome
15	49	RCA	DES	350	Complete occlusion at proximal stent border	Good stent apposition with no proliferation; adherent thrombus	Thrombus aspiration and abiximab
16	69	PIV	DES	683	Complete occlusion of stent (middle segment) implanted in distal RCA toward posterior descending artery	Underexpansion and adherent thrombus	Abciximab and postdilation
17	70	ADA	DES	1858	Image of organized stent thrombus with TIMI 1 distal flow	Stent underexpansion, malapposition, and residual thrombus	Thrombus aspiration, abiximab, and postdilation
18	51	RCA	BMS	2221	Complete occlusion of proximal border of first stent (2 overlapping stents)	Diffuse stent proliferation (probable neoatherosclerosis) with severe stenosis (Figure D)	Abciximab and DES implant

	Therapeutic management in both groups		*P*
	IVUS (n=18)	No IVUS (n=27)	
Early thrombosis	12 (66.7)	5 (3.7)	.01*
Late thrombosis	6 (33.3)	14 (51.8)	.22
Bare-metal stents	7 (38.9)	9 (33.3)	.71
Drug-eluting stents	11 (61.1)	18 (66.7)	.71
Aspiration	14 (77.8)	13 (48.2)	.05*
Abciximab	15 (83.3)	14 (51.8)	.03*
New stent implantation	6 (33.3)	17 (62.9)	.05*
Mortality	4 (22.2)	4 (14.8)	.52
Rethrombosis	1 (5.5)	3 (11.1)	.52

ADA, anterior descending artery; BMS, bare-metal stent; Cx, circumflex artery; DES, drug eluting stent; Dx, diagonal artery; GSV, great saphenous vein; IVUS, intravascular ultrasound; OM, obtuse marginal artery; PIV, posterior interventricular artery; RCA, right coronary artery; TIMI: *Thrombolysis in Myocardial Infarction*.
Malapposition is defined as 1 or more stent struts separated from the vessel wall, except at the origin of a secondary branch. Underexpansion is defined as minimal stent area <80% of the mean proximal and distal reference area.
* Statistically significant results.

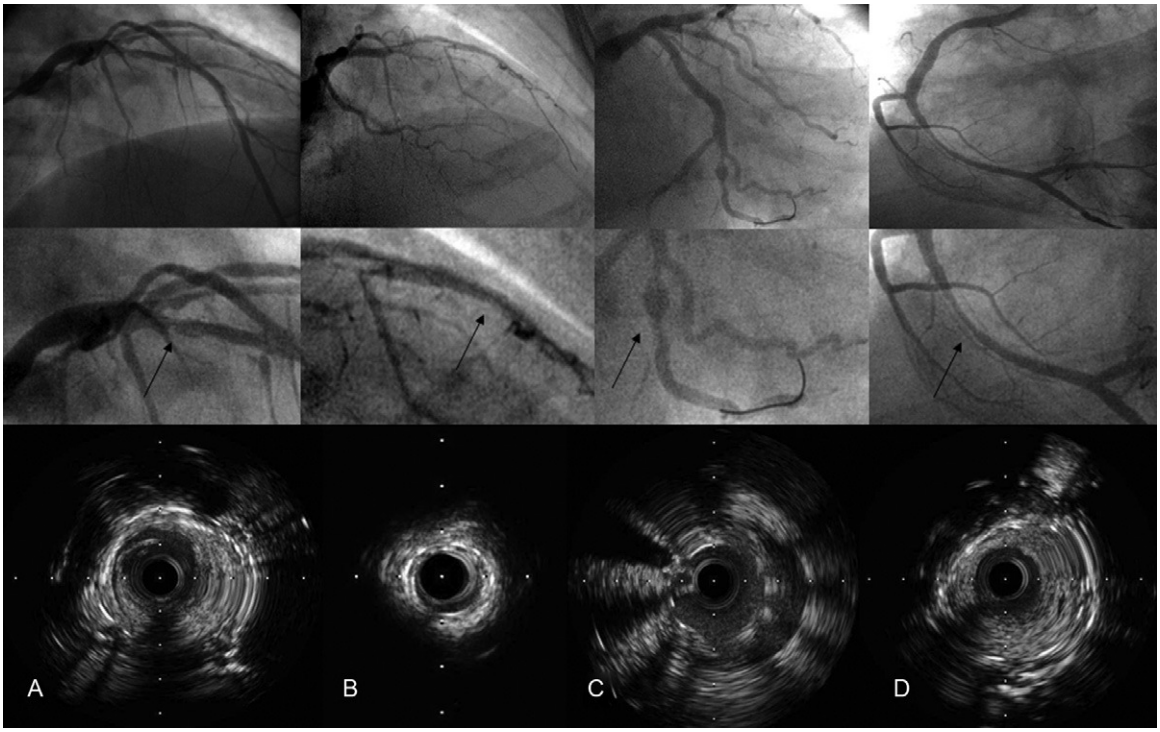

Figure. Four main mechanisms of stent thrombosis. The upper row shows the coronary angiographies once distal flow is recovered, the middle row provides a zoomed view of the stent, and the bottom row contains intravascular ultrasound cross-sectional images. A: Intimal flap at the stent border. B: Stent underexpansion. C: Malapposition due to positive vessel remodeling. D: Neointimal proliferation with severe stenosis.

ST recurrence in the ESTROFA register.[1] In our series, patients who underwent IVUS were less likely to receive a second stent as part of thrombosis management, but no differences in mortality or rethrombosis were observed. However, IVUS was more often used in early than late thrombosis, which could overestimate the value of IVUS in preventing implantation of a second stent.

IVUS is highly useful for investigating the causal mechanism of thrombosis symptoms, as it reveals pathophysiologic factors underestimated by conventional angiography and identifies patients who may benefit from implantation of an additional stent.

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Use of Tolvaptan in Patients With Hyponatremia Due to Heart Failure: Initial Experience

Uso de tolvaptán en pacientes con hiponatremia debida a insuficiencia cardiaca: experiencia inicial

To the Editor,

Hyponatremia is the most common electrolyte disturbance in patients with heart failure (HF) and, at the present time, there is no appropriate treatment strategy for its correction when associated with HF.[1]

The standard treatment for hypervolemia and fluid retention involves loop diuretics, drugs that produce marked natriuresis, which favors the development of hyponatremia in HF.

Arginine vasopressin concentration is known to be high in patients with decompensated HF with anasarca, which contributes to disease progression and is associated with a poorer prognosis.[2] Even a slight increase in arginine vasopressin can cause considerable fluid retention, limiting urinary excretion, a circumstance that