

multiple ICD shocks triggered by SMVT. During his stay he showed nonischemic left ventricle dysfunction (ejection fraction 35%) and thyroid hormone abnormalities (thyrotropin <0.01 $\mu\text{IU/mL}$; free thyroxine 7.7 ng/dL) associated with the use of amiodarone. During hospital admission he presented a new SMVT episode and received 3 ICD shocks. Amiodarone was not well tolerated and was replaced by dronedarone. After hospital discharge, the patient did not experience ICD shocks during 6 months of follow-up, except for 1 shock that occurred within the first 2 weeks.

The third patient, a 42-year-old man, presented with SMVT in 2005 and was diagnosed with arrhythmogenic right ventricular cardiomyopathy, for which he was treated with sotalol 160 mg/d. In 2012, he was admitted to the emergency department for palpitations and presyncope; SMVT was detected and terminated by electrical cardioversion. During hospitalization, he experienced new SMVT episodes that did not respond to treatment with metoprolol and procainamide. The electrophysiology study showed 3 SMVT morphologies, one of them similar to clinical VT but all of them with poor hemodynamic tolerance that degenerated into ventricular fibrillation. We decided to implant a single-chamber ICD and treat the patient with dronedarone 400 mg every 12 h to avoid the adverse effects associated with amiodarone. During the month following discharge, the patient had 4 SMVT episodes and received multiple shocks. As a result, dronedarone was discontinued and sotalol was restarted, at 160 mg every 12 h; a partial response was achieved.

Dronedarone is a benzofuran derivative that shares the antiarrhythmic properties of amiodarone, but with a better safety profile regarding organ toxicity. It has been proven effective in the treatment of atrial arrhythmias in selected populations.^{1,2} However, its effectiveness in treating ventricular arrhythmias is less well known. Animal studies have demonstrated its antiarrhythmic effect on ventricular myocardium.³ Its use in humans has been described in 3 isolated cases, with significant reductions reported in arrhythmia burden and the number of ICD shocks.^{4–6}

In this series, which is the largest published to date, a satisfactory response to dronedarone was obtained in 2 patients without structural heart disease; however, in the patient with arrhythmogenic right ventricular cardiomyopathy the decrease in arrhythmia burden was not significant and the drug was discontinued. Furthermore, no adverse clinical or laboratory events were observed and there were no changes in the ICD pacing and sensing parameters. These results, together with

previously published findings, support the use of dronedarone in patients with recurrent ventricular arrhythmias in whom other AAAs are considered unsuitable and with no contraindications for its use. However, like other AAAs, its use cannot be expected to be completely effective, especially when other drugs have failed.

CONFLICTS OF INTEREST

Dr. Merino has acted as a consultant and received financial remuneration for preparing educational presentations on behalf of Sanofi-Aventis.

Jorge L. Páez, David Doiny, Alejandro Estrada, Jorge Figueroa, David Filgueiras-Rama, and Jose L. Merino*

Unidad de Arritmias y Electrofisiología Cardíaca Robotizada, Servicio de Cardiología, Hospital Universitario La Paz, Madrid, Spain

*Corresponding author:

E-mail address: jlmerino@secardiologia.es (J.L. Merino).

Available online 20 June 2013

REFERENCES

1. Hohnloser SH, Crijns HJ, Van Eickels M, Gaudin C, Page RL, Torp-Pedersen C. Effect of dronedarone on cardiovascular events in atrial fibrillation. *N Engl J Med*. 2009;360:668–78.
2. Hernández-Madrid A, Francés RM, Moro C. Novedades en electrofisiología cardíaca y arritmia. *Rev Esp Cardiol*. 2011;64 Supl. 1:81–90.
3. Finance O, Manning A, Chatelain P. Effects of a new amiodarone-like agent, SR 33589, in comparison to amiodarone, D,L-sotalol, and lignocaine, on ischemia-induced ventricular arrhythmias in anesthetized pigs. *J Cardiovasc Pharmacol*. 1995;26:570–6.
4. Fink A, Duray GZ, Hohnloser SH. A patient with recurrent atrial fibrillation and monomorphic ventricular tachycardia treated successfully with dronedarone. *Europace*. 2011;13:284–5.
5. Shaaraoui M, Freudenberger R, Levin V, Marchlinski FE. Suppression of ventricular tachycardia with dronedarone: a case report. *J Cardiovasc Electrophysiol*. 2011;22:201–2.
6. Exposito V, Rodriguez-Entem F, Gonzalez-Enriquez S, Olalla JJ. Dronedarone for recurrent ventricular tachycardia: a real alternative? *Indian Pacing Electrophysiol J*. 2012;12:73–6.

<http://dx.doi.org/10.1016/j.rec.2013.02.008>

Nonvalvular Atrial Aneurysmal Dilation

Dilatación aneurismática auricular en pacientes no valvulares

To the Editor,

Left atrial (LA) aneurysmal dilation is often associated with rheumatic valve disease.¹ Our objective was to define the actual incidence of nonvalvular aneurysmal dilation of the LA and the clinical and echocardiographic characteristics of patients with this condition.

Aneurysmal dilation was defined as an anteroposterior LA diameter of more than 6 cm, measured in M-mode from the parasternal view. Echocardiographic studies recorded during 2010 and 2011 were reviewed. Patients with mitral valve prosthesis, mitral stenosis, or mitral regurgitation of more than mild severity and pericardial constriction were excluded. Patients were then classified in 1 of 2 groups: patients with LA aneurysmal dilation with diastolic dysfunction (ADDD) and those with isolated aneurysmal dilation (IAD). Diastolic dysfunction was

defined as an e' velocity of less than 8 cm/s at the septal mitral annulus.²

The echocardiographic parameters recorded (see Table) were as follows: anteroposterior LA diameter and indexed volume according to the Simpson rule (apical 4-chamber 2-chamber view), right atrial volume, left ventricular systolic and diastolic function, e' and S velocities at the septal mitral annulus, pulmonary pressure, tricuspid annular plane systolic excursion, and severity of regurgitation. In each case, the mean of 5 measurements was taken.

The clinical parameters recorded were age, sex, cardiac rhythm, smoking habit, diabetes mellitus, dyslipidemia, hemoglobin in blood, hypertension, chronic pulmonary disease, heart disease, functional class, and history of admission for heart failure.

Of 22 555 echocardiograms (Figure), 644 had LA >6.0 cm; 116 (18%) did not have any significant mitral valve lesions, and mitral valve prostheses were found in 43%, stenosis in 22%, mitral regurgitation in 9.3%, and annular calcification with hemodynamic impact in 6.8%. Eighty per cent of the patients without valve disease were in atrial fibrillation (AF). Twenty patients (17.2%) had $e' >8$ cm/s and 18 were in AF; these patients comprised the IAD

Table

Clinical and Echocardiographic Characteristics of Patients With Isolated Aneurysmal Dilatation and Aneurysmal Dilatation With Diastolic Dysfunction of the Atrium

	IAD (n=18)	ADDD (n=25)	P
Age, years	80.11±6.4	83.48±7.8	.086
Female	50.0	60.9	.697
Atrial fibrillation	100	100	1
Heart rate	84.33±14.3	77.04±12.0	.09
Hemoglobin, mg/dL	13.3±2.0	12.6±2.0	.289
Hypertension	66.7	84.0	.275
Diabetes mellitus	22.2	33.3	.430
Dyslipidemia	38.9	37.5	.927
Cigarette smoking	9.1	13.0	.375
Heart disease	33.3 ^a	32 ^b	1
Chronic pulmonary disease	44.4	20.8	.101
Digoxin	7 (38.8)	5 (20)	.301
ARB/ACE inhibitors	12 (66.6)	15 (60)	.755
Beta blockers	7 (38.8)	7 (28)	.521
Calcium antagonists	3 (16.6)	8 (32)	.309
Diuretics	10 (55.6)	23 (92)	.009
Anteroposterior diameter, mm	7.11±1.1	6.35±0.7	.021
LA volume/body surface area, cc/m ²	86.98±34.0	77.8±23.9	.312
RA volume/body surface area, cc/m ²	70.26±23.2	67.57±21.4	.759
LV diastolic diameter, cm	4.9±0.7	4.9±0.8	.786
LV systolic diameter, cm	3.2±0.6	3.2±0.8	.701
LVEF, %	64.3±8.1	63.1±9.4	.498
Ventricular septum, cm	1.0±0.1	1.1±0.1	.586
LV posterior wall, cm	1.0±0.2	1.0±0.2	.898
Mass/body surface area, g/m ²	140.98±45.0	144.22±47.5	.824
Mitral E, cm/s	98.28±27.1	113.60±22.8	.076
Time for mitral E deceleration, ms	171.11±65.0	191.80±43.6	.035
e' of septal annulus (cm/s) by pulsed DTI	9.39±2.0	4.96±0.88	<.001
E/e' ratio	10.83±3.3	23.44±6.9	<.001
S septal annulus (cm/s) by pulsed DTI	5.00±1.1	4.40±1.0	.075
Systolic pulmonary pressure, mmHg	45.17±12.5	53.12±13.3	.05
TAPSE, mm	17.89±3.7	17.00±3.7	.367
Tricuspid regurgitation 0-I-II-III	3-12-2-1	2-12-6-5	.099
Aortic regurgitation 0-I-II-III	9-9-0-0	13-12-0-0	.859
Mitral regurgitation 0-I-II-III	1-17-0-0	3-22-0-0	.854

ACE, angiotensin converting enzyme; AD DD, aneurysmal dilatation with diastolic dysfunction; AoE, aortic stenosis; ARB, angiotensin receptor blocker; AS, (operated) atrial shunt; Cor P, cor pulmonale; DCM, dilated cardiomyopathy; DTI, Doppler tissue imaging; HCM, hypertrophic cardiomyopathy; IAD, isolated aneurysmal dilatation; IHD, ischemic heart disease; LA, left atrium; LV, left ventricular; LVEF, left ventricular ejection fraction; RA, right atrium; TAPSE, tricuspid annular plane systolic excursion. Data expressed as percentage, no. (%), or mean±SD unless otherwise indicated. Continuous variables, Mann-Whitney U test; categorical variables, χ^2 test; ordinal variables, Fisher exact test and Mantel-Haenszel test. Statistical comparisons are bilateral (statistical package SPSS v17).

^a IHD, 2; HCM, 1; AS, 1; DCM, 1; Cor P, 1; amyloidosis, 0.

^b IHD, 4; HCM, 2; AS, 1; AoE, 1; amyloidosis, 0.

group. Ninety-six patients (82.8%) had e' <8 cm/s, 82 of whom were in AF. Twenty-five of these age-matched patients comprised the AD DD group.

The clinical differences between the IAD and AD DD groups were not significant (Table). Likewise, there were no differences in diameter, wall thickness, and ejection fraction. The anteroposterior LA diameter was greater in the IAD group, although the volume was similar. Patients in the IAD group had an e' wave velocity greater than expected for their age (9.39 cm/s) and greater than the e' wave velocity for the AD DD group. The E velocity and E/e' ratio in the IAD group were lower than those of the AD DD group. An E/e' ratio >15 was found less often in the IAD group (16.6% vs 92%). Systolic pulmonary pressure was lower in the IAD group. Within this group, patients without chronic pulmonary disease (66.6%)

had a lower systolic pulmonary pressure (36 vs 58 mmHg; $P<.001$) and greater tricuspid annular plane systolic excursion.

In the IAD group, a history of heart failure was less frequent (33% vs 75%; $P=.007$), patients had a better functional status (69% vs 36% with functional class <II), and diuretic treatment was less widely used (55.5% vs 92%). When patients with chronic pulmonary disease were excluded, only 1 patient had heart failure and all were in functional class I or II.

Although most atrial aneurysms continue to present in patients with mitral valve disease, 18% appear in patients without significant valve disease. Atrial fibrillation, present in 80% of the patients, accounts in part for the atrial dilatation. Although no information is available on the duration of AF, given the age of the patients the condition was likely to have been long-standing.

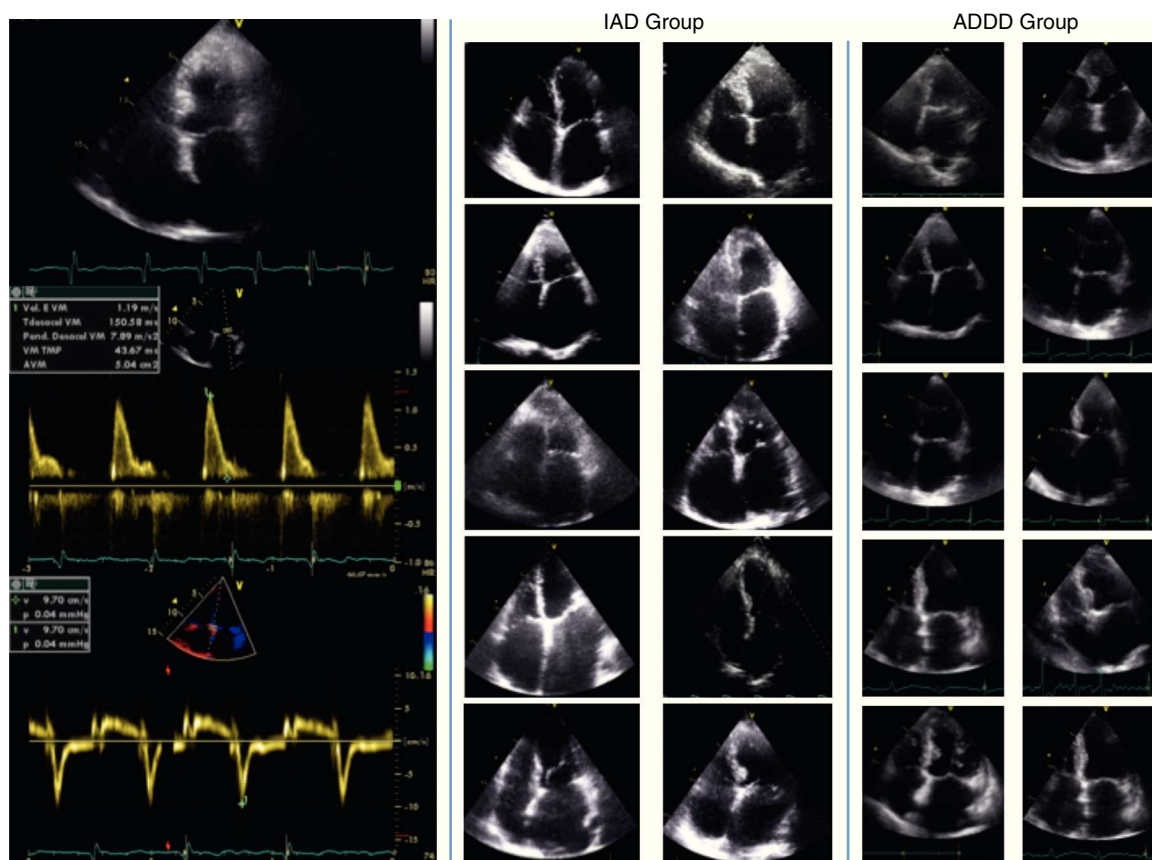


Figure. Apical, 4-chamber echocardiogram showing mitral filling and Doppler tissue imaging of the septal mitral annulus of a patient in the group of isolated aneurysmal dilation in the left atrium (left panel). Echocardiograms of the isolated aneurysmal dilation group (center) and aneurysmal dilation with diastolic dysfunction (right). ADDD, aneurysmal dilation with diastolic dysfunction; IAD, isolated aneurysmal dilation.

According to the guidelines, increased atrial volume without valve disease reflects diastolic dysfunction, raised pressures,² poor clinical condition, and poor prognosis.³ This did not occur in 17% of our patients with nonvalvular dilation. According to the guidelines, patients with atrial dilation and $e' > 8$ cm/s are athletes or have pericardial constriction, neither of which were applicable in our patients. Atrial remodeling without any increase in atrial pressure could be one explanation.⁴ Another possibility is a paradoxical behavior of e' in the presence of diastolic dysfunction.

The similar incidences of risk factors, heart disease, and even a decreased annulus S velocity in the 2 groups indicates that there is a certain degree of myocardial involvement in both groups. It may be that the patients in the IAD group have a pseudonormal e' wave pattern associated with AF with aneurysmal dilation. When the atrium does not contract, the entire annular movement occurs during protodiastole, and could be quicker due to the large atrial dilation. Moreover, with a more dilated LA, larger loads are possible without increasing the pressure and so clinical manifestations are more limited. In any case, patients with IAD should have better diastolic function and lower pressure as their functional class and therapeutic requirements are markedly lower and they have less history of heart failure. In fact, in this group, only patients with chronic pulmonary disease showed clinical deterioration.

This study is limited in that it was retrospective and had no invasive measurements. The definition of atrial aneurysm is arbitrary. Other studies have used the term giant left atrium. It would have been better to use the atrial volume, but the coding meant that such an analysis was impossible. Several patients had mild mitral regurgitation, which could be relevant if the severity increased with exercise.

Atrial aneurysmal dilation is not always associated with mitral valve disease. It can be associated with AF and old age, often in patients with diastolic dysfunction in a poor clinical state. Some elderly patients with atrial aneurysm have a good functional status and normal pulmonary pressure; they can be identified by a paradoxically elevated e' wave. In view of the tendency for rheumatic heart disease to disappear and given the aging of the population, nonvalvular atrial aneurysmal dilation will become increasingly common.

ACKNOWLEDGEMENTS

We thank Dr S. Ruiz Leria, Dr A. Megías Saez, and Dr A. García Martín, of the cardiology department, and Alfonso Muriel, of the biostatistics department of the Hospital Ramón y Cajal, Madrid.

Jose L. Moya-Mur,^{a,*} Derly Becker-Filho,^a José J. Jiménez-Nacher,^a Alberto García-Lledó,^b Covadonga Fernández-Golfín,^a and Jose L. Zamorano-Gómez^a

^aServicio de Cardiología, Hospital Universitario Ramón y Cajal, Madrid, Spain

^bServicio de Cardiología, Hospital Universitario Príncipe de Asturias, Alcalá de Henares, Madrid, Spain

*Corresponding author:

E-mail address: jmoya.hrc@salud.madrid.org (J.L. Moya-Mur).

Available online 29 May 2013

REFERENCES

1. Moral S, Fernández-Friera L, Sanz J. Aurícula izquierda gigante evaluada mediante resonancia magnética. *Rev Esp Cardiol*. 2011;64:232.
2. Nagueh SF, Appleton CP, Gillebert TC, Marino PN, Oh JK, Smiseth OA, et al. Recommendations for the evaluation of left ventricular diastolic function by echocardiography. Guidelines and standards. *Eur J Echocardiogr*. 2009;10:165–93.
3. Tsang TS, Barnes ME, Gersh BJ, Bailey KR, Seward JB. Left atrial volume as a morphophysiologic expression of left ventricular diastolic dysfunction and relation to cardiovascular risk burden. *Am J Cardiol*. 2002;90:1284–9.
4. Aronow WS, Schwartz KS, Koenigsberg M. Prevalence of enlarged left atrial dimension by echocardiography and its correlation with atrial fibrillation and an abnormal P terminal force in lead V1 of the electrocardiogram in 588 elderly persons. *Am J Cardiol*. 1987;59:1003–4.

<http://dx.doi.org/10.1016/j.rec.2013.02.010>

Aortitis: An Unusual Cause of Acute Chest Pain

Aortitis: una causa de dolor torácico agudo poco común

To the Editor,

A 71-year-old man with controlled hypertension and no family history of interest presented with a 3-h history of severe central chest pain radiating to his back. He reported having a headache and joint pains, particularly in his shoulders, for the last few days. Two years previously, he had undergone an echocardiogram, exercise testing, and cardiac catheterization for chest pain, with no significant findings. The physical examination was unremarkable. In view of the nature of the pain, we performed computed tomography angiography, which showed an ascending aortic aneurysm with thickening of the aortic wall (Fig. 1). The results of laboratory tests, including D-dimer, were normal, except for an elevated erythrocyte sedimentation rate (91 mm/h) and C-reactive protein (1.8 mg/L). We decided to confirm the suspected diagnosis and rule out intramural hematoma by means of magnetic resonance angiography, which showed a thoracic ascending aortic aneurysm with a maximum diameter of 47 mm and wall thickening of 7 mm, without aortic root or arch involvement (Figs. 2A and B). We also performed a single-photon emission computed tomography scan with ^{99m}Tc . Delayed imaging showed persistent activity in the thoracic aorta, indicating inflammation.

Aortitis was diagnosed from the clinical, laboratory and imaging findings. Based on the patient's age (> 50 years), clinical features (new-onset headache and symptoms of polymyalgia rheumatica), the elevated erythrocyte sedimentation rate and C-reactive protein, and the aforementioned findings of imaging studies, giant cell arteritis (GCA) was diagnosed. A temporal artery biopsy showed no vasculitis, and corticosteroid therapy was started. When investigating the etiology, we ruled out infectious causes.

During follow-up, the symptoms resolved completely, with a gradual decrease in erythrocyte sedimentation rate and C-reactive

protein and return to normal values, and therefore the corticosteroids were reduced. A follow-up magnetic resonance imaging scan was performed after 6 months and showed no thickening of the aortic wall or increased aneurysm size (Figs. 2C and D).

The term aortitis describes inflammation of the aortic wall. The leading causes are of rheumatic origin, in which GCA accounts for over 75% of cases, followed by Takayasu arteritis. Infectious and idiopathic etiologies are much less common.¹

Giant cell arteritis is a vasculitis that affects large and medium vessels^{2,3} with an incidence of 15–30/100 000 population aged more than 50 years.^{1,4} Vascular inflammation may be focal or widespread, which explains the high rate of false negative results in temporal artery biopsy.^{3,4} The aortic wall is affected in 15% to 22% of cases, and the risk of aortic aneurysm is 17 times higher than in healthy individuals.^{1,4,5} The pathogenesis is unknown but is believed to originate in antigen-driven cell-mediated autoimmune processes associated with specific human leukocyte antigens (HLA-DR4). Histopathology shows evidence of an inflammatory infiltrate of the media, adventitia and vasa vasorum, with a predominance of lymphocytes, macrophages, and multinucleated giant cells.^{1,4}

The clinical presentation of aortitis varies across a wide spectrum of signs and symptoms. Classic manifestations are headache, back pain, polymyalgia rheumatica, and fever.⁵ Aortitis can also present as severe aortic insufficiency or aortic aneurysm. Acute aortic syndrome is a less common manifestation, but it should be noted that patients with GCA are at a higher risk of aortic dissection.⁶

An initial evaluation of suspected aortitis should include determination of erythrocyte sedimentation rate, C-reactive protein, and specific antibodies, blood cultures, tuberculosis testing, and serology for syphilis and other infectious diseases.³

The diagnostic criteria for GCA are: age greater than or equal to 50 years, headache, temporal artery abnormality, erythrocyte sedimentation rate greater than or equal to 50 mm/h and arterial biopsy showing vasculitis. The presence of 3 of these criteria has a sensitivity of 94% and a specificity of 91%.^{1,3}

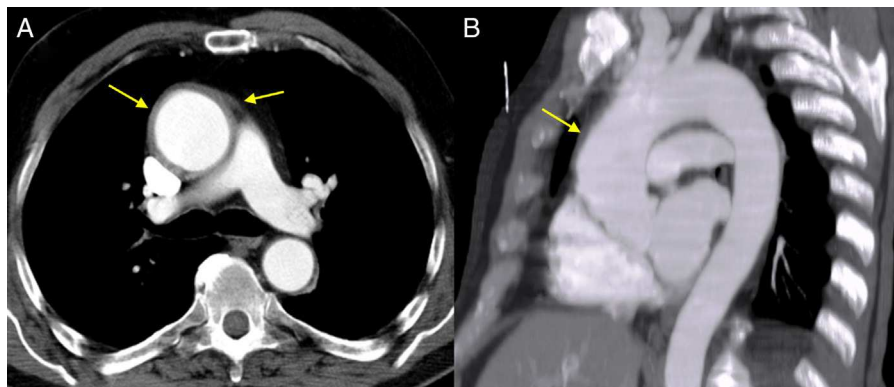


Figure 1. Chest computed tomography scan showing ascending aortic aneurysm and thickening of the aortic wall. A: Axial image. B: Sagittal-plane multiplanar reconstruction.