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
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 ADDD group.

The clinical differences between the IAD and ADDD 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 ADDD group. The E velocity and E/e' ratio in the IAD group were lower than those of the ADDD 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 dilation. 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.
According to the guidelines, increased atrial volume without valve disease reflects diastolic dysfunction, raised pressures, poor clinical condition, and poor prognosis. This did not occur in 17% of our patients with nonvalvular dilatation. According to the guidelines, patients with atrial dilatation and $e' > 8 \text{ cm/s}$ 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. 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 dilatation. When the atrium does not contract, the entire annular movement occurs during protodiastole, and could be quicker due to the large atrial dilatation. 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 dilatation 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 dilatation will become increasingly common.

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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 99mTc. 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.

Giant cell arteritis is a vasculitis that affects large and medium vessels with an incidence of 15–30/100 000 population aged more than 50 years. Vascular inflammation may be focal or widespread, which explains the high rate of false negative results in temporal artery biopsy. 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. 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.

The clinical presentation of aortitis varies across a wide spectrum of signs and symptoms. Classic manifestations are headache, back pain, polymyalgia rheumatica, and fever. 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.

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.

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%.

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