Table 1 *(Continued)*
Clinical baseline characteristics and exercise results in the 165 patients

Peak	1.16 ± 0.30
Left ventricular ejection fraction, %	
Rest	60 ± 6
Peak exercise	65 ± 11

ACEI, angiotensin-converting enzyme inhibitors, ARA-II, angiotensin II receptor antagonist, CAD coronary artery disease; HCM, hypertrophic cardiomyopathy.
Values are expressed as No. (%) or mean ± standard deviation.
[a] The day of the exercise test.
[b] Defined as either symptoms or ischemic electrocardiogram changes during testing.
[c] Exercise-induced left ventricle outflow tract gradient ≥ 50 mmHg and/or significant mitral regurgitation in patients with hypertrophic cardiomyopathies or valvulopathies.

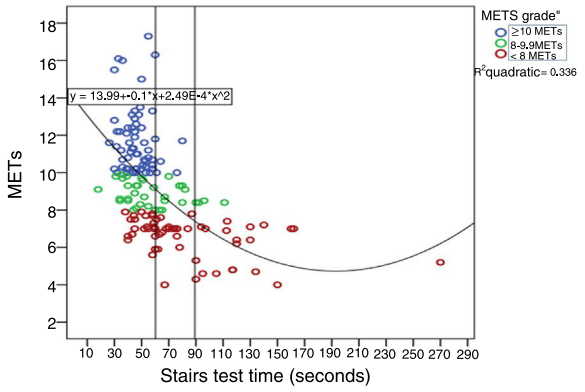

Figure 1. Relationship between stair-climbing time and METs achieved on treadmill exercise testing.

but high specificity (96%) and positive predictive value (83%) for predicting < 8 METs.

Abnormal results were seen in 58% of patients with limited exercise capacity, 30% with intermediate exercise capacity, and 29% with good exercise capacity ($P = .002$), as well as in 32% of patients who completed the stair-climbing test in at least 60 seconds compared with 52% of those that took between 61 and 89 seconds and 58% of those who took longer ($P = .018$).

In a previous study measuring O_2 consumption in healthy young volunteers, climbing up 70 steps in 1 minute equalled 8.6 ± 0.4 METs.[5] Most participants able to step up 4 flights of stairs in 1 minute in our study performed well during exercise testing. However, the lack of prediction in them is likely because the stair-climbing test consists of a mixture of aerobic and anaerobic evaluation, as it implies achieving a high workload in a short time.

Regardless of this consideration, individuals who cannot complete the stair-climbing test in 1.5 minutes are expected to have poor functional capacity and worse exercise test performance and results. In contrast, the range of achieved METs in patients who complete the stairs test in less than 1 minute varies more widely. These findings could be of interest for stress testing and stress echocardiography triage strategies.

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Aortitis: a simulator of intramural aortic hematoma

Aortitis: un imitador del hematoma intramural aórtico

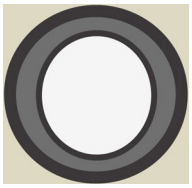
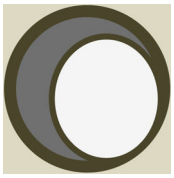
To the Editor,

Most cases of aortitis are noninfectious and their clinical presentation varies widely, ranging from asymptomatic aortic aneurysm to acute chest pain or heart failure.[1] Some patients with aortitis may simulate an acute aortic syndrome (AAS) at presentation.[2] Because treatment strategies for aortitis and AAS diverge widely, an accurate diagnosis is of the utmost importance.

We report 2 cases of IgG-4 aortitis simulating an intramural aortic hematoma (IAH). In addition, we provide a detailed literature-based review of patients with aortitis mimicking an IAH. The main diagnostic clues to differentiate these 2 types of aortic entities are presented in table 1.

Patient 1. A 76-year-old woman with a history of ascending aortic aneurysm (45 mm) and aortic regurgitation (AR) presented to the emergency department with acute and severe chest pain. Blood pressure was 170/80 mmHg and D-dimer value was 1130 ng/mL. Urgent computed tomography (CT) suggested an IAH in the aortic root, ascending aorta, and aortic arch. An ascending aortic aneurysm (50 mm) with a circular aortic wall thickening (8 mm) was documented (figure 1A,B). The patient underwent urgent surgery with replacement of the aortic root, ascending aorta, and arch. At surgery, a marked thickening of the aortic wall without signs of intramural hemorrhage was docu-

Table 1
Key differences in diagnosis between aortitis and IAH

	Aortitis	IAH
Long-lasting history of hypertension	+/-	+++
Aortic wall thickening	Circular 	 Semicircular
Intramural blood pools	—	+++
Ulcer-like projections	—	+++
D-dimer values	+/-	+++
FDG-PET/CT	+++	+/-

CT, computed tomography; FDG, fluorodeoxyglucose; IAH, intramural aortic hematoma; PET, positron emission tomography.

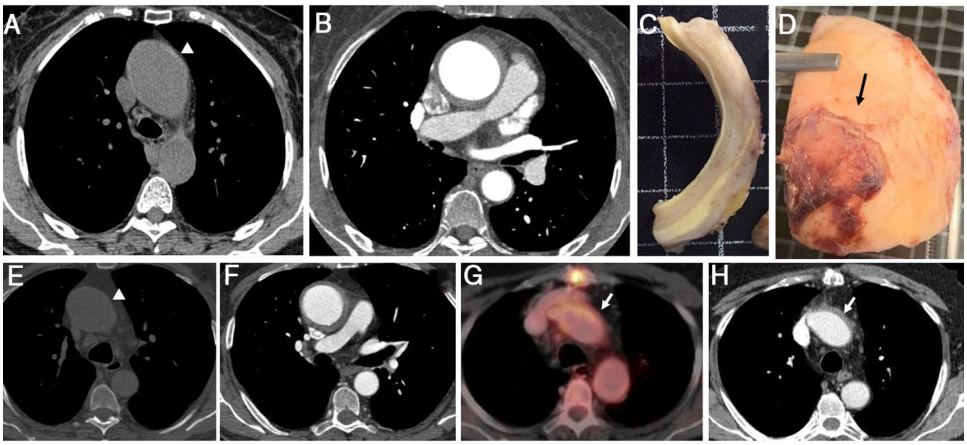


Figure 1. Series of anatomic pieces and imaging test corresponding to 2 patients with aortitis simulating an aortic intramural hematoma (A-H). A-E: noncontrast-enhanced computed tomography (CT) slice showing hyperattenuated thickened aortic wall involving the ascending aorta (arrowhead). Hypointense circular aortic wall thickening affecting the ascending aorta (B,F) and intimal calcium displacement (F). Macroscopical images of the excised ascending aorta showing a marked aortic wall thickening without intramural hemorrhage (C), and the presence of significant periaortitis (arrow) (D). Postsurgical CT and 18F-fluorodeoxyglucose positron emission tomography/CT slices in which the remaining thick aortic arch wall segment had an increased fluorodeoxyglucose uptake (arrow) (G,H).

mented (figure 1C). In addition, periaortic inflammation was evident (figure 1D). Pathologic examination of aortic tissue showed the presence of a large IgG-4 lymphoplasmacytic infiltration. A high-dose corticosteroid regimen was initiated and subsequently tapered with excellent clinical response and follow-up.

Patient 2. A 69-year-old man with no cardiovascular risk factors presented to the emergency room with syncope. Transthoracic echocardiography revealed the presence of a bicuspid aortic valve with moderate AR. A CT scan showed an ascending aortic aneurysm (54 mm) with a circular aortic wall thickening (10 mm) involving the ascending aorta, aortic arch (figure 1E,F), brachiocephalic trunk, and left carotid artery. D-dimer levels were 356 ng/mL. The patient was sent to surgery with the diagnostic suspicion of IAH. At surgery, there was no hematoma within the thick ascending aortic wall. Immunohistochemical analysis gave the diagnosis of IgG-4 aortitis. A postoperative follow-up positron emission tomography (PET)/CT showed a remaining thick aortic arch wall segment with intense fluorodeoxyglucose (FDG) uptake (figure 1G,H). The patient received a high-dose corticosteroid regimen with good clinical and PET/CT follow-up response.

As to specific cases of aortitis mimicking an IAH, we performed a review of the literature to collect data from all cases of aortitis simulating an IAH. Finally, we found 10 cases reported in the literature in addition to our 2 patients.

This series collects data from patients with aortitis who were incorrectly diagnosed with an IAH. Aortitis is an inflammatory disorder involving the aortic wall.[1] Diffuse arterial wall thickening (> 2 mm) and homogeneous wall enhancement are typical features of aortitis on contrast-enhanced CT.[3] In addition, PET/CT may depict the inflammatory process and monitor therapeutic response during follow-up.[4] Glucocorticoid therapy is the mainstay therapy in noninfectious aortitis.[1,2,4]

IAH is an acute aortic entity that belongs to what is widely known as AAS .[5] Diagnosis is usually made by CT, transesophageal echocardiography, or magnetic resonance. The aortic wall does not generally show enhancement with contrast administration on CT. Most cases of type A IAH are treated surgically.[5]

This case series of patients with aortitis mimicking a type A IAH shows several interesting features that should help in the differential diagnosis of these 2 entities. In all patients, the aortic wall thickening was circular instead of semicircular or

crescentic, as is frequently the case of IAH.[5] None of the cases showed intramural blood pools or ulcer-like projections, so characteristic of IAH.[2,4] However, some of the typical imaging signs of IAH were also present in patients with aortitis, such as central displacement of intimal calcification, hyperintensity in noncontrast CT images, and absence of enhancement with contrast administration.[2,4] Regarding the aortic valve, it is possible to have significant AR in both conditions. In aortitis, AR may result from aortic valve inflammation (7 cases in this series).

Laboratory parameters are not very specific; in aortitis, C-reactive protein and erythrocyte sedimentation rate are usually elevated (6 patients of this series),[2–4] but they can occasionally be normal. Acute phase reactants may also be increased in IAH. Thus, these parameters are not definitive. A laboratory parameter that could ultimately be useful in the emergency department is the D-dimer. In patients with AIH, D-dimers are usually high. D-dimer levels were slightly high in 1 of our patients and normal in the other. Unfortunately, they were not measured in the other patients.

PET/CT imaging may play a key role in establishing the diagnosis of aortitis and assessing the extent of the disease to other aortic segments. In addition, it is very helpful in the follow-up of these patients to monitor the therapeutic response.[4] In IAH FDG uptake is null or low intensity.

Interestingly, most cases of aortitis in this series (8 patients) were IgG-4 aortitis.[2] Hypothetically, this type of aortitis may be more likely to simulate an IAH. IgG4-related disease may also present as other cardiovascular conditions, such as pericarditis or intracardiac pseudotumors. Although heart failure as a consequence of valvular dysfunction is usually the form of presentation in cases of intracardiac mass, cardiac arrest and conduction disturbances have also been described as a result of IgG4-related disease.[6] Aortitis is no longer a rare condition, and must be considered in the differential diagnosis of patients with AAS, particularly IAH.

In summary, in patients presenting to the emergency room with chest pain and a thickened ascending aortic wall, the following features should give rise to suspicion of aortitis: absence of a long-lasting history of hypertension, circular aortic wall thickening with absent intramural blood pools or ulcer-like projections, and normal D-dimer values (table 1).

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Diagnostic accuracy of angiography-based quantitative flow ratio in patients with left main disease

Valor diagnóstico del cociente de flujo cuantitativo obtenido mediante angiografía en presencia de lesiones en el tronco común izquierdo

To the Editor,

A large mass of heart muscle is dependent on lesions in the left main coronary artery (LMCA), making revascularization of this structure perhaps more important than in other locations. In general, the decision to revascularize is based on angiography findings.

The quantitative flow ratio (QFR) is a new index that has shown good agreement with the fractional flow reserve (FFR), a value obtained invasively in several clinical situations. The QFR uses 2 coronary angiography views to estimate the FFR based on computational fluid dynamics and 3-dimensional reconstruction without the need for a pressure wire. There is very little evidence on QFR use in the LMCA, and the manufacturers themselves advise against using it in lesions affecting the ostium or bifurcation

This retrospective, observational study in daily clinical practice was designed to assess the diagnostic performance of the QFR to estimate the FFR obtained with an invasive technique and to compare it with angiographic evaluation in inconclusive LMCA lesions

Angiograms of all patients with LMCA stenosis between 30% and 70% by visual estimation and 1 FFR study were analyzed in a single center between January 1, 2018 and May 15, 2019. Studies not including 2 views separated by at least 25° angulation and those of insufficient quality for QFR assessment were excluded.

The visual assessment was carried out by 2 operators whose experience included more than 1000 procedures for functional assessment of coronary lesions. Operators were blinded to the FFR results.

The QFR was measured with the Medis Suite XA QAngio XA 3D QFR software, version 3.2.28.0 (Medis, The Netherlands),[1] taking the angiographic location of the pressure wire sensor as the distal point and without knowledge of the FFR value.

In total, 66 studies from 57 patients were analyzed. Fifty-four studies (81.8%) from 45 patients were suitable for determining the