Table 1
Summary findings of density values (Hounsfield units), monoenergetic imaging (Hounsfield units) at increasing energy levels, and iodine-based results measured at the thrombotic source and at the ascending aorta

	Conv (HU)	Monoenergetic (HU)				Iodine (mg/mL)	ANOVA
		40 keV	55 keV	70 keV	110 keV		
Aorta	103.6 ± 18.3	265.2 ± 69.0	156.4 ± 36.0	107.0 ± 20.7	63.2 ± 8.3	2.64 ± 0.8	
Thrombi	44.7 ± 13.7	69.0 ± 32.7	53.3 ± 19.2	46.3 ± 13.7	40.0 ± 10.0	0.38 ± 0.3	
P value	< .0001	< .0001	< .0001	< .0001	< .0001	< .0001	
Thrombi/aorta	0.44 ± 0.16	0.27 ± 0.13	0.36 ± 0.14	0.44 ± 0.16	0.65 ± 0.19	0.15 ± 0.12	< .0001

Conv, conventional; HU, Hounsfield units; ANOVA, 1-way analysis of variance.

AUTHORS' CONTRIBUTIONS

Conception of the work, data analysis and interpretation, drafting, final approval, and assumption of responsibilities related to the content of the article: G.A. Rodríguez-Granillo. Design of the work, data collection, critical revision, final approval, and acceptance of responsibilities related to the content of the article: J. Cirio and Pedro Lylyk. Data collection, critical revision, final approval, and acceptance of responsibilities related to the content of the article: C. Bleise and L. Fontana.

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None of the authors has any conflicts of interest to declare related to the content of the manuscript.

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Cardiac remodeling in patients with Marfan syndrome: impact of gender and vasodilator therapy

Remodelado cardiaco asociado con el síndrome de Marfan: impacto del sexo y el tratamiento vasodilatador

To the Editor,

Marfan syndrome (MS) is caused by a fibrillin-1 mutation, a component of the aorta and regulator of the signaling pathway of transforming growth factor beta. The aim of prescribing medications is to reduce the effects of fibrosis and blood pressure (BP) and prevent aortic dissection. Recent data support the existence of myocardial involvement in the presence of valves with normal function. Pathological myocardial remodeling is seen in the form of hypertrophy or dilatation, fibrosis, and left ventricular dysfunction. Systolic dysfunction occasionally remains subclinical and is only detected on myocardial strain measurement.[1] Postsystolic thickening (PST), which can be detected on echocardiography, has been described in states of pressure overload or myocardial ischemia.[1] The recently described high prevalence of PST among patients with MS even when BP is within normal limits indicates that the cardiomyopathy of MS has characteristics similar to states of pressure overload.[2]

Table 1
Comparison of postsystolic thickening by sex

	Women (n = 22)	Men (n = 23)	*P*	Women with PST+, n = 16 (73%)	Women with PST−, n = 6 (27%)	*P*	Men with PST+, n = 14 (61%)	Men with PST−, n = 9 (39%)	*P*
Age, y	29 ± 7	28 ± 8	0.7	31.2 ± 6.6	23.9 ± 7.2	0.059	30.3 ± 7.8	25.3 ± 7.7	.18
Aortic root, indexed, mm/m^2	20.6 ± 3	20.8 ± 2.8	0.83	20.5 ± 3.3	20.9 ± 2.5	0.59	21.1 ± 2.3	20.2 ± 3.5	.56
LVEDD, indexed, mm	26.1 ± 3.4	25.3 ± 4	0.50	25.3 ± 3	28.3 ± 2.8	0.021	25.4 ± 4.7	25.2 ± 3.1	.88
LVESD, indexed, mm	16.3 ± 2.5	16.2 ± 3.7	0.87	15.6 ± 2.5	17.6 ± 2.2	0.11	16.4 ± 4	15.8 ± 3.3	.98
LVEF, %	62.6 ± 5	60.0 ± 5.4	0.09	61.9 ± 4	64.2 ± 7.1	0.49	59.2 ± 5.8	60.9 ± 4.9	.40
Systolic BP, mmHg	118.9 ± 10	118.4 ± 13	0.89	120.8 ± 10	114 ± 9.7	0.23	117 ± 15	120.9 ± 10	.48
Diastolic BP, mmHg	73.4 ± 9	71.5 ± 9	0.51	75.8 ± 8.4	66.8 ± 8.4	0.049	71 ± 10	72.6 ± 8.5	.69
On treatment	13 (59)	17 (74)	0.29	9 (56)	4 (67)	1	13 (93)	4 (44)	.018

BP, blood pressure; LVEDD, left ventricular end-diastolic diameter; LVEF, left ventricular ejection fraction; LVEST, left ventricular end-systolic diameter; PST, postsystolic thickening; treatment, beta blockers or losartan.
Significant if $P < .05$. Continuous variables are shown as absolute numbers and were analyzed using the Student t test for independent samples and are expressed as mean ± standard deviation. The only categorical variable (on treatment) was analyzed using chi-squared test and is presented as No. (%).

The limited literature on the impact of sex on dilatation and aortic events in MS presents even more contentious results. While some authors advocate lowering the cutoff point for aortic dilatation by 5 mm for the indication for surgery in women,[3] others postulate that men are at higher risk of aortic dissection.[4,5]

In our study, we hypothesized on the impact of sex differences on the manifestation of signs of cardiomyopathy in MS. We analyzed the echocardiograms of 45 young patients with MS to detect potential sex differences in the presence of PST and its associated factors, such as BP and medication. The study was approved by the *Academisch Medisch Centrum* hospital Ethics Committee (Amsterdam) and the participants gave informed consent. All were seen in an MS clinic by a cardiologist and nurse, both specialized in the area. The cardiologist's decision to prescribe vasodilators was made on a case-by-case basis, depending on patterns in the patient's BP. The comparison by sex and by presence of PST is shown in table 1.

No differences were found between men and women (23 and 22, respectively) in terms of age, aortic dimensions, left ventricular dimensions, left ventricular ejection fraction, or systolic or diastolic BP. However, when we studied the presence of PST, we found that 16 women (73%) and 14 men (61%) had PST. Seventeen men (74%) and 13 women (59%; $P = .292$) were on medication. This difference in pharmacological treatment of patients with MS became even more evident when we examined the difference between the subgroups of women with MS and PST+ and men with MS and PST+: significantly fewer women were medicated (9 [56%] vs 13 [93%]; $P = .039$) despite the absence of significant differences in aortic dilatation or ventricular dimensions. Understandably, their BP was approximately 5 mmHg higher (women with MS and PST+: systolic BP, 120.8 ± 10 mmHg and diastolic BP, 75.8 ± 8 mmHg; men with MS and PST+: systolic BP, 117 ± 15 mmHg mmHg and diastolic BP, 71 ± 10 mmHg; $P = .4$ and $P = .157$, respectively). This difference in BP reached statistical significance despite the small sample size when we compared women with MS and PST+ vs women with MS without PST: diastolic BP of 75.8 ± 8 vs 66.8 ± 8 mmHg ($P = .049$) and systolic BP 120.8 ± 10 vs 114 ± 9.7 mmHg ($P = .23$).

In our group of patients with MS there was a high prevalence of PST, higher in women, without reaching statistical significance. The affected women had a higher presence of abnormal strain than the men with MS. This characteristic was combined with women being less likely to receive treatment despite the aortic and ventricular diameters being very similar between the sexes. It is difficult to explain in this study why the women with MS were less likely to receive pharmacological treatment. However, this difference has repercussions for BP, as the diastolic BP was higher in the women with PST+ than those without it.

In this series, female patients were less likely to receive treatment but had more left ventricular remodeling. The reasons for these differences should be studied in future research, and a common effort should be made to improve the treatment rates for these patients.

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AUTHORS' CONTRIBUTIONS

A. Mas-Stachurska contributed to the study design and data collection, analysis, and interpretation; she wrote the manuscript and is responsible for all aspects of the article and agrees to investigate and resolve any issues related to the accuracy and truthfulness of any part of this work.

R. de Bruin, B.J. Bouma, B. Mulder, and B. Bijnens contributed substantially to the study concept and design and data collection, analysis, and interpretation, and critically reviewed the content.

M. Sitges made substantial contributions to the study concept and design, analysis and interpretation, and writing the article, and critically reviewed its intellectual content, and also accepts responsibility for all aspects of the article and agrees to investigate and resolve any issues related to the accuracy and truthfulness of any part of this work.

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None of the authors have any conflicts of interest to declare.

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