Myocardial Deformation Imaging by Two-Dimensional Speckle-Tracking Echocardiography in Comparison to Late Gadolinium Enhancement Cardiac Magnetic Resonance for Analysis of Myocardial Fibrosis in Severe Aortic Stenosis

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Myocardial deformation analysis by speckle-tracking echocardiography (STE) has been used for analysis of myocardial viability and myocardial fibrosis. Patients with severe aortic stenosis are known to develop myocardial fibrosis. This study evaluated the association between myocardial fibrosis determined by late gadolinium enhancement (LGE) cardiac magnetic resonance (CMR) and 2-dimensional STE in patients with severe aortic stenosis. In 30 patients (78 ± 7 years) with severe aortic stenosis (mean gradient 53 ± 21 mm Hg), peak systolic circumferential strain based on 2-dimensional echocardiographic parasternal short-axis views and peak systolic longitudinal strain based on apical views were determined for analysis of regional function. LGE CMR was performed to define the amount of fibrosis in each segment within 24 hours of echocardiography. Relative amount of fibrosis was determined based on LGE CMR as gray-scale threshold 6 SDs above the mean signal intensity of the normal remote myocardium. There was a decrease in LGE from base to apex (14.4 ± 8.7% for basal segments, 3.4 ± 3.0% for midventricular segments, and 2.1 ± 3.0% for apical segments; $p < 0.001$). Simultaneously, there was an increase in myocardial deformation expressed as peak systolic longitudinal strain from base to apex (−11.6 ± 7.0% for basal segments, −16.9 ± 6.5% for midventricular segments, and −17.4 ± 7.7% for apical segments; $p = 0.001$). There was a negative correlation between the amount of myocardial fibrosis determined by LGE CMR and peak systolic longitudinal strain for the total left ventricle ($r = -0.538$, $p = 0.007$). Myocardial fibrosis defined as LGE >10% could be identified by peak systolic longitudinal strain less than −11.6%, with a sensitivity of 65% and a specificity of 75% (area under the receiver operating characteristic curve 0.69). In conclusion, myocardial fibrosis increases from apical to basal left ventricular segments in patients with severe aortic stenosis. There is an association between severity of myocardial fibrosis defined by LGE CMR and myocardial deformation by STE. **(Am J Cardiol 2014;114:1083−1088)**

In patients with aortic stenosis, left ventricular (LV) hypertrophy and interstitial myocardial fibrosis are known sequel of chronic pressure overload.[1,2] The changes result in gradual impairment of systolic and diastolic function and may result in the typical symptoms of angina pectoris and dyspnea.[3,4] Typical parameters to evaluate aortic stenosis include hemodynamic parameters such as pressure gradients, aortic valve area, and LV ejection fraction. In contrast, alterations of myocardial texture resulting from aortic stenosis such as myocardial fibrosis are hardly evaluated in clinical practice as there is no imaging tool easily providing information about fibrotic changes. Late gadolinium enhancement (LGE) cardiac magnetic resonance (CMR) imaging has been described as a tool for reliable analysis of myocardial fibrosis.[5−7] Speckle-tracking echocardiography (STE)[8,9] allows accurate analysis of regional myocardial function. It has been found to accurately reflect myocardial viability and extent of necrosis in ischemic cardiomyopathy.[10,11] There are only little data on the potential of STE to define myocardial function and the degree of myocardial fibrosis in patients with severe aortic stenosis. This study evaluated the extent and distribution of myocardial fibrosis in patients with symptomatic severe aortic stenosis using LGE CMR. Furthermore, findings of LGE CMR were compared with those obtained by 2-dimensional (2D) STE.

Methods

We screened 42 consecutive patients with symptomatic aortic stenosis undergoing cardiac catheterization. Contraindications for LGE CMR such as device therapy or severe renal dysfunction and insufficient echocardiographic

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Table 1
Patient characteristics, late gadolinium enhancement CMR data as well as longitudinal and circumferential myocardial strain data at the basal, midventricular and apical level for each of the 30 patients

Patient	Age	Gender	NYHA	LVEDV	LVESV	LV-EF	CMR Mass	Mean Grad	Max Grad	AVA	LE Bas	LE Mid	LE Apic	LE Total	Circ Bas	Long Bas	Circ Mid	Long Mid	Circ Apic	Long Apic	Circ Glob	Long Glob
1	71	M	2	128	49	62	216	70	88	0.5	10	7	3	8	13.7	7.0	13.8	9.5	18.7	10.2	15.4	8.9
2	75	M	3	142	57	60	190	50	73	0.8	9	2	2	5	24.8	17.2	24.5	17.2	27.7	18.2	25.7	17.5
3	71	M	3	171	88	49	245	36	60	1.1	14	4	2	9	10.0	13.2	9.2	22.3	15.8	23.4	11.7	19.6
4	71	M	4	146	79	46	319	56	77	0.7	28	5	1	11	10.0	6.8	9.6	12.4	20.5	13.1	13.4	11.2
5	70	F	3	138	53	62	197	80	131	0.5	12	2	0	7	23.3	16.0	23.5	23.2	16.6	25.0	21.1	21.4
6	79	F	3	109	49	55	87	51	76	0.6	0	0	0	0	27.0	31.2	27.4	29.8	22.3	35.5	25.6	32.1
7	87	F	3	133	65	51	90	42	58	0.9	0	0	0	0	14.0	16.9	14.3	21.2	16.7	13.0	15.0	19.0
8	89	M	3	89	35	61	139	66	90	0.7	26	0	0	10	21.7	15.0	21.6	31.9	32.2	38.0	25.2	28.3
9	67	M	3	191	82	57	244	68	100	0.8	4	0	0	2	19.6	21.3	15.5	24.2	19.3	25.0	18.1	23.4
10	74	M	3	215	98	54	215	65	100	0.6	16	8	3	10	14.8	6.9	16.1	13.2	15.8	13.1	15.6	11.1
11	89	F	4	85	34	60	92	24	37	1.0	26	7	5	13	12.3	1.0	12.7	16.2	19.4	16.1	14.8	11.1
12	85	F	3	160	66	59	210	87	88	0.5	9	4	2	6	14.6	7.0	14.8	11.8	20.7	12.4	16.7	10.4
13	83	M	3	156	66	58	190	51	85	0.8	1	0	0	0	16.2	12.1	18.0	16.7	11.4	16.9	15.2	15.2
14	82	F	4	184	100	46	265	59	90	0.8	20	7	3	12	17.3	10.2	16.8	17.9	17.2	20.1	17.1	16.0
15	76	F	3	101	42	58	162	56	85	0.8	11	9	13	12	17.1	4.8	17.2	5.9	22.4	6.3	18.9	5.7
16	79	M	3	116	52	55	210	55	80	0.8	10	1	0	4	23.3	19.0	23.6	17.2	20.2	17.3	22.4	17.8
17	72	M	3	160	71	56	254	52	90	0.8	11	2	0	5	9.2	15.1	8.5	11.1	12.7	8.3	10.1	11.5
18	80	F	4	356	265	26	283	24	40	1.3	13	5	2	8	13.2	12.8	13.4	8.9	14.7	9.3	13.8	10.4
19	81	F	3	128	53	59	156	90	105	0.4	22	5	0	10	17.0	8.2	16.8	21.2	20.7	21.2	18.2	16.8
20	83	F	3	100	37	63	134	81	99	0.6	30	9	2	17	14.0	4.0	13.5	8.3	13.5	7.9	13.7	6.7
21	68	M	3	175	70	60	181	48	68	0.9	32	8	7	19	18.3	2.9	18.4	10.9	25.4	11.2	20.7	8.3
22	76	M	4	199	132	34	237	30	55	1.0	12	0	1	4	20.5	13.0	22.1	14.3	18.8	14.1	20.5	13.8
23	87	M	4	227	166	27	283	35	55	0.6	16	2	9	9	12.0	6.2	11.7	10.6	10.7	11.1	11.5	9.2
24	78	F	3	80	26	67	87	77	87	0.6	8	3	0	5	20.0	12.8	21.4	19.1	20.2	19.2	20.5	17.1
25	83	M	3	89	32	64	149	14	26	1.3	20	0	0	9	19.8	5.0	19.6	18.3	19.2	18.3	19.5	13.9
26	65	F	3	134	60	55	155	50	76	0.8	13	5	3	8	24.6	17.0	24.3	17.8	24.2	18.2	24.4	17.7
27	87	M	1	150	54	64	141	31	48	0.8	3	3	0	2	23.5	25.3	23.5	15.0	14.3	14.9	20.4	18.3
28	67	M	3	188	86	54	236	61	91	0.6	16	0	0	7	18.0	11.8	18.0	14.8	14.5	13.1	16.8	13.3
29	73	M	3	167	72	57	211	59	73	0.8	21	1	4	10	11.3	3.0	10.9	29.3	26.5	31.4	16.2	21.2
30	77	F	3	148	62	58	136	23	32	1.2	20	2	2	9	10.0	6.0	10.0	17.6	20.3	19.0	13.4	14.2
Mean	78	17 m		152	73	55	190	53	75	0.79	14.4	3.4	2.1	7.7	17.0	11.6	17.0	16.9	19.1	17.4	17.7	15.4
SD	7			55	47	10	63	20	24	0.23	8.7	3.0	3.0	4.6	5.1	7.0	5.3	6.5	5.0	7.7	4.3	6.1

Apic = apical level; AVA = aortic valve area (cm^2); Bas = basal left ventricular level; Circ (in %) = average peak circumferential strain of a left ventricular level; Circ glob (in %) = average peak circumferential strain of all left ventricular segments; CMR mass (in g) = left ventricular mass by cardiac magnetic resonance; LE (in %) = late enhancement; Long (in %) = average peak circumferential strain of a left ventricular level; Long glob (in %) = average peak longitudinal strain of all left ventricular segments; LV-EF (in %) = left ventricular ejection fraction; LVEDV (in ml) = left ventricular end-diastolic volume; LVESV (in ml) = left ventricular end-systolic volume; Max grad (in mm Hg) = maximum gradient of the aortic valve; Mean grad (in mm Hg) = mean gradient of the aortic valve; Mid = midventricular level; NYHA = New York Heart Association class.