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MRI regional strain analysis in patients with hypertrophic cardiomyopathy

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Abstract

Purpose: To describe regional left ventricular myocardial deformation in patients with hypertrophic cardiomyopathy (HCM) using cardiac MRI.

Methods: Twenty-five patients with HCM and 25 control participants with normal cardiac MRI underwent 1.5-T imaging. Regional radial, circumferential and longitudinal deformation was assessed using feature tracking. For this correction, the published aggregate results were reviewed descriptively. Individual-level data were unavailable for independent reanalysis.

Results: The tabulated mean radial strain was 20.10% in hypertrophied HCM segments, 27.71% in HCM segments of apparently normal thickness and 30.56% in controls. Corresponding circumferential values were −11.38%, −14.96% and −17.22%; longitudinal values were −5.95%, −12.21% and −13.59%. The tables reported 400 HCM segments for radial and circumferential analysis and 392 for longitudinal analysis. Statistical significance and previously reported AI classification performance could not be independently verified.

Conclusion: The reported aggregate values suggest reduced deformation magnitude in hypertrophied segments, with intermediate values in HCM segments of apparently normal thickness. These observations are descriptive and hypothesis-generating. They do not establish diagnostic accuracy, treatment-monitoring benefit or performance in genotype-positive individuals without hypertrophy.

Keywords: hypertrophic cardiomyopathy; regional strain analysis; cardiac MRI

Version information and declarations appear on page 47.

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Introduction

Hypertrophic cardiomyopathy (HCM) is a heterogeneous myocardial disease associated with a broad range of clinical outcomes. Estimates of its prevalence depend on the population studied and the methods used to identify affected individuals. Population-based analyses and reviews have described its clinical and public-health importance [1, 2].

HCM may be inherited and is often associated with variants in genes encoding sarcomeric proteins. Its phenotypic expression varies substantially, and affected individuals may be asymptomatic or present with symptoms, arrhythmia or heart failure. Cardiac MRI provides information about the distribution of hypertrophy, ventricular function and myocardial tissue characteristics [3, 4].

Histopathological findings include cardiomyocyte hypertrophy, myocyte disarray, interstitial or replacement fibrosis and abnormalities of the intramural coronary arteries. These structural changes provide a context for investigating regional myocardial deformation, although imaging-derived strain is not itself a histological measurement [5, 6].

Global ejection fraction and regional deformation describe different aspects of ventricular performance. A preserved or high ejection fraction does not necessarily imply normal regional myocardial mechanics. The clinical course of HCM also varies, and adverse remodeling with impaired systolic function may occur in some patients [8, 9].

Molecular hypercontractility, myocardial deformation and global chamber emptying should not be treated as interchangeable concepts. Mechanistic accounts of sarcomeric hypercontractility address a different level of myocardial function from the regional imaging measurements reported here [7]. This study does not directly test molecular contractile mechanisms.

Regional tissue tracking permits the description of displacement, strain, systolic and diastolic strain rates and velocities, and time to peak displacement and strain. Measurements in radial, circumferential and longitudinal directions can help characterize spatial variation within the ventricle. In this article, these parameters are considered as descriptive imaging measurements.

The purpose of the study was to describe regional left ventricular deformation in hypertrophied HCM segments and in HCM segments of apparently normal thickness, with comparison to participants whose cardiac MRI was normal. The corrected interpretation focuses on the reported aggregate measurements and their limitations.

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Materials and Methods

Study population

The study was approved by the ethical committee of Ain Shams University Hospitals. It included 25 patients with HCM and 25 control participants. The original study description states that control participants were investigated for suspected abnormalities but had normal MRI examinations.

The HCM group comprised 21 male and four female participants, with a mean age of 39.4 years (range, 7-65 years). The control group comprised 16 male and nine female participants, with a mean age of 33 years (range, 13-56 years).

The study reported the following inclusion criteria, with reference to the clinical guidance and pediatric measurement literature cited in the original article [10, 11]: wall thickness of at least 15 mm in the absence of another cause of hypertrophy; end-diastolic wall thickness of at least 13 mm with a family history of HCM; focal asymmetric basal anterior septal hypertrophy with a septal-to-posterior-wall thickness ratio above 1.3 in a normotensive patient; or, in pediatric participants, a wall-thickness Z-score above +2 with the stated adjustment for sex and body surface area.

Exclusion criteria included hypertension, diabetes, amyloidosis, sarcoidosis and other conditions contributing to myocardial hypertrophy. For controls, the original report also specified exclusion of underlying disease even when controlled and uncomplicated.

Preparation and monitoring

The reported protocol included explanation of the examination and possible contrast reactions, screening for MRI-incompatible devices and review of renal function. It specified avoidance of contrast administration at an estimated glomerular filtration rate below 30 mL/min/1.73 m^2. Physiological monitoring, hearing protection, an ECG signal for cardiac gating and access to an emergency management plan were described. These statements reproduce the study protocol and are not presented as a current clinical practice guideline.

Scanner and cine imaging

Imaging was performed on a Siemens MAGNETOM Aera 1.5-T scanner. Two- and four-chamber long-axis views and short-axis and/or axial stacks covered both ventricles from base to apex. Retrospectively ECG-gated steady-state free-precession cine imaging was acquired during breath-holding when possible and during free breathing otherwise. Thirty reconstructed phases per R-R interval were reported; prospective gating with a minimum of 18 phases was used in some patients with arrhythmia.

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MRI protocol

Cine acquisition parameters

The reported cine parameters were repetition time (TR), 51 ms; echo time (TE), 1.4 ms; flip angle, 50°; slice thickness, 7-8 mm without an interslice gap; and matrix, 156 × 256. Field of view and voxel size were adjusted to patient size. Breath-holding was preferred where possible; four to five signal averages were described for free-breathing acquisition.

T2-weighted imaging

ECG-gated, fat-suppressed triple-inversion-recovery short-axis stacks were acquired across both ventricles. The reported parameters were TR >2000 ms; TE >60 ms; flip angle, 90°; inversion time, 120-170 ms; slice thickness, 8 mm with a gap; and matrix, 208 × 208. Acquisition was performed during breath-holding where possible or free breathing with two to three signal averages. The field of view and voxel size were adjusted to patient size.

Phase-contrast flow assessment

Aortic and subaortic left ventricular outflow tract phase-contrast imaging was used to assess flow and peak systolic velocity. The protocol described free-breathing acquisition with two signal averages, retrospective ECG gating and 30 reconstructed phases per R-R interval. The reported parameters were TR, 105.2 ms; TE, 2.97 ms; flip angle, 20°; slice thickness, 8 mm; and matrix, 119 × 192. Aortic velocity encoding started at 150 cm/s.

Contrast administration and late gadolinium enhancement

Gadopentetate dimeglumine (Magnevist, Bayer) was administered at a dose of 0.2 mL/kg (0.1 mmol/kg). Late gadolinium enhancement images were acquired 5-10 minutes after administration.

The available sequences included free-breathing single-shot late gadolinium enhancement and phase-sensitive inversion recovery performed with breath-holding or free breathing with additional averages. Images were acquired in the same orientation as the cine images. The reported parameters were TR, 906.4 ms; TE, 1.3 ms; flip angle, 40°; slice thickness, 8 mm; and matrix, 122 × 192. Acquisition was performed in diastole, triggered every three to four heartbeats. Field of view and voxel size were adjusted to patient size.

Ventricular and flow measurements

The reported post-processing included end-diastolic and end-systolic volumes, stroke volume, ejection fraction and myocardial mass, with body-surface-area-indexed values where applicable. Ventricular values were compared with published reference ranges [14, 15]. Aortic forward and backward flow, net flow, peak velocity and regurgitant fraction were assessed. Mitral and tricuspid regurgitation were assessed indirectly from ventricular stroke volume and forward aortic or pulmonary flow, as described in the original protocol.

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MR image analysis

Regional myocardial deformation was assessed using cmr42 (version 5.11.4; Circle Cardiovascular Imaging, Calgary, Canada) and a 16-segment American Heart Association model. Endocardial and epicardial contours were manually traced at end systole and end diastole and propagated through the remaining phases. Papillary muscles were excluded from strain assessment and included in ventricular volumetric analysis. The regional parameters included displacement, strain, systolic and diastolic strain rates and velocities, and time to peak displacement and strain in the radial, circumferential and longitudinal directions.

Statistical reporting

The original report described analysis of variance and t tests, with a significance threshold of $p < 0.05$. For this correction, the submitted manuscript, submitted tables and published article were reviewed. Individual measurements linked to patients and the original analysis code were not available to the editors for independent reanalysis. The aggregate results are therefore presented descriptively, without newly verified inferential statistics. The published statistical description does not establish how dependence among segments from the same participant was handled.

Results

The radial and circumferential analyses were reported with 220 hypertrophied segments and 180 segments of apparently normal thickness in the HCM group, for a total of 400 segments (Tables 1 and 2). The longitudinal analysis was reported with 216 hypertrophied and 176 apparently normal-thickness segments, for a total of 392 (Table 3). These denominators follow the original tables, whose total means and standard deviations are internally consistent with the tabulated group sizes and aggregate statistics. The original report stated 400 segments for the control group.

In the order hypertrophied HCM segments, HCM segments of apparently normal thickness, and control segments, the tabulated mean radial strain values were 20.10%, 27.71% and 30.56%. Corresponding circumferential values were −11.38%, −14.96% and −17.22%; longitudinal values were −5.95%, −12.21% and −13.59%. These are rounded summaries of the previously reported means, not results from a new analysis. Across the three directions, the reported strain magnitudes were lowest in hypertrophied segments and intermediate in HCM segments of apparently normal thickness. The available information is insufficient to independently verify the original significance claims or to quantify participant-level uncertainty.

The detailed regional measurements are presented in Table 1 (radial), Table 2 (circumferential) and Table 3 (longitudinal). The reported control means are shown in Table 4. Figure 1 reproduces the illustrative case supplied with the original article.

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Discussion

The tabulated strain values are compatible with heterogeneous regional deformation in HCM. The hypertrophied segments showed lower deformation magnitudes than the control segments in all three reported directions, while segments of apparently normal thickness had intermediate values. These aggregate observations do not establish a specific molecular mechanism of myocardial contraction or prove the absence of compensatory function.

Interpretation is limited by the small cohort and the absence of individual-level data for independent verification. Multiple segments originate from the same participant, and the published statistical description does not establish how this dependence was handled. The comparator group was described as individuals with normal MRI after investigation for suspected abnormalities, which also limits generalization to an unselected healthy population. The study does not report treatment-response follow-up or a defined cohort of genotype-positive individuals without hypertrophy. Clinical utility in either setting therefore remains unestablished. Reproducible patient-level analyses and appropriately designed follow-up studies would be needed to test these questions.

Previous research has evaluated echocardiographic functional parameters and global longitudinal strain in relation to outcomes in HCM [12, 13]. Those studies address prognostic questions using their own populations and follow-up. Their results should not be taken as a validation of the regional comparisons in the present cohort or of a classifier trained on this dataset.

The distinction between imaging deformation and molecular contraction is also relevant to the interpretation of apparently preserved ejection fraction. The present measurements cannot resolve the mechanistic questions discussed in the molecular and clinical literature [7, 8, 9]. The revised conclusion is therefore restricted to the observed pattern of reported aggregate strain values.

Conclusion

The reported aggregate values suggest reduced deformation magnitude in hypertrophied segments, with intermediate values in HCM segments of apparently normal thickness. These observations are descriptive and hypothesis-generating. They do not establish diagnostic accuracy, treatment-monitoring benefit or performance in genotype-positive individuals without hypertrophy.

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Table 1. Radial measurements in HCM

Reported segment-level means and standard deviations. HCM: hypertrophic cardiomyopathy.

Parameter	Segment group	N	Mean	SD
Displacement (mm)	Hypertrophied	220	3.7500	2.06423
	Apparently normal	180	5.3017	2.45007
	Total	400	4.4483	2.37259
Strain (%)	Hypertrophied	220	20.0977	13.09198
	Apparently normal	180	27.7128	16.94304
	Total	400	23.5245	15.40289
Peak diastolic strain rate (1/s)	Hypertrophied	220	-1.2455	1.05589
	Apparently normal	180	-1.6506	1.29574
	Total	400	-1.4278	1.18569
Peak diastolic velocity (mm/s)	Hypertrophied	220	-22.5109	13.05833
	Apparently normal	180	-25.9861	14.34507
	Total	400	-24.0748	13.74438
Peak systolic strain rate (1/s)	Hypertrophied	220	1.4527	1.37214
	Apparently normal	180	2.0639	1.35691
	Total	400	1.7278	1.39717
Peak systolic velocity (mm/s)	Hypertrophied	220	26.8573	16.86500
	Apparently normal	180	33.6544	16.54626
	Total	400	29.9160	17.04116
Time to peak displacement (ms)	Hypertrophied	220	354.6468	106.16875
	Apparently normal	180	336.5578	85.54762
	Total	400	346.5068	97.73002
Time to peak strain (ms)	Hypertrophied	220	339.2036	111.04739
	Apparently normal	180	344.1128	93.94762
	Total	400	341.4128	103.60508

N is the reported number of myocardial segments, not the number of independent participants. Apparently normal denotes HCM segments of apparently normal wall thickness. Total denotes the two HCM segment groups combined. Means and standard deviations are retained as reported and interpreted descriptively.

Displacement and velocity units have been standardized by direction: mm and mm/s for radial and longitudinal measurements; deg and deg/s for circumferential measurements. Strain rate is expressed in 1/s and time to peak in ms.

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Table 2. Circumferential measurements in HCM

Reported segment-level means and standard deviations. HCM: hypertrophic cardiomyopathy.

Parameter	Segment group	N	Mean	SD
Displacement (deg)	Hypertrophied	220	1.4095	3.79885
	Apparently normal	180	1.2656	4.54223
	Total	400	1.3448	4.14511
Strain (%)	Hypertrophied	220	-11.3777	5.97949
	Apparently normal	180	-14.9606	7.67467
	Total	400	-12.9900	7.01667
Peak diastolic strain rate (1/s)	Hypertrophied	220	0.6950	0.58686
	Apparently normal	180	0.9717	0.75667
	Total	400	0.8195	0.68182
Peak diastolic velocity (deg/s)	Hypertrophied	220	-8.1750	30.74744
	Apparently normal	180	-4.2944	35.11116
	Total	400	-6.4288	32.79787
Peak systolic strain rate (1/s)	Hypertrophied	220	-0.9041	0.72549
	Apparently normal	180	-1.2789	0.73970
	Total	400	-1.0728	0.75446
Peak systolic velocity (deg/s)	Hypertrophied	220	7.9386	38.07865
	Apparently normal	180	3.6900	43.46649
	Total	400	6.0268	40.59472
Time to peak displacement (ms)	Hypertrophied	220	339.4059	167.06305
	Apparently normal	180	323.9167	152.94768
	Total	400	332.4358	160.85144
Time to peak strain (ms)	Hypertrophied	220	337.5836	121.95465
	Apparently normal	180	347.5556	99.16645
	Total	400	342.0710	112.24861

N is the reported number of myocardial segments, not the number of independent participants. Apparently normal denotes HCM segments of apparently normal wall thickness. Total denotes the two HCM segment groups combined. Means and standard deviations are retained as reported and interpreted descriptively.

Displacement and velocity units have been standardized by direction: mm and mm/s for radial and longitudinal measurements; deg and deg/s for circumferential measurements. Strain rate is expressed in 1/s and time to peak in ms.

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Table 3. Longitudinal measurements in HCM

Reported segment-level means and standard deviations. HCM: hypertrophic cardiomyopathy.

Parameter	Segment group	N	Mean	SD
Displacement (mm)	Hypertrophied	216	2.8569	2.41196
	Apparently normal	176	2.9801	3.04526
	Total	392	2.9122	2.71169
Strain (%)	Hypertrophied	216	-5.9542	11.59742
	Apparently normal	176	-12.2125	11.14986
	Total	392	-8.7640	11.80314
Peak diastolic strain rate (1/s)	Hypertrophied	216	0.4995	1.25125
	Apparently normal	176	0.7847	1.27308
	Total	392	0.6276	1.26746
Peak diastolic velocity (mm/s)	Hypertrophied	216	-17.8653	21.10452
	Apparently normal	176	-20.5591	23.45234
	Total	392	-19.0747	22.20095
Peak systolic strain rate (1/s)	Hypertrophied	216	-0.7662	1.64343
	Apparently normal	176	-1.0784	1.28969
	Total	392	-0.9064	1.50125
Peak systolic velocity (mm/s)	Hypertrophied	216	20.4245	24.51892
	Apparently normal	176	21.0784	27.04525
	Total	392	20.7181	25.65249
Time to peak displacement (ms)	Hypertrophied	216	369.5435	144.80205
	Apparently normal	176	359.6636	139.15729
	Total	392	365.1077	142.19992
Time to peak strain (ms)	Hypertrophied	216	354.1847	163.71557
	Apparently normal	176	362.4784	155.10324
	Total	392	357.9084	159.75723

N is the reported number of myocardial segments, not the number of independent participants. Apparently normal denotes HCM segments of apparently normal wall thickness. Total denotes the two HCM segment groups combined. Means and standard deviations are retained as reported and interpreted descriptively.

Displacement and velocity units have been standardized by direction: mm and mm/s for radial and longitudinal measurements; deg and deg/s for circumferential measurements. Strain rate is expressed in 1/s and time to peak in ms.

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Table 4. Regional measurements in controls

Reported mean values in control participants with normal cardiac MRI.

Parameter	Radial	Circumferential	Longitudinal
Displacement*	5.41	1.42	3.64
Strain (%)	30.56	-17.22	-13.59
Peak diastolic strain rate (1/s)	-1.62	0.88	0.76
Peak diastolic velocity*	-27.25	-6.73	-20.07
Peak systolic strain rate (1/s)	1.91	-1.12	NR†
Peak systolic velocity*	30.74	16.81	25.53
Time to peak displacement (ms)	338.22	294.97	316.59
Time to peak strain (ms)	331.93	333.49	341.89

* Displacement: mm for radial and longitudinal measurements, deg for circumferential measurements. Velocity: mm/s for radial and longitudinal measurements, deg/s for circumferential measurements.

† NR: not reliably interpretable. The original longitudinal peak systolic strain-rate cell reads "-.0.99", which is not a valid numerical entry. No replacement value has been inferred.

The original report stated 25 control participants and 400 control segments. This table supplies means only; standard deviations and participant-level uncertainty were not provided. The original mean values are retained, except that the invalid numerical entry is identified explicitly.

Reading the descriptive results

The mean strain values used in the abstract and main Results are reproduced directly from Tables 1-4, with rounding to two decimal places. Negative circumferential and longitudinal values represent the reported sign convention; comparisons of deformation magnitude use their absolute size. No new significance tests or diagnostic accuracy estimates are derived from this table.

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Descriptive overview

Table 5. Summary of reported mean strain values

Direction	Hypertrophied HCM	Apparently normal HCM	Controls
Radial	20.10%	27.71%	30.56%
Circumferential	-11.38%	-14.96%	-17.22%
Longitudinal	-5.95%	-12.21%	-13.59%

Summary of the previously reported mean strain values in Tables 1-4, rounded to two decimal places. This overview contains no new measurements, significance tests or AI classification results.

Declarations

Ethics and consent. The original article reported approval by the ethical committee of Ain Shams University Hospitals, written informed consent, protection of patient anonymity and conduct in accordance with the Declaration of Helsinki.

Funding and competing interests. The authors reported no funding for the submitted work and no financial or non-financial competing interests.

Data availability. The original article stated that data were available from the corresponding author upon reasonable request. Individual-level data and analysis code were not available to the editors during this correction. The revision is based on the submitted manuscript, submitted tables and published article.

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Version information. This revised version incorporates corrections to contrast-dose reporting, segment counts, unit labels and references. The original AI classification table is replaced by the descriptive overview above because the development and testing methods could not be independently verified. The abstract and conclusions are revised accordingly. The accompanying change notice records the changes in detail.

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Figure 1. Illustrative MRI case

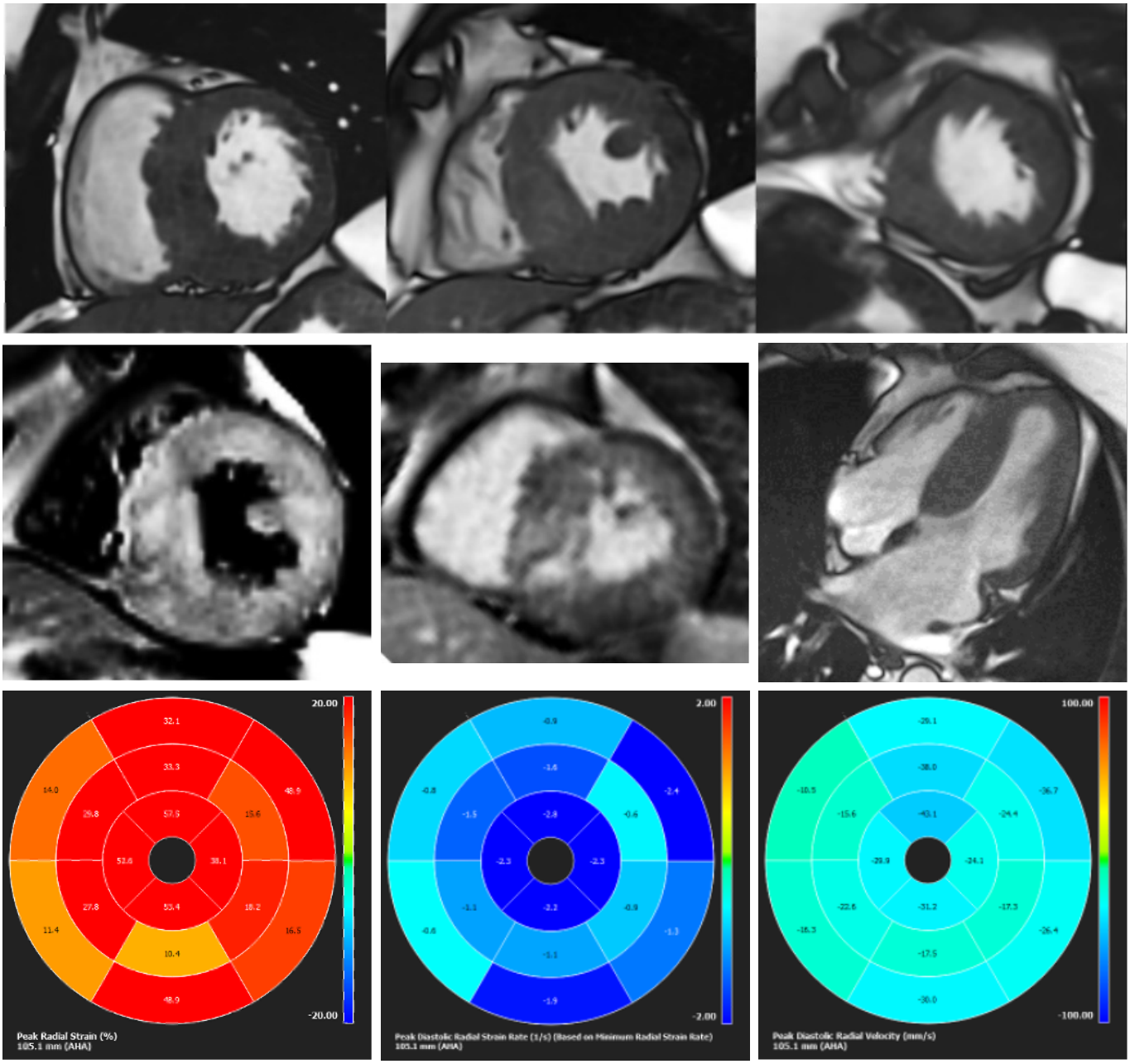

Figure 1A. Original illustrative case of a 44-year-old man with asymmetric left ventricular hypertrophy involving septal segments. The source caption describes patchy increased T2-weighted signal with corresponding late gadolinium enhancement. The lower panels show regional radial strain, peak diastolic radial strain rate and peak diastolic radial velocity. Images and displayed values are reproduced from the original article. The illustration does not independently establish histological tissue breakdown or statistical significance.

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Figure 1. Regional maps continued

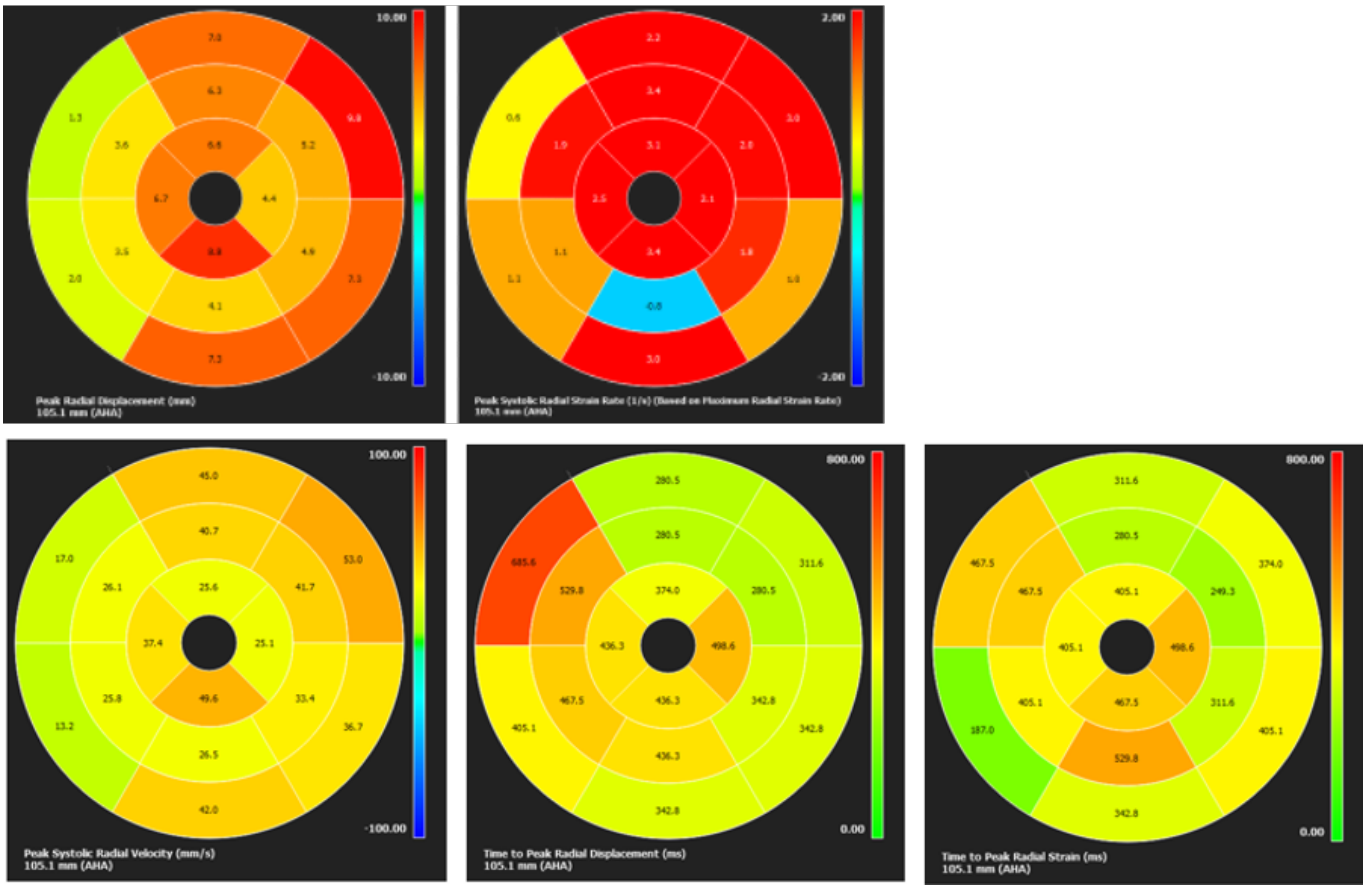

Figure 1B. Additional original polar maps from the same illustrative case. The upper row shows peak radial displacement and peak systolic radial strain rate. The lower row shows peak systolic radial velocity, time to peak radial displacement and time to peak radial strain. The source labels express displacement in mm, velocity in mm/s, strain rate in 1/s and timing in ms. Image content and numerical labels are unchanged.

The displayed maps provide an illustration of regional measurements in one patient. They are not a separate test cohort or a validation of the aggregate comparisons.