MRI Regional Strain Analysis In Patients With Hypertrophic Cardiomyopathy

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Abstract

Purpose: *To evaluate the regional left ventricular myocardial strain in patient with hypertrophic cardiomyopathy (HCM) especially young apparently compensated patients by magnetic resonance imaging.*

Materials and Methods: *25 HCM patients representing all age groups and 25 healthy volunteers underwent 1.5 Tesla MRI examination for cardiac volumes, and mass, followed by regional strain analysis in radial, circumferential, and longitudinal directions as regard the displacement, strain, peak diastolic and systolic strain rate, peak diastolic and systolic velocity, time to peak displacement and time to peak strain.*

Results: *In the HCM group, hypertrophic segments showing delayed gadolinium enhancement (DGE) were significantly different from non-hypertrophic apparently normal showing no enhancement concerning most of the regional radial strain parameters.*
In longitudinal and circumferential directions, hypertrophic segments showing DGE were significantly different from apparently normal segments with no enhancement as regard the strain, peak diastolic and systolic strain rate. Compared to normal volunteers, the hypertrophic segments with DGE were significantly different concerning most of the radial and longitudinal strain parameters, while apparently normal segments with no enhancement don't present a similar significant difference.
In circumferential analysis, hypertrophic enhancing segments were significantly different as regard the strain, peak diastolic strain rate, peak systolic velocity, and time to peak displacement, while the apparently normal non-enhancing segments present difference concerning the strain, peak systolic velocity, and time to peak displacement.

Conclusion: *The hypertrophied segments are more affected than the segments of apparently normal thickness in HCM patients, especially in the radial direction, the apparently normal segments are also affected with no tendency of functional compensation.*
Our data show that HCM muscle fibers show mostly hypofunction and reduced contraction rather than hypercontraction and even the apparently normal segments are impaired to a certain degree in comparison to normal. These findings are essential in follow up studies especially with patient receiving myosin inhibitors as well as young patients presenting preserved cardiac function and for gene positive apparently normal with absent penetration of the gene in the form of a concentric thickening can be seen by imaging, do we need more sophisticated imaging process like strain analysis to ensure absent penetration.

Keywords: *hypertrophic cardiomyopathy, regional strain analysis , cardiac MRI*

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Introduction

Hypertrophic cardiomyopathy is an inherited autosomal dominant disease with an estimated prevalence of 1:200 affecting approximately 0.2–0.5% of the general population (1, 2).

It is heterogenous in nature with a broad spectrum of phenotypic expression and clinical presentation caused by more than 1500 mutations in 11 or more genes encoding cardiac sarcomere proteins, including cardiac β-myosin heavy chain, cardiac myosin binding protein C, and troponin T (3, 4).

HCM presents a wide spectrum of manifestations ranging from asymptomatic with normal life expectancy to ventricular arrhythmia, sudden cardiac death, and heart failure (4).

The histopathological findings in HCM include myocardial fibers disarray, hypertrophy of cardiomyocytes, myocyte disarray, interstitial and replacement fibrosis as well as abnormal intramural coronary arteries (5, 6).

Hemodynamics

The left ventricle in HCM patients usually presents a diastolic dysfunction and restrictive ventricular filling with normal or reduced EDVI, a reduced ESVI, a high-normal or pseudo normal EF. It is caused by the increased interstitial fibrosis and the slowed relaxation and increased stiffness of the hypertrophied myocardial segments.

There is a debate and controversy if the HCM muscle fibers show hypercontraction and so we need a treatment to reduce the myosin overactivation (7) or hypocontraction and inevitable heart failure (8) or it is an evolutive process.

Also, it comes to question about the gene positive apparently normal with absent penetration of the gene in the form of a concentric thickening can be seen by imaging, is this cohort normal? Or do we need more sophisticated imaging process like strain analysis to ensure absent penetration of the gene?

Regional systolic myocardial function is also commonly impaired, as detected by pulsed and tissue Doppler imaging, 3-dimensional speckle-tracking echocardiography, and magnetic resonance imaging. Finally systolic function declines, the left ventricular wall thins with adverse remodeling, the cavity dilates, and heart failure with reduced EF develops, sometimes referred to as burnt out HCM or stage IV of overt dysfunction (8, 9).

Measuring the regional segmental deformation and dysfunction by tissue tracking and strain analysis is more informative than simple measurement of EF in HCM patients as it can quantitatively measure segmental myocardium function in radial, circumferential, and longitudinal directions, by several parameters including displacement, strain, peak diastolic strain rate, peak diastolic velocity, peak systolic strain rate, peak systolic velocity, time to peak displacement and time to peak strain.

The purpose of the present work was to quantify and compare the regional myocardial strain in the hypertrophied segments and segments of apparently normal thickness in comparison to normal persons.

Materials and Methods

Study Population

Our study was approved by the ethical committee of Ain Shams University Hospitals, 25 HCM patients were included and 25 normal control persons who were included came to our institute with suspected abnormalities, yet their MRI examinations were normal.

According to American Heart Association (AHA) guidelines (10, 11), inclusion criteria were determined as:

1) Wall thickness equal or above 15 mm in the absence of other causes of hypertrophy.

2) End diastolic wall thickness equal or above 13 mm with a family history of HCM.

3) Focal asymmetric hypertrophy of the basal anterior septum, defined as septal/posterior wall thickness ratio above 1.3 in a normotensive patient.

4) In the pediatric population, hypertrophy is diagnosed when the myocardial wall thickness (or its corresponding Z-score) is greater than +2, adjusted for sex and body surface area (BSA).

The exclusion criteria included hypertension, diabetes, amyloidosis, sarcoidosis, any other pathology that contributes to myocardial hypertrophy.

HCM patients 21 male and 4 female; mean age 39.4-year, range 7: 65 years.

Healthy control 16 male and 9 female; mean age 33-year, range 13-56 years.

With exclusion criteria of hypertension, diabetes, or any underlying disease even if controlled and non-complicated.

MR Protocol
Preparation before procedure:
- Explanation of the procedure to the patient especially the side effects of the contrast agent including coldness, warmth, or pain at the injection site, nausea, vomiting, headache, paresthesias, dizziness and itching.

- They should be aware of the severe life-threatening anaphylactoid or nonallergic anaphylactic and nephropathic effect.

- Exclusion of any hazards like functioning MRI-incompatible pacemaker, etc.

- Review kidney function test to avoid nephrogenic systemic fibrosis which may happen in case of contrast administration with acute or chronic renal failure (contrast shouldn't be given in estimated glomerular filtration rate <30 ml/min/1.73 m2).

During procedure:
- Physiological monitoring devices and hearing protection are put in place.

- A high-quality electrocardiogram (ECG) signal is essential for optimum data quality in cardiac-gated sequences.

- The imaging coil should be chosen to maximize the signal-to-noise ratio over the body region to be examined.

- An appropriately equipped resuscitation cart and emergency management plan for the MR environment should be in place.

Technique of imaging:
- Machine used is Siemens MAGNETOM Aera 1.5T MRI Scanner

- White blood images:
They help in assessment of EF, EDV, ESV, EDVI, ESVI, SV, SVI, cardiac mass, and segmental motion pattern of both left and right ventricles.
- Long-axis 2 and 4 chamber views and short-axis (and or axial) Stacks, covering both ventricles from the base of the heart to the apex were acquired using a retrospective ECG-gated steady-state free precession sequence during breath hold in cooperative patients and with free breathing in uncooperative patients, the reconstructed phases per R-R interval are 30 phases, yet, some patients presented with arrhythmias need prospective gating with minimum accepted phases per R-R interval are 18 phases.

- Scan parameters were Repetition time (TR) 51 msec; echo time (TE) 1.4 msec; flip angle (FA) 50 degree; slice thickness 7-8 mm without slice gap; matrix is 156x256, field-of-view is rotated to avoid wrap according to the size of each patient and voxel size is modified according to the field of view.

- Respiratory motion artifact can be minimized by breath-holding (preferred when possible) or by acquiring 4-5 signal averages (= number of excitations) with the patient breathing.

- Black blood images (T2 fat suppression):
They help in assessment myocardial edema in cases of acute infarction, active inflammation, and tumors.

- Short-axis Stacks, covering both ventricles from the base of the heart to the apex were acquired using ECG-gated fat suppression triple inversion recovery sequence during breath hold in cooperative patients and with free breathing in uncooperative patients.

- Scan parameters were Repetition time (TR) > 2000 msec; echo time (TE) > 60 msec; flip angle (FA) 90 degree; inversion time (TI) 120-170 msec; slice thickness 8 mm with slice gap; matrix is 208x208, field-of-view is rotated to avoid wrap according to the size of each patient and voxel size is modified according to the field of view.

- Respiratory motion artifact can be minimized by breath-holding (preferred when

possible) or by acquiring 2–3 signal averages (= number of excitations) with the patient breathing.

- Phase contrast and flow assessment:
Aorta and sub-aorta left ventricular outflow tract (LVOT) Q-flow phase contrast images which help determination of the peak systolic velocity and pressure gradient during rest across the narrowest point in LVOT, in blood flow volume quantification for internal validation of the SV, indirect assessment of mitral and tricuspid regurgitation if present, and cardiac output of the left ventricle.

- Measurements were performed with free-breathing and multiple signal averages (2) to prevent the effect of breath holding on the amount of blood passing in and out of the heart which confounds clinical interpretation.

- Scan parameters were Repetition time (TR) 105.2 msec; echo time (TE) 2.97 msec; flip angle (FA) 20 degree; slice thickness 8 mm; matrix is 119x192, field-of-view is rotated to avoid wrap according to the size of each patient and voxel size is modified according to the field of view.

- Images are acquired with retrospective ECG-gating, the reconstructed phases are 30 phases per R-R interval, velocity encoding starts from starts from 150 cm/sec in aorta.

- Injection of gadolinium-based contrast agent (Magnevist, Bayer AG, Germany, 0.5 mmol/kg).

- Delayed gadolinium enhancement (DGE):
In our cases we used the available late gadolinium sequences in our centre; the free breathing single shot LGE and breath hold or free breathing with high average phase sensitive inversion recovery (PSIR) to calculate volume of replacement fibrosis.

- 5-10 minutes after injection of a gadolinium-based contrast agent DGE images were acquired in the same orientation as the cine images using scan parameters: Repetition time (TR) 906.4 msec; echo time (TE) 1.3 msec; flip angle (FA) 40 degree; slice thickness 8 mm; matrix is 122x192, images were acquired in diastole and triggered every 3-4 heartbeat, field-of-view is rotated to avoid wrap according the size of each patient and voxel size is modified according to the field of view.

Post processing:
Was done using segmentational software.
We calculated:
1) The functional parameters of each ventricle; end diastolic volume (EDV), end systolic volume (ESV), stroke volume (SV), end diastolic volume indexed to body surface area (EDVI), end systolic volume indexed to body surface area (ESVI), stroke volume indexed to body surface area (SVI) and ejection fraction (EF).

The values were compared with the normal values of previous studies by (Kawel et al, 2015 & 2020) (14, 15).

2) Flow parameters for the aortic valve; peak systolic velocity, forward flow, backward flow, net flow, and regurgitation fraction.

3) Tricuspid and mitral valve regurgitation were assessed indirectly by subtraction of the forward flow of aortic or pulmonary valves from the ventricular stroke volume then the product is subdivided by the ventricular stroke volume.

MR Image Analysis
MR image analyses were performed with commercially available software (cmr42, v. 5.11.4; Circle Cardiovascular Imaging, Calgary, Canada), According to 16 AHA segmentation of a bull's-eye plot.
2-chamber and 4-chamber complementary images were used into the strain analysis, epicardial and endocardial surfaces were traced manually in end systolic and end diastolic phases, then propagated through other phases with papillary muscles exclusion only in strain assessment, they were included in volumetric and functional ventricular analysis.

Regional strain parameters of the radial, circumferential, and longitudinal directions as regard the displacement, strain, peak diastolic strain rate, peak diastolic velocity, peak systolic strain rate, peak systolic velocity, time to peak displacement and time to peak strain were automatically computed.

Cardiac function concerning the end diastolic volume absolute and indexed values (EDV and EDVI), end systolic volume absolute and indexed values (ESV and ESVI), stroke volume absolute and indexed values (SV and SVI), myocardial mass absolute and

indexed values, and ejection fraction (EF) were calculated.

Statistical Analysis
Statistical analysis was performed with ANOVA and T-Test methods. The data are presented as mean and +/- standard deviation (SD). P value less than 0.05 was considered a statistically significant difference for all comparisons.

Results

Study Population
HCM patients 21 male and 4 female; mean age 39.4-year, range 7: 65 years.
Healthy control 16 male and 9 female; mean age 33-year, range 13-56 years.
In the HCM group, there were 220 hypertrophied segments and 120 segments of an apparently normal thickness. Normal controls had 400 segments.
The regional myocardium strain analysis in HCM patients in radial, circumferential, and longitudinal directions parameters are shown in **table 1-3** compared to healthy control **table 4 & 5**.

In the HCM group regional analysis
The **radial direction** parameters in hypertrophic enhancing segments were significantly different from non-hypertrophic non-enhancing apparently normal segments as regard the displacement, strain, peak diastolic strain rate, peak diastolic velocity, peak systolic strain rate and peak systolic velocity (P<0.05). There was no significant difference as regard the time to peak displacement (P<0.065, approaching the significant cut off value) and time to peak strain.

The **longitudinal direction** parameters in hypertrophic enhancing segments were significantly different from non-hypertrophic non-enhancing apparently normal as regard the strain, peak diastolic strain rate and peak systolic strain rate (P<0.05). There was no significant difference as regard the displacement and peak systolic velocity. time to peak displacement and time to peak strain and peak diastolic velocity.

The **circumferential direction** parameters in hypertrophic enhancing segments were significantly different from non-hypertrophic non-enhancing apparently normal as regard the strain, peak diastolic strain rate and peak systolic strain rate (P<0.05). There was no significant difference as regard to the displacement and peak systolic velocity, time to peak displacement and time to peak strain and peak diastolic velocity.

In comparison, hypertrophic and normal control persons
The radial regional analysis, the hypertrophic enhancing segments were significantly different from the normal as regard the displacement, strain, peak diastolic strain rate, peak diastolic velocity, peak systolic strain rate, peak systolic velocity, time to peak displacement and time to peak strain (P <0.05). There was no significant difference as regard the time to peak strain, while the apparently normal non-enhancing segments in HCM patients present significant difference compared with normal persons as regard strain and peak systolic velocity only, despite absent significant difference as regard the time to peak strain, yet P <0.085, approaching the significant cut off value.

The longitudinal regional analysis, the hypertrophic enhancing segments were significantly different from the normal as regard the displacement, strain, peak diastolic strain rate, peak systolic strain rate, peak systolic velocity, and time to peak displacement (P <0.05), while the apparently normal non-enhancing segments in HCM patients present significant difference compared with normal persons as regard the displacement, and peak systolic velocity, despite absent significant difference as regard the time to peak strain, yet P <0.08, approaching the significant cut off value.

The regional circumferential analysis, the hypertrophic enhancing segments were significantly different from the normal as regard the strain, peak diastolic strain rate, peak systolic velocity, and time to peak displacement (P<0.05), while the apparently normal non-enhancing segments in HCM patients present significant difference compared with normal persons as regard the strain, and peak systolic velocity, despite absent significant difference as regard the time to peak strain, yet P <0.059, approaching the significant cut off value.

Discussion

In patients with hypertrophic cardiomyopathy (HCM), particularly in stage II (classic phenotype), ejection fraction (EF) often appears normal or even supranormal (pseudonormal). However, regional wall deformation and functional impairment are already present—not only in the hypertrophied segments but also in segments with apparently normal wall thickness. Radial strain parameters are more severely affected than circumferential and longitudinal strain, reflecting the predominantly concentric nature of myocardial hypertrophy, which occurs primarily in the radial direction rather than eccentric expansion.

The non-enhancing segments with apparently normal thickness also exhibit significant functional impairment, albeit to a lesser degree. However, they show no signs of compensatory function.

Previous research demonstrated that strain analysis can have long-term predictive values in identifying the HCM patients at increased risk for adverse events in follow-up heart failure (stages of adverse remodeling and overt dysfunction) (8, 12, 13).

Conclusion

In patients with hypertrophic cardiomyopathy (HCM), the hypertrophied segments are more affected than those of apparently normal thickness, particularly in the radial direction. However, the seemingly normal segments also exhibit dysfunction, with no evidence of compensatory hyperfunction.

Our data indicate that myocardial fibers in HCM predominantly exhibit hypocontractility and reduced function, rather than hypercontractility. Even segments that appear morphologically normal show measurable impairment compared to healthy myocardium.

These findings are particularly important in follow-up studies, especially in patients receiving myosin inhibitors, as well as in young individuals with preserved cardiac function and gene-positive status. In such cases, concentric thickening may be detectable on imaging despite the apparent absence of phenotypic expression. This raises the question of whether more advanced imaging techniques, such as strain analysis, are needed to confirm the absence of gene penetration.

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Study limitations

The population of study was limited.

Declarations

The authors did not receive support from any organization for the submitted work. All authors certify that they have no affiliations with or involvement in any organization or entity with any financial interest or non-financial interest in the subject matter or materials discussed in this manuscript.

Consent for publication: The author clarifies that written informed consent was obtained and the anonymity of the patient was ensured. This study submitted to Swiss J. Rad. Nucl. Med. has been conducted in accordance with the Declaration of Helsinki and according to requirements of all applicable local and international standards.

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The authors declare that there were no conflicts of interest within the meaning of the recommendations of the International Committee of Medical Journal Editors when the article was written.

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References

1) Minhas, A. M. K., Wyand, R. A., Ariss, R. W., Nazir, S., Shahzeb Khan, M., Jia, X., Greene, S. J., Fudim, M., Wang, A., Warraich, H. J., Kalra, A., Alam, M., & Virani, S. S. (2022). Demographic and Regional Trends of Hypertrophic Cardiomyopathy-Related Mortality in the United States, 1999 to 2019. Circulation: Heart Failure, 15(9), E009292. https://doi.org/10.1161/CIRCHEARTFAILURE.121.009292.

2) Semsarian C, Ingles J, Maron MS, Maron BJ. New perspectives on the prevalence of hypertrophic cardiomyopathy. J Am Coll Cardiol (2015); 65:1249–1254. https://www.jacc.org/doi/full/10.1016/j.jacc.2015.01.019

3) Baxi AJ, Restrepo CS, Vargas D, et al. Hypertrophic cardiomyopathy from A to Z: Genetics, pathophysiology, imaging, and management. Radiographics (2016);36(2):335-54. https://doi.org/10.1148/rg.2016150137

4) Amano, Y., Kitamura, M., Takano, H., Yanagisawa, F., Tachi, M., Suzuki, Y., Kumita, S., & Takayama, M. (2018). Cardiac MR imaging of hypertrophic cardiomyopathy: Techniques, findings, and clinical relevance. In Magnetic Resonance in Medical Sciences (Vol. 17, Issue 2, pp. 120–131). Japanese Society for Magnetic Resonance in Medicine. https://doi.org/10.2463/mrms.rev.2017-0145

5) Cui, H., Schaff, H. v., Lentz Carvalho, J., Nishimura, R. A., Geske, J. B., Dearani, J. A., Lahr, B. D., Lee, A. T., Bos, J. M., Ackerman, M. J., Ommen, S. R., & Maleszewski, J. J. (2021). Myocardial Histopathology in Patients With Obstructive Hypertrophic Cardiomyopathy. Journal of the American College of Cardiology, 77(17), 2159–2170. https://doi.org/10.1016/j.jacc.2021.03.008

6) Almaas, V., and Amlie, P. Histopathological Changes and Clinical Implications in Patients with Hypertrophic Cardiomyopathy, European Cardiology (2010);6(2):88–90 https://doi.org/10.15420/ecr.2010.6.2.88

7) James A. Spudich J. Three perspectives on the molecular basis of hypercontractility caused by hypertrophic cardiomyopathy mutations. European Journal of Physiology (2019) 471:701–717. https://doi.org/10.1007/s00424-019-02259-2

8) Olivotto, I., Cecchi, F., Poggesi, C., & Yacoub, M. H. (2012). Patterns of disease progression in hypertrophic cardiomyopathy an individualized approach to clinical staging. Circulation: Heart Failure, 5(4), 535–546. https://doi.org/10.1161/CIRCHEARTFAILURE.112.967026

9) Marian, A. J., & Braunwald, E. (2017). Hypertrophic cardiomyopathy: Genetics, pathogenesis, clinical manifestations, diagnosis, and therapy. Circulation Research, 121(7), 749–770. https://doi.org/10.1161/CIRCRESAHA.117.311059

10) Ommen SR, Mital S, Burke MA, Day SM, Deswal A, Elliott P, et al. (2020) AHA/ACC Guideline for the Diagnosis and Treatment of Patients With Hypertrophic Cardiomyopathy: A Report of the American College of Cardiology/American Heart Association Joint Committee on Clinical Practice Guidelines. J Am Coll Cardiol 2020;162:e23-106. https://doi.org/10.1161/CIR.0000000000000937

11) Lopez L, Colan S, Stylianou M, Granger S, Trachtenberg F, Frommelt P, et al. Relationship of Echocardiographic Z Scores Adjusted for Body Surface Area to Age, Sex, Race, and Ethnicity: The Pediatric Heart Network Normal Echocardiogram Database. Circ Cardiovasc Imaging (2017);10: e006979. https://doi.org/10.1161/CIRCIMAGING.117.006979

12) Reant P, Reynaud A, Pillois X, et al. Comparison of resting and exercise echocardiographic as indicators of outcomes in hypertrophic. J Am Soc Echocardiogr (2015);28:194–203. https://doi.org/10.1016/j.echo.2014.10.001

13) Reant P, Mirabel M, Lloyd G, et al. Global longitudinal strain is associated with heart failure outcomes in hypertrophic cardiomyopathy. Heart (2016);102:741–747. https://doi.org/10.1136/heartjnl-2015-308576

14) Kawel-Boehm, N., Maceira, A., Valsangiacomo-Buechel, E. R., Vogel-Claussen, J., Turkbey, E. B., Williams, R., Plein, S., Tee, M., Eng, J., & Bluemke, D. A. (2015). Normal values for cardiovascular magnetic resonance in adults and children. In Journal of Cardiovascular Magnetic Resonance (Vol. 17, Issue 1). BioMed Central Ltd. https://doi.org/10.1186/s12968-015-0111-7

15) Kawel-Boehm, N., Hetzel, S. J., Ambale-Venkatesh, B., Captur, G., Francois, C. J., Jerosch-Herold, M., Salerno, M., Teague, S. D., Valsangiacomo-Buechel, E., van der Geest, R. J., & Bluemke, D. A. (2020). Reference ranges ("normal values") for cardiovascular magnetic resonance (CMR) in adults and children: 2020 update. In Journal of Cardiovascular Magnetic Resonance (Vol. 22, Issue 1). BioMed Central Ltd. https://doi.org/10.1186/s12968-020-00683-3

Tables:

Table 1: Radial strain analysis in HCM patients				
		N	Mean	Standard deviation (SD)
displacement(deg)	HCM	220	3.7500	2.06423
	Apparently normal	180	5.3017	2.45007
	Total	400	4.4483	2.37259
strain (%)	HCM	220	20.0977	13.09198
	Apparently normal	180	27.7128	16.94304
	Total	400	23.5245	15.40289
peak diastolic strain Rate(1/s)	HCM	220	-1.2455	1.05589
	Apparently normal	180	-1.6506	1.29574
	Total	400	-1.4278	1.18569
Peak diastolic Velocity(deg/s)	HCM	220	-22.5109	13.05833
	Apparently normal	180	-25.9861	14.34507
	Total	400	-24.0748	13.74438
Peak systolic strain rate(1/s)	HCM	220	1.4527	1.37214
	Apparently normal	180	2.0639	1.35691
	Total	400	1.7278	1.39717
peak systolic velocity(deg/s)	HCM	220	26.8573	16.86500
	Apparently normal	180	33.6544	16.54626
	Total	400	29.9160	17.04116
time to peak displacement(ms)	HCM	220	354.6468	106.16875
	Apparently normal	180	336.5578	85.54762
	Total	400	346.5068	97.73002
Time to peak strain(ms)	HCM	220	339.2036	111.04739
	Apparently normal	180	344.1128	93.94762
	Total	400	341.4128	103.60508

In the HCM group regional analysis

The **radial direction** parameters in hypertrophic segments were significantly different from non-hypertrophic apparently normal segments as regard the displacement, strain, peak diastolic strain rate, peak diastolic velocity, peak systolic strain rate and peak systolic velocity ($P<0.05$). There was no significant difference as regard the time to peak displacement ($P<0.065$, approaching the significant cut off value) and time to peak strain

Table 2: Circumferential strain analysis in HCM patients				
		N	Mean	Standard deviation (SD)
displacement(deg)	HCM	220	1.4095	3.79885
	Apparently normal	180	1.2656	4.54223
	Total	400	1.3448	4.14511
strain (%)	HCM	220	-11.3777	5.97949
	Apparently normal	180	-14.9606	7.67467
	Total	400	-12.9900	7.01667
peak diastolic strain Rate(1/s)	HCM	220	0.6950	0.58686
	Apparently normal	180	0.9717	0.75667
	Total	400	0.8195	0.68182
Peak diastolic Velocity(deg/s)	HCM	220	-8.1750	30.74744
	Apparently normal	180	-4.2944	35.11116
	Total	400	-6.4288	32.79787
Peak systolic strain rate(1/s)	HCM	220	-0.9041	0.72549
	Apparently normal	180	-1.2789	0.73970
	Total	400	-1.0728	0.75446
peak systolic velocity(deg/s)	HCM	220	7.9386	38.07865
	Apparently normal	180	3.6900	43.46649
	Total	400	6.0268	40.59472
time to peak displacement(ms)	HCM	220	339.4059	167.06305
	Apparently normal	180	323.9167	152.94768
	Total	400	332.4358	160.85144
Time to peak strain(ms)	HCM	220	337.5836	121.95465
	Apparently normal	180	347.5556	99.16645
	Total	400	342.0710	112.24861

The **circumferential direction** parameters in hypertrophic segments were significantly different from non-hypertrophic apparently normal as regard the strain, peak diastolic strain rate and peak systolic strain rate (P<0.05). There was no significant difference as regard the displacement and peak systolic velocity. time to peak displacement and time to peak strain and peak diastolic velocity.

Table 3: Longitudinal strain analysis in HCM patients				
		N	Mean	Standard deviation (SD)
displacement(deg)	HCM	216	2.8569	2.41196
	Apparently normal	176	2.9801	3.04526
	Total	392	2.9122	2.71169
strain (%)	HCM	216	-5.9542	11.59742
	Apparently normal	176	-12.2125	11.14986
	Total	392	-8.7640	11.80314
peak diastolic strain Rate(1/s)	HCM	216	0.4995	1.25125
	Apparently normal	176	0.7847	1.27308
	Total	392	0.6276	1.26746
peak diastolic Velocity(deg/s)	HCM	216	-17.8653	21.10452
	Apparently normal	176	-20.5591	23.45234
	Total	392	-19.0747	22.20095
peak systolic strain rate(1/s)	HCM	216	-0.7662	1.64343
	Apparently normal	176	-1.0784	1.28969
	Total	392	-0.9064	1.50125
peak systolic velocity(deg/s)	HCM	216	20.4245	24.51892
	Apparently normal	176	21.0784	27.04525
	Total	392	20.7181	25.65249
time to peak displacement(ms)	HCM	216	369.5435	144.80205
	Apparently normal	176	359.6636	139.15729
	Total	392	365.1077	142.19992
time to peak strain(ms)	HCM	216	354.1847	163.71557
	Apparently normal	176	362.4784	155.10324
	Total	392	357.9084	159.75723

The **longitudinal direction** parameters in hypertrophic segments were significantly different from non-hypertrophic apparently normal as regard the strain, peak diastolic strain rate and peak systolic strain rate (P<0.05). There was no significant difference as regard the displacement and peak systolic velocity. time to peak displacement and time to peak strain and peak diastolic velocity.

Table 4 strain analysis in healthy control

Normal mean	Displacement (deg)	Strain (%)	Peak diastolic strain rate (1/s)	Peak diastolic velocity(deg/s)	Peak systolic strain rate (deg/s)	Peak systolic velocity (deg/s)	Time to peak displacement (m/s)	Time to peak strain (m/s)
Circumferential strain	1.42	-17.22	0.88	-6.73	-1.12	16.81	294.97	333.49
Radial strain	5.41	30.56	-1.62	-27.25	1.91	30.74	338.22	331.93
Longitudinal strain	3.64	-13.59	0.76	-20.07	-.0.99	25.53	316.59	341.89

Table 5. Performance analysis of deep learning CNN models for classifying HKM patients from normal using myocardial strain measured at the 3 individual dimensions (radial, longitudinal and circumferential) in addition to all dataset after data reduction using principal component analysis (PCA). A wide range of PCs was tested to investigate the classification performance of the 3 different directions, namely at 1, 2, 3, 5, 10, 20 and 30 PCs. The variance explained by PCs as well as performance metrics including sensitivity, specificity, accuracy, and ROC of each PC(s) for every direction of myocardial strain is demonstrated.

Dimension	PCs	% Variance Explained	Sensitivity	Specificity	Accuracy	ROC
Radial	1	20.4	0.429	0.625	0.533	0.696
	2	33.1	0.429	0.875	0.667	0.812
	3	29.5	0.714	1.0	0.867	1.0
	5	53.9	0.714	1.0	0.867	0.911
	10	72.9	0.714	1.0	0.867	**0.929**
	15	**84.1**	**0.857**	**1.0**	**0.933**	0.893
	20	91.1	0.857	0.875	0.867	0.875
	30	98.6	0.857	0.750	0.800	0.875
Circumferential	1	12.1	0.375	1	0.667	0.643
	2	21.9	0.375	1.0	0.667	0.911
	3	**29.5**	**0.625**	**1.0**	**0.800**	**0.946**
	5	43.2	0.625	0.857	0.733	0.839
	10	66.2	0.625	0.857	0.733	0.786
	15	79.9	0.625	0.857	0.733	0.732

	20	88.9	0.625	0.857	0.733	0.750
	30	98.7	0.625	0.857	0.733	0.714
Longitudinal	**1**	**37.6**	**1**	**1**	**1**	**1**
	2	**45.2**				
	3	51.4				
	5	60.6				
	10	76.1				
	15	85.8				
	20	92.6				
	30	99.4				
All directions	1	34.1	0.381	0.619	0.500	0.537
	2	43.7	0.476	0.667	0.571	0.615
	3	48.3	0.476	0.762	0.619	0.633
	5	55.4	0.476	0.810	0.643	0.633
	10	66.1	0.667	0.762	0.714	**0.864**
	15	73.5	**0.714**	0.810	**0.762**	0.846
	20	79.3	0.571	0.905	0.738	0.832
	30	87.7	0.571	**0.952**	**0.762**	0.839

Figure 1:

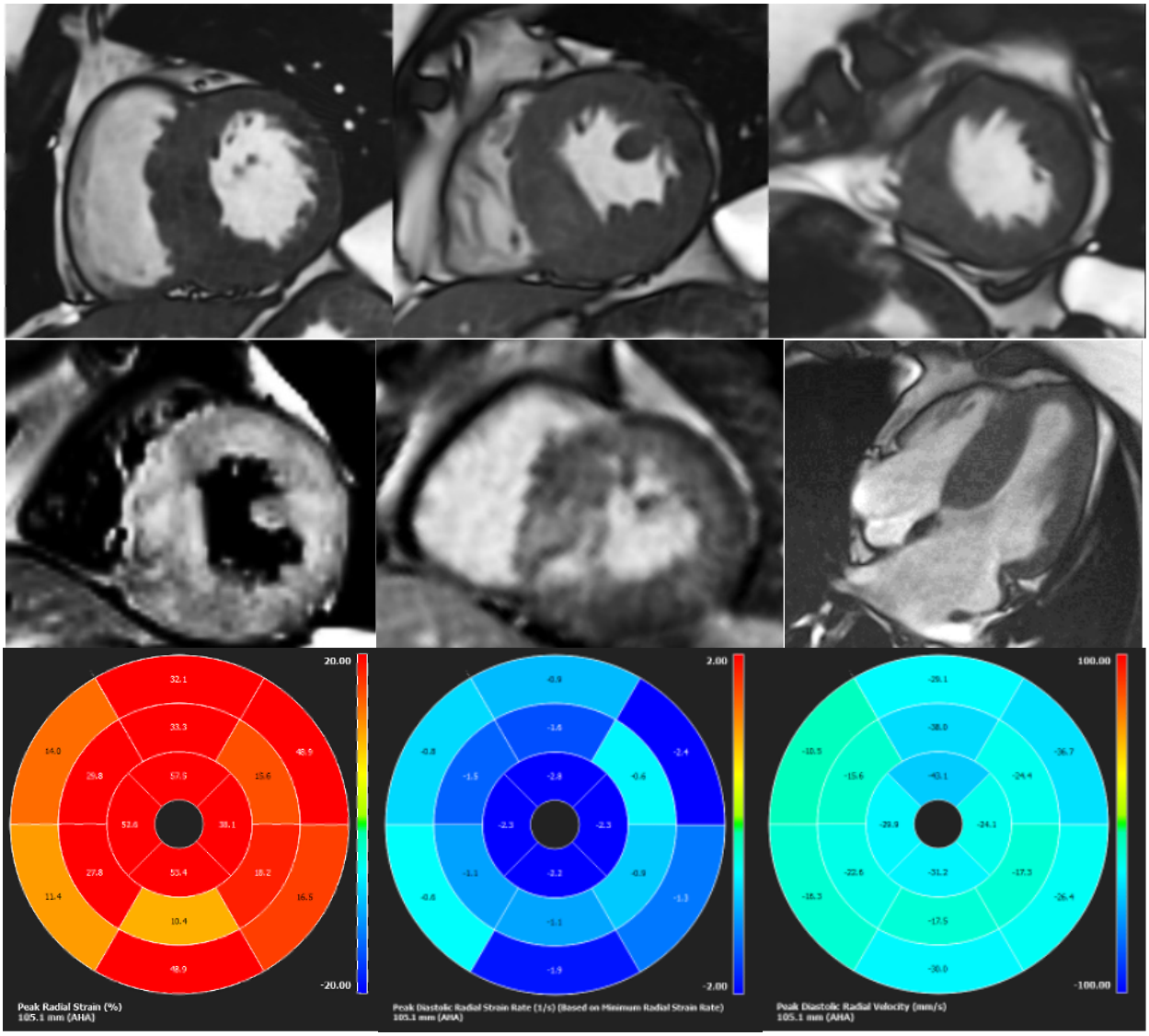

Figure 1:
Illustrative case, 44-year-old male patient presents with asymmetrical hypertrophied left ventricular myocardium involving septal segments, acute myocardial break down as regard the patchy focal increased signal in T2 fat suppression WI with corresponding delayed enhancement.

Significantly affected regional radial strain parameters in basal septal segments as regard the displacement, strain, peak diastolic strain rate, peak diastolic velocity, peak systolic strain rate and peak systolic velocity.

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