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Left ventricular septal convexity in differentiating hypertrophic cardiomyopathy from hypertensive heart disease – a cardiac magnetic resonance study

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Both hypertrophic cardiomyopathy (HCM) and hypertensive heart disease (HHD) are characterized by left ventricular hypertrophy. Distinguishing HCM from HHD is a common clinical problem, particularly in subjects with moderate left ventricular maximal wall thickness (LVMWT) < 18 mm. Previously, we showed that cardiac magnetic resonance (CMR)-derived septal convexity (SC) into the left ventricle is increased in subjects with HCM-causing mutations with and without LVH. Our objective now was to determine whether CMR-derived SC differentiates HCM from HHD. We measured the maximal distance between the LV septal endocardial border and a line connecting the proximal and distal septal mid-wall points in 4-chamber images, in subjects with hypertension and LVH (*n* = 29), in subjects with HCM (*n* = 49), and in healthy controls (*n* = 20). Here, we show significantly increased SC in subjects with HCM compared to subjects with HHD both in non-indexed and in BSA-indexed measurements. Cutoff SC values of 7.9 mm and 3.7 mm/m2 in all HCM patients and SC values of 7.9 mm and 3.8 mm/m2 in HCM patients with LVMWT < 18 mm differentiated between HCM and HHD with good sensitivity and excellent specificity: SC cutoff value of 7.9 mm had a sensitivity of 77% and a specificity of 90% in all HCM patients, and 67% and 90% in HCM patients with LVMWT < 18 mm, respectively. Our study shows that measuring CMR-derived septal convexity is straightforward and enhances diagnostic performance, providing a novel technique to distinguish between HCM caused by sarcomere mutations and hypertension-induced LVH.

Keywords Cardiac magnetic resonance, Septal convexity, Left ventricular hypertrophy, Hypertrophic cardiomyopathy, Hypertensive heart disease, Differential diagnosis

It is estimated that 26% of the adult population worldwide is hypertensive[1]. Hypertensive heart disease (HHD), characterized by left ventricular hypertrophy (LVH), is a common complication of high blood pressure[2,3] and is the most common cause of LVH[4]. HHD increases the risk of sudden cardiac death[5] and heart failure[6]. Hypertrophic cardiomyopathy (HCM), also characterized by LVH, is the most common genetic cardiac disease caused mainly by mutations in sarcomere genes[7,8] and it is considered the leading cause of sudden cardiac death among young people and athletes[9].

Differentiating HCM from HHD is a common clinical problem, particularly in patients with moderate LVH. Asymmetric LVH, previously considered to define HCM, is also prevalent in patients with HHD[2,3]. In

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a cardiac magnetic resonance (CMR) study, asymmetric LVH in the basal or mid-LV septum was present in 21% of hypertensive subjects[3]. There are limited data on the use of 2-D echocardiography, tissue Doppler and strain imaging in differentiating HCM from HHD[10]. CMR, the gold standard for investigating the anatomy of the left ventricle[11,12] and characterizing myocardial tissue[13] is currently available for discriminating between HCM and HHD. In our previous CMR study, the maximal wall thickness (LVMWT) ≥ 17 mm was the best anatomical parameter for differentiating HCM from HHD[2]. Increased LV mass, the absence of late gadolinium enhancement (LGE), and the absence of systolic anterior motion (SAM) of the mitral valve have been found to discriminate HHD from HCM[14]. There is, however, first, considerable overlap in the LV mass between patients with HHD and those with HCM. Second, LGE is not present in all patients with HCM[15] and may also be found in HHD[16]. Third, SAM of the mitral valve is present only in a minority of subjects with HCM[15]. In a recent study, CMR-derived T1 mapping values performed best in the differential diagnosis of HCM and HHD; however, there was considerable overlap between the two groups[17]. Thus, discrimination between HCM and HHD remains challenging, particularly in subjects with mild to moderate asymmetric LVH.

Recently, two studies have shown that CMR-derived septal convexity (SC) into the LV is increased in subjects with HCM-causing sarcomere gene mutations with and without LVH[18,19]. To our knowledge, there have been no previous studies on SC in HHD. Therefore, we investigated whether CMR-derived SC is a useful measure for distinguishing between HCM and HHD.

Methods

We have previously published several articles[2,8,19–22] on the genetics of HCM in Finland and the phenotypic expression of hypertrophic cardiomyopathy in CMR in subjects with the major Finnish HCM-causing mutations *MYBPC3*-Q1061X ($n = 32$) and *TPM1*-D175N ($n = 17$). We also investigated the CMR characteristics of HHD patients and compared them with those of HCM patients caused by TPM1-D175N[2]. However, we have not previously compared septal convexity between patients with HCM and those with HHD.

Figure 1. Flowchart of the study. Some subjects with hypertrophic cardiomyopathy (HCM) were excluded because they had a pacemaker or implantable cardioverter defibrillator, and not all eligible subjects who were invited wanted to participate in the cardiac magnetic resonance studies.

Subjects with hypertension

Altogether, 169 outpatient subjects with hypertension were recruited as previously described[2]. The inclusion criteria were (1) age under 55 years, (2) self-reported hypertension, (3) no antihypertensive treatment, and (4) no other detected cardiac disease on CMR imaging or echocardiography. The participants received written instructions and individual guidance to measure blood pressure (BP) at home during three sequential days in three sessions (morning, afternoon and evening) with a clinically validated semiautomatic sphygmomanometer

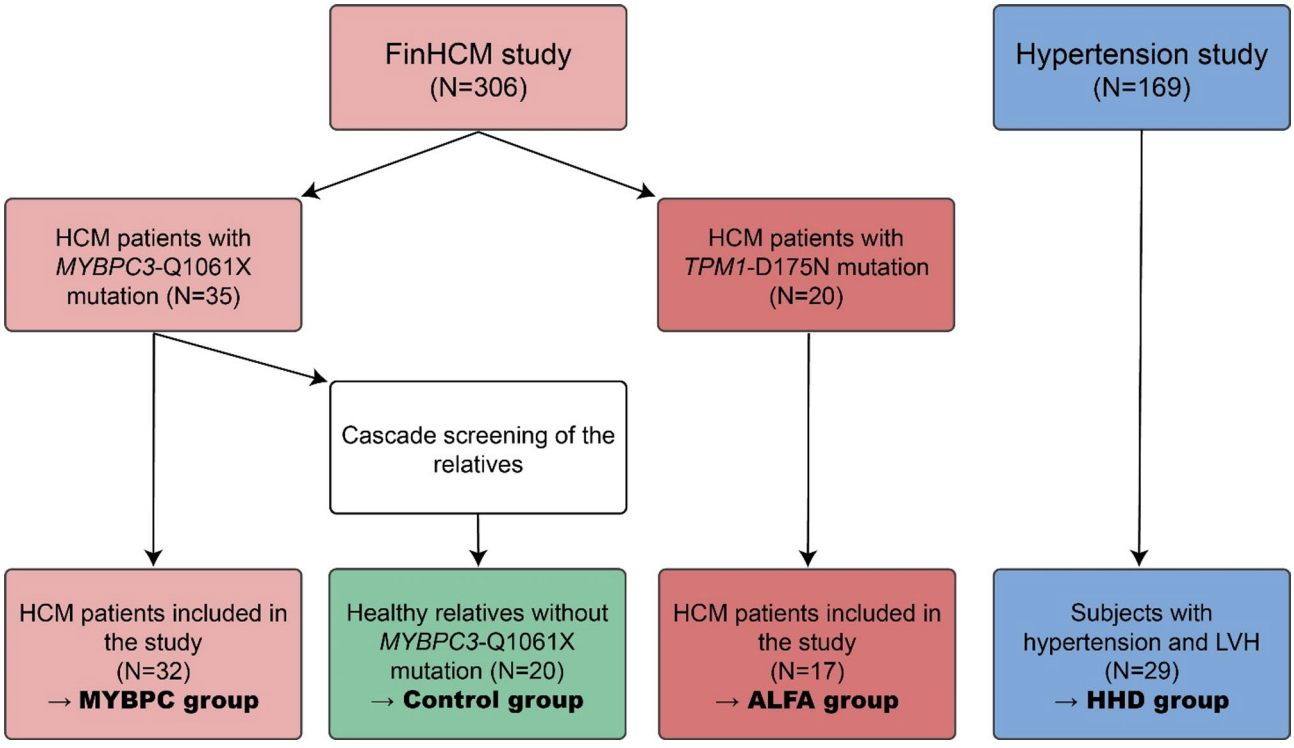

Fig.1. shows the flowchart of the study subjects. Some subjects with HCM were excluded because they had a pacemaker or implantable cardioverter defibrillator (ICD), and not all eligible subjects who were invited wanted to participate in the CMR studies.

Omron HEM-737 Intellisense (Omron Healthcare GmbH, Hamburg, Germany). BP measurements were taken with an appropriately sized cuff placed at the heart level from the right arm after the patient sat at rest for 5 min, with ≥2 min between each measurement. All measurements were performed in triplicate and recorded in the logbook. We used the average of the measurements recorded on the second and third monitoring days. Among the 169 enrolled subjects, all 95 hypertensive subjects (average BP ≥ 140/90 mmHg) were referred to CMR.

The LVMWT was measured in end-diastolic cine MR images in the short-axis orientation by two experienced analysers (P.S. and M.H.). Among the hypertensive subjects, 29 had LVMWT ≥ 13 mm on CMR and were included in the present study (HHD group). The hypertensive subjects were not tested for HCM-causing mutations, but as the prevalence of HCM is 1:500[15], it is very improbable that hypertensive subjects from the general population had HCM (probability for HCM 0.058 for one subject of the study, 0.002[29] for all subjects having HCM).

Subjects with HCM

In our FinHCM Study, we investigated the genetics of HCM in 306 Finnish European subjects with HCM[8,23,24]. In subsequent ALFA and MYBPC studies, altogether 49 subjects with mutation-confirmed HCM were recruited, including 17 subjects with HCM (LVMWT ≥ 13 mm in CMR) attributable to the D175N mutation of the α-tropomyosin gene (*TPM1*-D175N; the ALFA group)[2,20] and 32 subjects with HCM attributable to the Q1061X mutation of the cardiac myosin-binding protein C gene *(MYBPC3*-Q1061X; the MYBPC group)[19,21,22]. The founder mutation *MYBPC3*-Q1061X is the most common HCM-causing mutation in Finland, accounting for 11.3% of HCM cases in the FinHCM Study of 382 unrelated patients[8]. *TPM1*-D175N is the third most common HCM-causing mutation in Finland, accounting for 6.3% of cases in the FinHCM Study[8]. Significant left ventricular outflow tract obstruction (gradient > 30 mmHg) was present only in one HCM patient with the *MYBPC3* mutation.

Control subjects

Healthy relatives ($n = 20$) without an HCM-causing mutation in the family and LVMWT < 13 mm on CMR were recruited from families with *MYBPC3*-Q1061X as control subjects.

The study conforms to the principles outlined in the Declaration of Helsinki. The ethics committees at Kuopio and Helsinki University Hospitals approved the study protocol. All the participants provided written informed consent.

CMR

All of the subjects underwent a CMR study using a 1.5-T clinical MR imaging unit. Hypertensive subjects and subjects with the *TPMI*-D175N mutation were scanned at Kuopio University Hospital, and subjects with the *MYBPC3*-Q1061X mutation and control subjects were scanned at Kuopio and Helsinki University Hospitals. The study protocols have been explained in detail previously[2,19,20].

Image analyses and SC measurements

Image analysis was performed by experienced analysts (M.T., K.L., P.S., and M.H. with more than 10 years of experience in CMR) blinded to the clinical or genetic findings of the study subjects. SC into the LV was measured in end-diastolic 4-chamber images as the maximal distance between the LV septal endocardial border and a line connecting septal mid-wall points at the level of tricuspid valve insertion and at the level of apical right ventricular insertion on the LV, as described previously[18]. Figure 2 shows the SC measurements in a subject with HCM and in a subject with HHD. Previously, SC measurements have been used in only two reported studies, including our own group, and have demonstrated high reproducibility[18,19]. All other anatomical measurements of the CMR images were performed as described previously[2,21]. All the HCM patients had septal asymmetric hypertrophy and other types of HCM such as apical or concentric hypertrophy were not detected.

Statistical analyses

Baseline data are presented as the mean ± standard deviation (SD.). Many of the variables had skewed distributions and were compared using Kruskal-Wallis one-way analysis of variance and the independent samples Mann–Whitney U test. Pearson's correlation coefficients were calculated to test the correlation between SC and other variables. Receiver operating characteristic (ROC) curves were constructed to test the ability of SC to differentiate between HCM and HHD. Statistical analyses and graphing were performed with IBM SPSS Statistics V14.0 and V22.0. The statistical significance level was set at $P < 0.05$.

Results

Clinical and CMR characteristics

The clinical and CMR characteristics of the study subjects are shown in Table 1. Comparisons were made over all four groups. In the control group, there were fewer men, and they had lower body surface area (BSA) compared to the other groups. Blood pressure (BP) was higher in the HHD group compared to the other groups, which were normotensive. As expected, the LVMWT and indexed left ventricular mass (LVMI) were lower in the control group than in the other groups. The LVMWT was significantly higher in patients with HCM (ALFA and MYBPC groups combined) compared to the HHD group, but there was no significant difference in the LVMI between these groups. However, the LVMI was significantly lower in the MYBPC group than in the HHD group ($P < 0.001$), but there was no significant difference between the ALFA and HHD groups ($P = 0.162$). The BSA-indexed left ventricular end-diastolic volume (LVEDVI) was lower in the HHD and ALFA groups than in the other two groups. There were no significant differences in age, BSA, BSA-indexed left ventricular end-systolic volume (LVESVI) or left ventricular ejection fraction (LVEF) between the four groups.

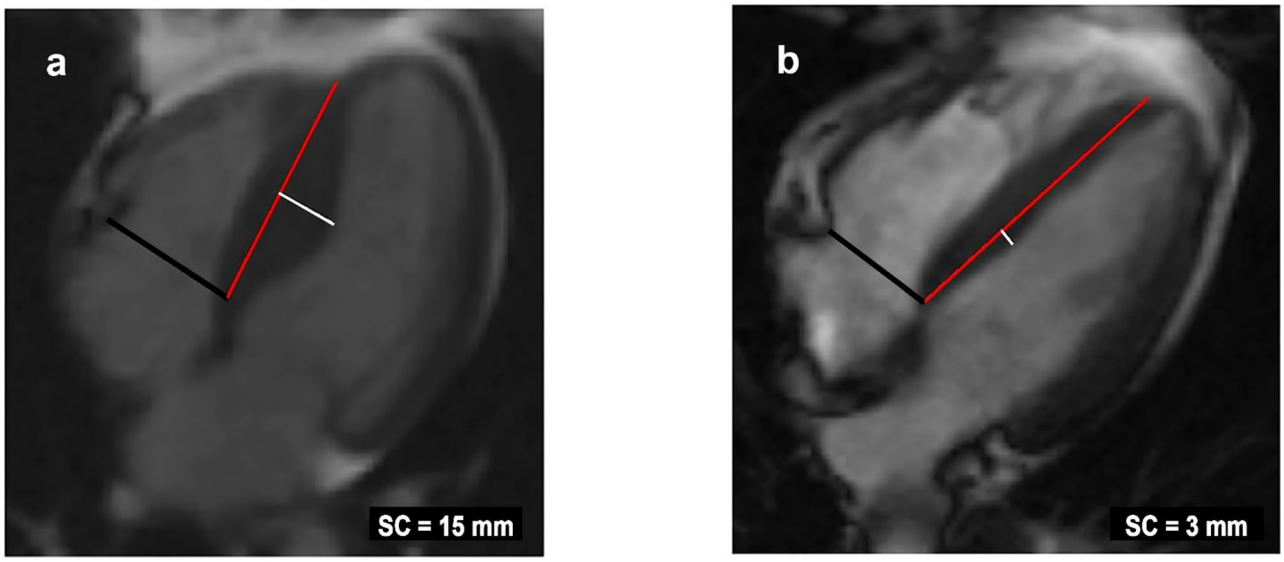

Fig. 2. Measurement of septal convexity (SC) in end-diastolic 4-chamber cardiac magnetic resonance images (**a**) from a subject with hypertrophic cardiomyopathy and (**b**) from a subject with hypertensive heart disease. The black line is the tricuspid valve annulus. The red line is a reference line between the septal mid-wall at the level of the tricuspid valve and the insertion point of the right ventricle into the left ventricle at the apex. The white line represents the measured SC.

Septal convexity

The differences between non-indexed SC values of control (2.7 ± 3.2 mm), HHD (4.9 ± 2.7 mm), ALFA (9.3 ± 3.1 mm) and MYPBC (11.4 ± 4.3) groups were statistically significant ($P < 0.001$) (Fig. 3; Table 1). Additionally, the BSA-indexed SC was significantly higher in the ALFA and MYBPC groups (4.6 ± 1.7 and 5.9 ± 1.7 mm/m2, respectively) compared to the HHD group (2.4 ± 1.3 mm/m2) ($P < 0.001$). Figure 4 represents the statistical difference between SC and BSA-indexed SC in combined HCM (ALFA and MYBPC) group and HHD group (10.7 ± 4.0 vs. 4.9 ± 2.7 mm, $P < 0.001$; 5.5 ± 2.1 vs. 2.4 ± 1.3 mm/m2, $P < 0.001$). There were no significant differences in the SC or BSA-indexed SC between the ALFA and MYBPC groups ($P = 0.108$ and $P = 0.087$, respectively).

In the HHD group, SC correlated with young age ($r = -0.435$, $P = 0.018$) and low LVMI ($r = -0.504$, $P = 0.005$) but not with the LVMWT or other CMR parameters, BSA, or average systolic or diastolic blood pressure measured at home. In the combined HCM group, SC correlated with age ($r = 0.274$, $P = 0.045$), LVMI ($r = 0.289$, $P = 0.034$) and LVMWT ($r = 0.525$, $P < 0.001$).

Figure 5 shows the receiver operating characteristic (ROC) curves of SC as a predictor of HCM. Subjects from the HHD and combined HCM groups, but not controls, were included in the analysis. The area under the curve (AUC) was 0.89. A cutoff value of 7.9 mm performed best, with a sensitivity of 77% and specificity of 90%. A cutoff value of 5.1 mm yielded 96% sensitivity and 55% specificity. When indexed for BSA, the AUC was 0.91, and a cutoff value of 3.7 mm/m2 performed best, with a sensitivity of 81% and specificity of 86% (a cutoff value of 2.6 mm/m2 with a sensitivity of 98% and specificity of 59%).

As we have previously shown[2] LVMWT ≥ 17 mm is suggestive of HCM and not HHD. None of the patients in the present study with HHD had LVMWT > 17 mm. Therefore, we also tested SC as a predictor of HCM when only HCM patients with LVMWT < 18 mm were included in the analyses ($n = 18$, Fig. 6). In this HCM subgroup, the SC was 8.8 ± 2.6 mm (range 5.0–12.9 mm), and the BSA-indexed SC was 4.5 ± 1.3 (range 2.3–6.5 mm). The SC cutoff value of 7.9 mm yielded a sensitivity of 67% and a specificity of 90% (6.4 mm yielded 83% sensitivity and 65% specificity). The area under the curve was 0.85. With BSA-indexed SC values, a cutoff of 3.8 mm/m2 performed best, with a sensitivity of 72% and specificity of 90% (3.2 mm/m2 with a sensitivity of 83% and specificity of 69%). The area under the curve was 0.87.

Discussion

In the present CMR study, we found significantly higher values of SC in the left ventricle in subjects with HCM caused by well-characterized pathogenic sarcomere gene variants compared to subjects with HHD. In addition, HHD patients had higher SC values than controls did. To predict HCM, the SC cutoff value of 7.9 mm performed best, with a sensitivity of 77% and specificity of 90%. In HCM patients with LVMWT < 18 mm, an SC cutoff value of 7.9 mm differentiated HCM from HHD, with a sensitivity of 67% and specificity of 90%.

Diagnosing HCM is not always difficult. Marked asymmetric hypertrophy with CMR-derived LVMWT ≥ 17 mm, LV outflow tract obstruction, SAM of the mitral valve, and septal LGE in a patient with normal blood pressure, a family history of HCM and an HCM-causing mutation in genetic testing poses no diagnostic challenge. However, pathogenic mutations in sarcomere protein genes are found in only up to 60% of

	Control	HHD	ALFA	MYBPC	*P*
Patients, (n)	20	29	17	32	
Age, (y)	46 ± 17	47 ± 5	40 ± 13	49 ± 11	0.823
Men, (n)	5 (25%)◆	25 (86%)	9 (53%)†	22 (69%)	< 0.001*
BSA, (m^2)	1.87 ± 0.28◆	2.10 ± 0.24	2.00 ± 0.27	1.95 ± 0.20◆	0.001*
BMI, (kg/m^2)	27 ± 8	28 ± 4	29 ± 7	27 ± 4	0.200
Systolic BP, (mmHg)	130 ± 12◆	152 ± 13	136 ± 17†	128 ± 13◆	< 0.001*
Diastolic BP, (mmHg)	76 ± 9◆	94 ± 9	84 ± 12	77 ± 8◆	< 0.001*
LVMWT, (mm)	9.6 ± 1.7◆ (7–13)	14.2 ± 1.4 (13–17)	16.3 ± 2.4◆ (13–20)	22.1 ± 5.7◆ (13–32)	< 0.001*
LVMI, (g/m^2)	45 ± 9◆	87 ± 19	81 ± 26	68 ± 21◆	< 0.001*
LVEDVI, (ml/m^2)	78 ± 13◆	66 ± 22	61 ± 22	74 ± 14†	< 0.001*
LVESVI, (ml/m^2)	32 ± 10	27 ± 13	24 ± 11	28 ± 10	0.064
LVEF, (%)	60 ± 8	60 ± 8	58 ± 8	63 ± 9	0.181
SC, (mm)	2.7 ± 3.2† (−6.0–8.9)	4.9 ± 2.7 (0.0–9.8)	9.3 ± 3.1◆Ω (5.0–16.8)	11.4 ± 4.3◆Ω (4.8–20.7)	< 0.001*
SC index, (mm/m^2)	1.5 ± 1.6 (−2.1–4.9)	2.4 ± 1.3 (0.0–4.9)	4.6 ± 1.7◆ (2.3–8.2)	5.9 ± 1.7◆ (2.7–10.4)	< 0.001*

Table 1. Clinical and cardiac magnetic resonance findings in the study groups. *Control* healthy controls without arterial hypertension or hypertrophic cardiomyopathy (HCM)-causing gene mutation, *HHD* subjects with hypertensive heart disease, *ALFA* subjects with HCM attributable to the D175N mutation of the alfa-tropomyosin gene *(TPM1),MYBPC* subjects with HCM attributable to the Q1061X mutation of the cardiac myosin binding protein C gene *(MYBPC3), BSA* body surface area, *BMI* body mass index, *BP* blood pressure, *LVMWT* left ventricular maximal wall thickness, *LVMI* left ventricular mass index, *LVEDV* left ventricular end-diastolic volume index, *LVESVI* left ventricular end-systolic volume index, *LVEF* left ventricular ejection fraction, *SC* septal convexity into the left ventricle, *SC* index; BSA-indexed septal convexity into the left ventricle. The values are presented as the means ± SDs. * Significance between all four study groups (Kruskal–Wallis one-way analysis of variance). † Significance $P < 0.05$, compared to the HHD group (independent-samples Mann–Whitney U test). ◆ Significance $P < 0.01$, compared to the HHD group (independent-samples Mann–Whitney U test). Ω Significance $P = 0.108$ between the ALFA and MYBPC groups (independent-samples Mann–Whitney U test).

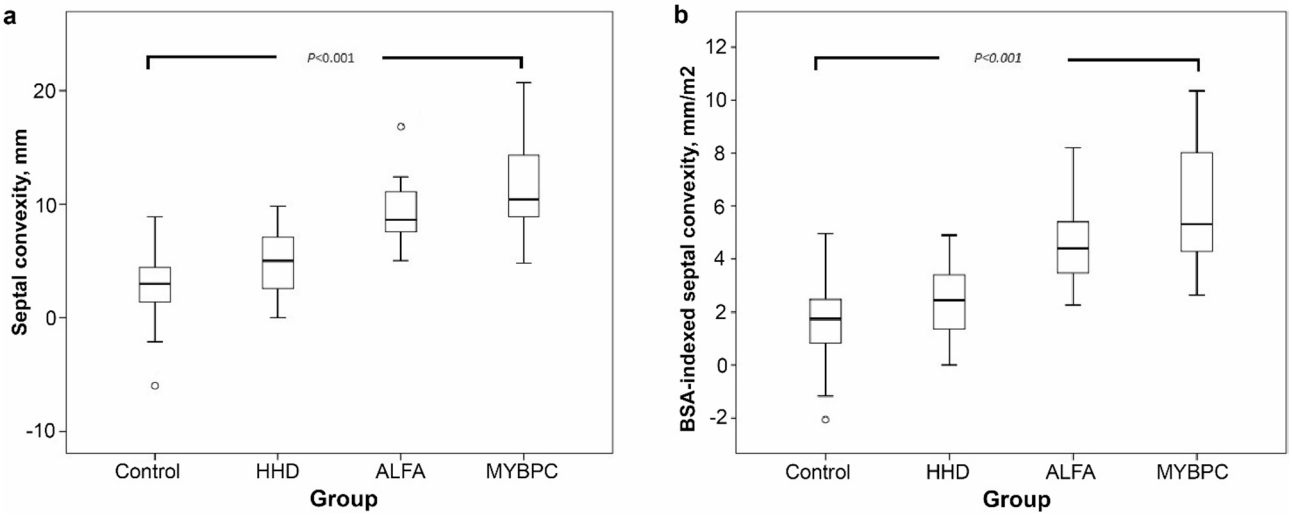

Fig. 3. Box plot showing septal convexity in the four study groups. The upper edge of the box represents the 75th percentile, and the lower edge represents the 25th percentile. The line across the box indicates the median. The small circles represent outliers. Compared with the hypertensive heart disease (HHD) and control groups, the hypertrophic cardiomyopathy (HCM) groups (ALFA and MYBPC) presented increased values in both the (**a)** non-indexed and (**b**) body surface area (BSA)-indexed analyses.

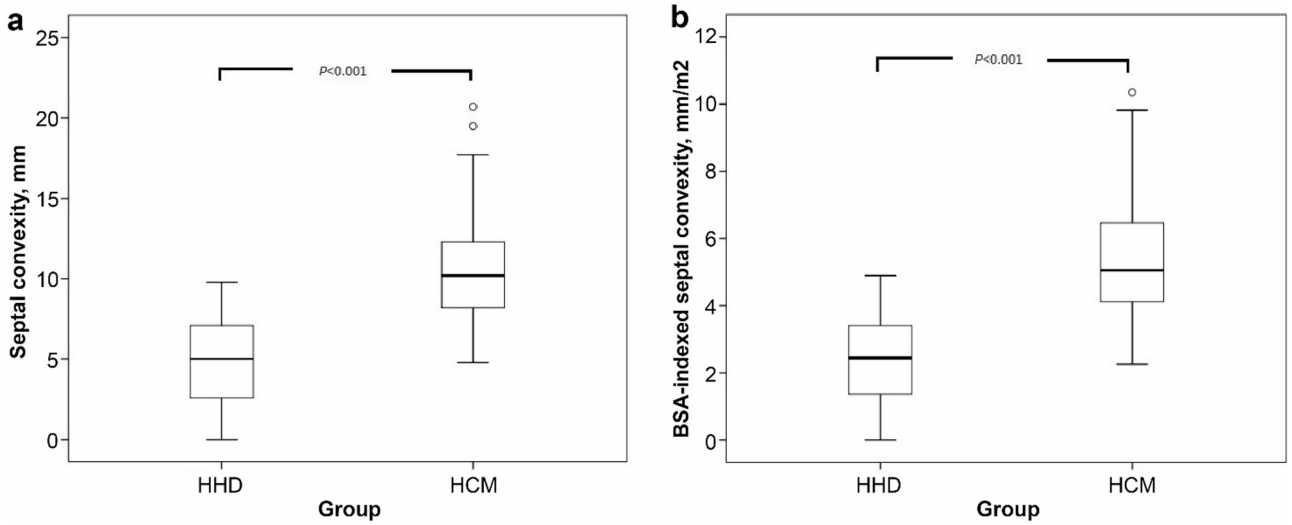

Fig. 4. Box plot of SC in the hypertensive (HHD) group and in the hypertrophic cardiomyopathy (HCM) groups (ALFA and MYBPC groups combined). The upper edge of the box represents the 75th percentile, and the lower edge represents the 25th percentile. The line across the box indicates the median. The small circles represent outliers. There was a significant increase in SC in the combined HCM group compared to the HHD group in both the (**a**) non-indexed and (**b**) BSA-indexed analyses.

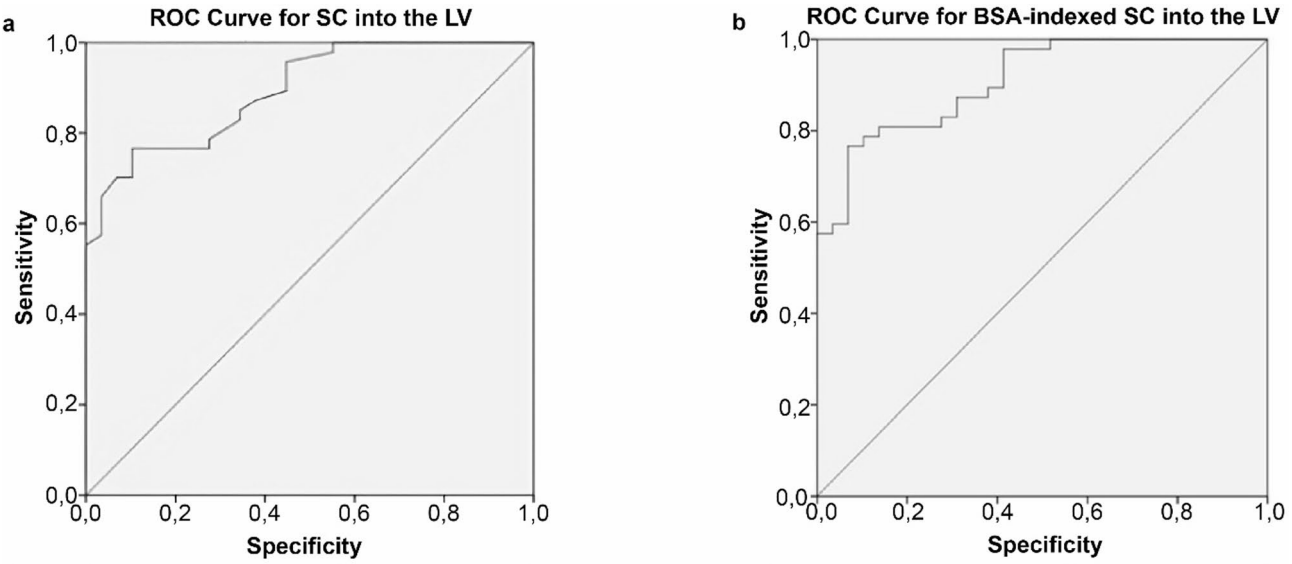

Fig. 5. Receiver operating characteristic (ROC) curves of septal convexity (SC) as a predictor of hypertrophic cardiomyopathy (ALFA and MYBPC groups combined) rather than hypertensive heart disease. Controls are not included. SC with a cutoff point of 7.9 mm performed best, with a sensitivity of 77% and specificity of 90% in the non-indexed study (**a**). The body surface area (BSA)-indexed SC has a cutoff point of 3.7 mm/m2, with a sensitivity of 81% and specificity of 86% (**b**).

HCM cases[8,25] and even in familial cases, the causative mutation is often not identified by genetic testing. On the other hand, many hypertensive patients present with mild to moderate asymmetric LVH. In these patients, the use of increased indexed LV mass and the absence of LGE[22] or SAM of the mitral valve to discriminate between HCM and HHD[13] is often not helpful. T1 mapping has shown encouraging results in the discrimination of HCM and HHD[17,26]; however, global native T1 values differ between HCM and HHD patients only modestly, with an accuracy of 64%. In the total CMR-derived radiomic texture analysis, which is not available in clinical practice, six selected texture features provided a maximum diagnostic accuracy of 86.2%[22]. Consequently, there is a group of patients with hypertension and mild-to-moderate LVH in whom it has thus far been difficult to confirm or rule out HCM.

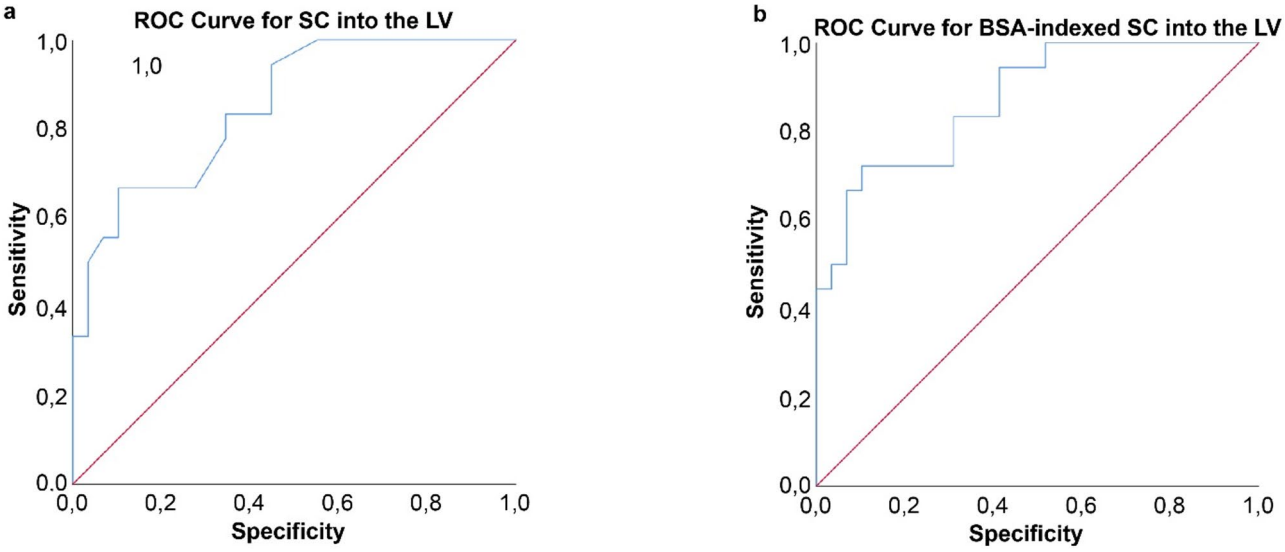

Fig. 6. Receiver operating characteristic (ROC) curves of SC as a predictor of hypertrophic cardiomyopathy (HCM) rather than hypertensive heart disease when including HCM patients with left ventricular maximal wall thickness < 18 mm only. A cutoff value of 7.9 mm yielded 67% sensitivity and 90% specificity (6.4 mm with a sensitivity of 83% and specificity of 65%) (**a**). With BSA-indexed SC values, a cutoff of 3.8 mm/m2 performed best, with a sensitivity of 72% and specificity of 90% (3.2 mm/m2 with a sensitivity of 83% and specificity of 69%) (**b**).

LV SC with a cutoff point of > 7.9 mm differentiated HCM from HHD, with an AUC of 89%, a sensitivity of 77%, and a specificity of 90%. When indexed for BSA, SC sensitivity was higher, but specificity was somewhat lower. In the subgroup analysis including HHD patients vs. HCM patients with moderate LVH, SC of 7.9 mm performed with comparable sensitivity and specificity. Consequently, including SC in the pattern of CMR measurements in patients with LVH of unknown etiology appears worthwhile.

In the current study and our previous investigation, subjects with HCM-causing mutations exhibited significant correlations between SC and age, as well as CMR-derived LV maximal thickness and LV mass. These findings suggest age-related LV septal remodelling in HCM, with SC representing one component of this process[19]. In the present study, in subjects with HHD, SC was negatively correlated with age and LV mass, and there was no correlation with BP, BSA or CMR-derived LV maximal thickness, suggesting that in HHD, SC is an early manifestation of LV remodelling and is attenuated with progressing LVH. Indeed, the LV septal remodelling process, reflected as an increased SC in subjects with HCM, appears to differ from that in subjects with LVH due to hypertension.

SC is measured in 4-chamber CMR images, which are usually included in CMR protocols used in the clinical setting. Therefore, they are readily available for measurement in every subject undergoing CMR. The reproducibility of SC measurements is high. In our previous study, we reported a 96% correlation in SC intraobserver measurements and a 97% correlation in interobserver measurements[19].

The study had several strengths. First, the study populations of the present study are well defined, including patients with confirmed hypertension and CMR-derived LVH, and HCM caused by two major Finnish founder mutations, *MYBPC3*-Q1061X and *TPM1*-D175N[2,7,25]. *MYBPC3* mutations account for approximately 40% of all HCM cases worldwide[27]. The Q1061X mutation in the *MYBPC3* gene leads to a truncated protein, similar to two-thirds of all *MYBPC3* mutations[24,28,29]. *TPM1*-D175N is a well-characterized missense mutation in another sarcomere gene[8,20,23,30]. These two pathogenic mechanisms, haploinsufficiency and poison polypeptide incorporation, are the mechanisms by which sarcomere gene mutations cause HCM[7]. As these major Finnish mutations represent different pathogenic mechanisms, we studied SC separately in both mutation groups and found that both mutation types induce increased SC. The SC values in subjects with the *MYBPC3*-Q1061X mutation were somewhat higher than those in subjects with *TPM1*-D175N, but the difference between the groups was not statistically significant. Thus, the results of the present study are likely applicable to HCM caused by various sarcomere mutations, particularly in cases caused by truncating mutations of the *MYBPC3* gene, which are very common causes of HCM. Second, the CMR protocol of the study was consistent, and the SC values were highly reproducible. Third, the clear-cut differences in SC between the HCM and HHD groups suggest that SC measurements may be useful in clinical evaluation of patients with LVH. However, the number of subjects in the present study was relatively small, and larger studies on patients with hypertensive heart disease and HCM caused by many different mutations are needed to confirm the utility of measuring SC. In addition, as there were only HCM patients with asymmetrical septal hypertrophy in the present study, the value of SC in the differentiation between apical or concentric HCM and HHD could not be studied.

Distinguishing HCM from HHD is a common clinical problem, particularly in patients with moderate LVH. CMR-derived SC measurement is a new and simple method for discriminating HCM due to sarcomere

mutations from HHD. Our findings support the concept of applying not only genetic testing but also CMR, including SC measurement, in the diagnostic work-up of patients with LVH of unknown etiology. Measuring SC appears particularly useful in subjects with moderate LVH with LV maximal wall thickness less than 18 mm, in whom differential diagnosis cannot be based on the severity of LVH only.

Conclusions

CMR-derived septal convexity values are higher in subjects with HCM than in those with HHD. Our study shows that measuring CMR-derived septal convexity is straightforward and enhances diagnostic performance, providing a novel and promising technique to distinguish between HCM caused by sarcomere mutations and hypertension-induced LVH.

Data availability

Data contain medical information of the patients and are not publicly available to preserve individuals' privacy. The analysed data are partly available from the corresponding author on reasonable request.

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Declarations

Competing interests
The authors declare no competing interests.

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