Prevalence and predictors of asymmetric hypertensive heart disease: insights from cardiac and aortic function with cardiovascular magnetic resonance

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Aims
We sought to determine the prevalence of asymmetric hypertensive heart disease (HHD) overlapping morphologically with hypertrophic cardiomyopathy (HCM) and to determine predictors of this pattern of hypertensive remodelling.

Methods and results
One hundred and fifty hypertensive patients underwent 1.5 T cardiovascular magnetic resonance imaging. Twenty-one patients were excluded due to concomitant cardiac pathology that may confound the hypertrophic response, e.g. myocardial infarction, moderate–severe valvular disease, or other cardiomyopathy. Asymmetric HHD was defined as a segmental wall thickness of ≥15 mm and ≥1.5-fold the opposing wall in ≥1 myocardial segments, measured from short-axis cine stack at end-diastole. Ambulatory blood pressure, myocardial replacement fibrosis, aortic distensibility and aortoseptal angle were investigated as predictors of asymmetric HHD by multivariate logistic regression. Out of 129 hypertensive subjects (age: 51±15 years, 50% male, systolic blood pressure: 170±30 mmHg, diastolic blood pressure: 97±16 mmHg), asymmetric HHD occurred in 21%. Where present, maximal end-diastolic wall thickness (EDWT) was 17.8±1.9 mm and located exclusively in the basal or mid septum. In asymmetric HHD, aortoseptal angle (114±10° vs. 125±9° vs. 123±12°, P<0.05, respectively) was significantly reduced compared to concentric left ventricular hypertrophy (LVH) and compared to no LVH, respectively. Aortic distensibility in asymmetric HHD (1.01±0.60 vs. 1.83±1.65 mm²/mmHg × 10⁻³, P<0.05, respectively) was significantly reduced compared to subjects with no LVH. Age (odds ratio [95th confidence interval]: 1.10 [1.02–1.18], P<0.05) and indexed LV mass (1.09 [0.98–1.28], P<0.0001) were significant, independent predictors of asymmetric HDD.

Conclusions
Asymmetric HHD morphologically overlapping with HCM, according to the current ESC guidelines, is common. Postulating a diagnosis of HCM on the basis of EDWT of ≥15 mm should be made with caution in the presence of arterial hypertension particular in male subjects with elevated LV mass.

Keywords
hypertension • hypertrophy • hypertrophic cardiomyopathy • cardiac magnetic resonance

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Introduction
Arterial hypertension affects an estimated 25% of the worldwide adult population. Different patterns of hypertensive heart disease (HHD) are recognized. Both the original echocardiographic classification of hypertensive left ventricular (LV) remodelling and hypertrophy and the more recent cardiovascular magnetic resonance imaging (CMR) revision to this classification describe symmetrical patterns of HHD only. Asymmetric patterns of HHD have been described with 2D echocardiography. CMR offers precise measurements of LV mass, volume, and wall thickness and is the current non-invasive gold standard investigation for assessing these parameters and LV systolic function.

The prevalence and asymmetric LV phenotypes according to the CMR gold standard have previously been described in the context of aortic stenosis. However, no such comprehensive CMR data currently exist for arterial hypertension, which is the common disease state of increased afterload.

CMR is emerging as a useful imaging technique in certain subjects with arterial hypertension and has been recognized in the recent European Society of Hypertension/Cardiology hypertension guidelines, particularly due to its tissue characterization properties. As such, understanding the prevalence of asymmetric HHD is important as an increasing number of patients with hypertension and/or suspected hypertrophic cardiomyopathy (HCM) are being referred for CMR to distinguish between the two pathologies and the number is set to increase with the increased availability of CMR.

Consequently, the aims of this study were to describe the prevalence and predictors of asymmetric HHD, using CMR.

Methods
Study population
Patients with hypertension were recruited from the Bristol Heart Institute tertiary hypertension clinic between February 2012 and April 2015. The local research ethics committee confirmed that the study conformed to the governance arrangements for research ethics committees. Subjects provided written consent. Baseline demographic and clinical characteristics were recorded, including reviewing the baseline 12-lead electrocardiograph for the presence of LV hypertrophy (LVH) by Sokolow–Lyon voltage criteria and for ECG-strain pattern, defined as ≥1 mm concave down-sloping ST-segment depression and asymmetrical T-wave inversion in the lateral leads, by an experienced clinician blinded to the CMR data. To investigate asymmetric HHD only, the study cohort was carefully selected to exclude patients with any concomitant myocardial pathology that may confound the remodelling pattern and/or the hypertrophic response. Exclusion criteria therefore included any evidence of moderate–severe valvular heart disease, acquired or inherited cardiomyopathy, and suspected athlete’s heart. Aortic valve pathology was excluded by radial cine imaging of the aortic valve and phase contrast magnetic resonance angiography images in the aortic root. Mitral valve disease was excluded by visual assessment on the four-chamber, three-chamber, two-chamber, and short-axis cines. In particular, HCM was excluded on the basis of clinical data, family history, and electrocardiographic features supportive of this diagnosis. A severely decreased estimated glomerular filtration rate of <30 mL/min/1.73 m² was also an exclusion criterion.

Average office systolic (SBP) and diastolic blood pressures (DBP) were acquired in all subjects after seated rest from both arms, assessed using standard automated sphygmomanometry with an appropriately sized cuff. Patients were stratified by hypertension severity on the basis of their office blood pressure level in accordance with the 2013 ESH/ESC hypertension guidelines. In a subgroup of hypertensive subjects (n = 85), standard 24-h ambulatory blood pressure monitoring was also performed.

CMR protocol
CMR was performed at 1.5 T (Avanto, Siemens, Erlangen, Germany). Steady-state free precession short-axis whole LV cines (8 mm slice thickness, no slice gap, temporal resolution 38.1 ms, echo time 1.07 ms, representative field-of-view in-plane pixel size 1.5 × 0.8 mm) were used for the estimation of LV mass (LVM) and volumes, which were then indexed to body surface area, as previously described. Previ-ously validated threshold detection software (CMR42, Circle Cardiovascular Imaging, Inc., Calgary, Canada) was used to include papillary muscles and LV trabeculation in LVM estimation in accordance with the latest Society of CMR guidelines. Papillary muscles and trabeculations were then included in the blood pool volume for the assessment of end-diastolic volume (EDV), end-systolic volume (ESV), and stroke volume (SV), as described previously. LVH was defined as indexed LV mass > upper 95th confidence interval of established age- and gender-specific CMR reference ranges. The LV mass/volume ratio (M/V), CMR equivalent of the echocardiogram-derived relative wall thickness measurement, was measured by dividing LVM by EDV. Maximal wall thickness was defined as the end-diastolic wall thickness (EDWT) measured in the middle of the thickest segment according to the American Heart Association 17-segment model from the LV short-axis cines, excluding LV and right ventricular (RV) trabeculations. Asymmetric wall thickness was defined as a regional wall thickness of ≥15 mm in ≥1 myocardial segments, in accordance with European guidelines on the diagnosis of HCM, and segmental EDWT > 1.5-fold the opposing LV segment, as previously described (Figure 1). Such measurements from short-axis cine CMR images have previously demonstrated good inter- and intraobserver variability. Global longitudinal strain was measured with voxel-tracking post-processing software (TissueTracking, CMR42, Circle Cardiovascular Imaging, Inc., Calgary, Canada) using four-chamber and two-chamber cines. All measurements were performed by an experienced CMR reader, blinded to clinical data.

Myocardial replacement fibrosis was assessed by late gadolinium enhancement (LGE). An inversion-recovery fast gradient-echo sequence carried out in two phase-encoding directions was performed ∼10–15 min after intravenous administration of 0.1 mmol/kg gadobutrol (Gadovist, Bayer Pharma AG, Germany). Tailored inversion times were used in each patient to null the myocardium. The presence of LGE was quantified by visual analysis by two independent experienced CMR readers blinded to the clinical and remodelling/hypertrophy data. Any discrepancies were resolved by consensus.

Aortic distensibility
As previously described, ascending aortic distensibility was estimated as follows: distensibility = ΔP/(Aaortic × ΔP), measured from cine images perpendicular to the ascending aorta at the level of the right pulmonary artery, where Aaortic is the ascending aortic area at end-diastole and ΔP (in mmHg) is the pulse pressure estimated from SBP minus DBP. Excellent interobserver agreement and reproducibility of this measure have previously been reported. Aortic distensibility measurements were...
carried out by an experienced CMR reader, blinded to all other CMR and clinical data. The aortoseptal angle was measured from the three-chamber CMR cine with a previously described and reproducible method, which is a modification of the original echocardiographic technique. The aortoseptal angle was defined as the angle between a line drawn along the border of the right and left interventricular septum (parallel to the proximal RV endocardial border) and a line drawn through the long axis of the aortic root, where a value of 180° would be a straight line from septum to aorta and reducing values representing increased angulation (Figure 2). Aortoseptal angle measurements were performed by an experienced CMR reader, blinded to all other CMR and clinical data.

Statistical analysis
Statistical analysis was performed using SPSS Version 21 (IBM Corp., Armonk, NY, USA). Normally distributed continuous variables were expressed as mean ± standard deviation and compared using unpaired Student’s t-test, with post hoc correction for multiple t-tests, or one-way analysis of variance with least significant difference post hoc correction as appropriate. Categorical variables were expressed as percentages and analysed using the Fisher’s exact test. R-values quoted are for Pearson’s correlation coefficient. Univariate and multivariate logistic regression analysis was performed to identify predictors of asymmetric HHD morphologically overlapping with HCM. Statistical significance was set at two-sided P < 0.05.

Results
Study population
One hundred and fifty hypertensive patients underwent CMR. Twenty-one patients were excluded (Figure 3), including nine subjects with subendocardial LGE consistent with previous myocardial infarction (MI), resulting in a final study size of 129 patients (age: 50.8 ± 15.2 years, 49.6% male, SBP: 170.4 ± 30.0 mmHg, DBP: 97.3 ± 15.5 mmHg). There was no difference in the prevalence of diabetes mellitus or history of ischaemic heart disease between the cohorts. ECG evidence of LVH was significantly more common in subjects with CMR-defined LVH with no asymmetric wall thickening, ECG-strain pattern was significantly more common in subjects with asymmetric wall thickening, but the overall prevalence of these ECG features was low (Table 1).

Prevalence of asymmetric HHD
In our cohort consisting exclusively of patients with hypertension, asymmetric EDWT of ≥15 mm in ≥1 myocardial segments and ≥1.5-fold the opposing LV wall occurred in 21% (n = 27) (Table 2). Subjects with asymmetric EDWT were significantly older than both subjects with concentric LVH and subjects with normal indexed LV mass (57 ± 13 vs. 48 ± 14 vs. 49 ± 16 years, P < 0.05, respectively), and there was a significantly higher proportion of male subjects (74 vs. 48 vs. 43%, P < 0.05, respectively). Despite similar LV ejection fraction, subjects with asymmetric wall thickness had the lowest global longitudinal strain (Table 2).

Location and magnitude of the asymmetric hypertrophic response
Patients with asymmetric HHD had significantly higher maximal EDWT compared with those with concentric LVH (18 ± 2 vs. 13 ± 1 mm, P < 0.05; Table 2). Furthermore, indexed LV mass was significantly higher in subjects with asymmetric HHD compared with subjects with LVH but without wall asymmetry (109 ± 27 vs. 96 ± 10 g/m², P < 0.05). In asymmetric HHD, the maximal EDWT was exclusively located in the basal or mid septum. The segmental distribution and magnitude of asymmetrical EDWT is demonstrated in Figure 4.

Myocardial replacement fibrosis
The anatomical location of replacement fibrosis is demonstrated in Figure 5. Mid-wall myocardial replacement fibrosis was significantly more common in subjects with asymmetric EDWT (15 vs. 0 vs. 1%, P < 0.05, respectively). However, the overall prevalence of mid-wall LGE was low in our patient population at 4% (n = 5). The prevalence of RV insertion point LGE was significantly higher in subjects with asymmetric wall thickness compared with subjects

Figure 1 (A) Normal, (B) concentric LV hypertrophy, and (C) asymmetric LV hypertrophy forms of HHD.
without LVH (41 vs. 9%, $P < 0.05$), but not significantly different from those subjects with concentric LVH (41 vs. 22%, $P = 0.07$).

**Aortic function**

The aortoseptal angle in subjects with asymmetric HHD was significantly lower (implying a more acute angle between the anatomical structures) than in subjects with concentric LVH and than in subjects without LVH (114 $\pm$ 10° vs. 125 $\pm$ 9° vs. 123 $\pm$ 12°, $P < 0.05$, respectively; Table 2). Aortic distensibility was significantly reduced in subjects with asymmetric EDWT compared with those without wall asymmetry and without LVH (1.01 $\pm$ 0.60 vs. 1.83 $\pm$ 1.65 mm$^2$/mmHg $\times 10^3$, $P < 0.05$). Increasing EDWT correlated with significant reduction in aortic distensibility ($R = -0.302$, $P < 0.001$) and significant reduction in aortoseptal angulation ($R = -0.414$, $P < 0.0001$).

**Predictors of asymmetric HHD**

In univariate analysis, increasing age, male gender, increasing body mass index, increasing indexed LV mass, lower aortic distensibility, and lower aortoseptal angle were all significant predictors of asymmetric HHD (see Supplementary data online). However, only increasing age (odds ratio [95th confidence interval]: 1.10 [1.02–1.18], $P < 0.05$) and increasing indexed LV mass (1.09 [1.04–1.14], $P < 0.05$) remained significant predictors in the multivariate logistic regression statistical model.

**Discussion**

To our knowledge, this is the first study to define the prevalence of asymmetric HHD with CMR. Asymmetric EDWT of $\geq 15$ mm and $>1.5$-fold the opposing myocardial segment in $\geq 1$ segments...
occurred in 21% of our purely hypertensive cohort. Our results demonstrate how frequently HHD overlaps morphologically with HCM according to the EDWT threshold of 15 mm advocated by the European HCM guidelines.

We also show that advanced hypertrophic response and increasing age are independent predictors of the asymmetric hypertensive phenotype. Multivariate logistic regression analysis confirms that the higher prevalence of male gender and higher BMI in the asymmetric cohort, which may be potential confounding factors of the hypertrophic process, do not exert significant independent effects.

Asymmetric LV responses have been recognized in health and disease. Goor et al. first coined the term ‘sigmoid septum’, describing variations in the septal contour in 50 ex vivo human hearts of varying ages. More recently, in a CMR study of young healthy army recruits, the prevalence of LV asymmetry, as defined as EDWT of ≥13 mm and >1.5-fold the opposing myocardial segment, was 2.2% at baseline, increasing to 10% following a period of intensive physical training. In the context of hypertension, Wicker et al. have previously documented a prevalence of 5% of asymmetric septal hypertrophy in a 2D echocardiographic study. Their definition of LV asymmetry consisted of >1.3 times the free LV wall, and did not have an absolute EDWT threshold. In contrast, we observed a higher prevalence of hypertensive LV asymmetry with CMR. A putative explanation for this relates to the better whole heart 3D coverage with contiguous short-axis cines and better tissue contrast of CMR, facilitating the identification of endocardial contours, relative to 2D echocardiography, which is a well-recognized phenomenon.

In our cohort, asymmetric wall thickness was exclusively located in the basal or mid septum. Asymmetric septal thickness has been described in echocardiographic and CMR studies of LVH secondary to aortic stenosis, the latter reported a prevalence of 27%, where a definition of asymmetry of ≥13 mm and >1.5-fold the opposing myocardial segment was employed. Interestingly, those subjects with aortic stenosis and asymmetric septal thickness in both aforementioned studies had high prevalence of concomitant hypertension. Our results, in a cohort with strict exclusion of valvular heart disease and other potential hypertrophic confounding pathologies, raise the question of the relative important of the type of afterload (aortic stenosis or arterial hypertension or a combination thereof) in the development of the asymmetric phenotype.

The reason that some patients develop asymmetric thickening is unclear. The fact that the basal septum is a site of increased wall stress may be implicated and may explain the common appearance in both aortic stenosis and systemic hypertension, which both have increased afterload. Puntmann et al. demonstrated that impaired deformation follows the areas of increased wall stress in HHD. Our data show more acute aortoseptal angulation and less aortic distensibility in hypertensives with basal or mid septal myocardial asymmetrical thickening. This may result in increased LV wall stress in this region of myocardium, driving asymmetric wall thickening. Our findings are consistent with those of Goor et al. who found that increased aortic root angulation was associated with increasing septal prominence in their study of 50 ex vivo human hearts. We are unable to distinguish cause from effect between aortic function and asymmetric LVH in our observational study. Age-related

Figure 3. Study flow chart. *Image artefact from an implantable loop recorder device precluding volumetric assessment from LV short-axis stack. CMR, cardiovascular magnetic resonance imaging; MI, myocardial infarction (defined as subendocardial LGE on CMR); HCM, hypertrophic cardiomyopathy; LVNC, left ventricular non-compaction cardiomyopathy; DCM, idiopathic dilated cardiomyopathy; Mod AR, moderate aortic regurgitation; AVR, aortic valve replacement.
changes in aortic configuration and function and/or duration of hypertension may be important factors in this observed relationship. Septal thickness increasing with age is consistent with previous work.  

Equally, the denser sympathetic innervation of the interventricular septal relative to the lateral wall has been postulated as a pathophysiological explanation for asymmetric LV phenotypes.  

Certainly, sympathetic activation is recognized in some, but not all, patients with essential hypertension, which may account for the heterogeneity of this appearance within hypertensive subjects.  

A further putative mechanism relates to the AT1 angiotensin II receptor subtype which has been shown to mediate protein synthesis and hypertrophy in rat models.  

Furthermore, AT1 receptor up-regulation has been demonstrated in spontaneously hypertensive and renovascular hypertensive rats with LVH.  

Differences in location and expression of AT1 could, theoretically, account for asymmetric LV wall thickening.  

The exclusive location of asymmetry occurring in the basal to mid-septal myocardial segments and the absolute mean wall thickness of 18 ± 2 mm in our cohort may have clinical implications. The European Society of Cardiology guidelines advocate that a diagnosis of HCM be considered if regional wall thickness is \( \geq 15 \) mm in one or more LV myocardial segments or \( \geq 13 \) mm in a first-degree relative of someone with HCM, measured by any imaging technique. The guidelines concede that the diagnosis should only be made in the absence of any abnormal loading conditions, but do not provide a description of the predictable LV appearances in hypertension, a state of abnormal afterload. Our study demonstrates that an asymmetric hypertensive phenotype with morphological overlap with the conventional HCM EDWT criterion occurs in approximately one in five hypertensive subjects. These results highlight that the diagnosis of HCM on the basis of wall thickness alone should be made with caution in the context of concomitant hypertension. The low prevalence of LGE in our cohort, which is consistent with other studies of HHD, may be a useful discriminator as LGE has been described in up to 72% of patients with HCM. LGE is a marker of focal replacement fibrosis. Future study may involve assessment of the extent and distribution of diffuse myocardial fibrosis, which can now be reliably measured with native and post-contrast CMR T1-mapping techniques.
aortoseptal angulation of $\leq 100^\circ$ had 91% specificity for predicting provocable LV outflow tract obstruction in HCM. This degree of angulation is even more acute than our cohort of hypertensive subjects with asymmetric wall thickening. However, studies directly comparing appropriately matched subjects with asymmetric HHD and HCM are required to confirm these findings.

**Table 2** CMR parameters

<table>
<thead>
<tr>
<th>CMR volumetrics and wall thickness</th>
<th>All patients ($N = 129$)</th>
<th>No asymmetric wall thickening No LVH ($N = 79$)</th>
<th>LVH ($N = 23$)</th>
<th>Asymmetric wall thickening ($N = 27$)</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Maximal EDWT (mm)</td>
<td>13 ± 3</td>
<td>12 ± 2</td>
<td>13 ± 1</td>
<td>18 ± 2</td>
<td>$&lt;0.0001^{abc}$</td>
</tr>
<tr>
<td>iLVM (g/m$^2$)</td>
<td>84 ± 22</td>
<td>72 ± 10</td>
<td>96 ± 10</td>
<td>109 ± 27</td>
<td>$&lt;0.0001^{abc}$</td>
</tr>
<tr>
<td>LVEF (%)</td>
<td>68 ± 9</td>
<td>69 ± 7</td>
<td>64 ± 11</td>
<td>70 ± 12</td>
<td>$&lt;0.05^{bc}$</td>
</tr>
<tr>
<td>iEDV (mL/m$^2$)</td>
<td>77 ± 17</td>
<td>72 ± 12</td>
<td>91 ± 15</td>
<td>79 ± 24</td>
<td>$&lt;0.0001^{bc}$</td>
</tr>
<tr>
<td>iESV (mL/m$^2$)</td>
<td>25 ± 12</td>
<td>23 ± 7</td>
<td>33 ± 13</td>
<td>25 ± 18</td>
<td>$&lt;0.001^{bc}$</td>
</tr>
<tr>
<td>iSV (mL/m$^2$)</td>
<td>52 ± 8</td>
<td>50 ± 7</td>
<td>55 ± 8</td>
<td>52 ± 9</td>
<td>$&lt;0.05^{c}$</td>
</tr>
<tr>
<td>MVi (g/mL)</td>
<td>1.12 ± 0.28</td>
<td>1.02 ± 0.21</td>
<td>1.08 ± 0.18</td>
<td>1.44 ± 0.28</td>
<td>$&lt;0.0001^{ab}$</td>
</tr>
<tr>
<td>Cardiac output (L/min)</td>
<td>7.49 ± 1.87</td>
<td>7.34 ± 1.75</td>
<td>7.63 ± 1.89</td>
<td>7.80 ± 2.21</td>
<td>0.67</td>
</tr>
<tr>
<td>Cardiac index (L/min/m$^2$)</td>
<td>3.67 ± 0.77</td>
<td>3.71 ± 0.77</td>
<td>3.69 ± 0.70</td>
<td>3.56 ± 0.85</td>
<td>0.51</td>
</tr>
<tr>
<td>Global longitudinal strain (%)</td>
<td>−16.6 ± 4.0</td>
<td>−17.6 ± 3.5</td>
<td>−15.8 ± 4.6</td>
<td>−14.6 ± 3.9</td>
<td>$&lt;0.05^{bc}$</td>
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<tr>
<td>Replacement fibrosis</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>LGE present, n (%)</td>
<td>27 (21)</td>
<td>7 (9)</td>
<td>6 (26)</td>
<td>14 (52)</td>
<td>$&lt;0.005^{ab}$</td>
</tr>
<tr>
<td>Mid-wall LGE, n (%)</td>
<td>5 (4)</td>
<td>1 (1)</td>
<td>0 (0)</td>
<td>4 (15)</td>
<td>$&lt;0.005^{ab}$</td>
</tr>
<tr>
<td>RV insertion point LGE, n (%)</td>
<td>23 (18)</td>
<td>7 (9)</td>
<td>5 (22)</td>
<td>11 (41)</td>
<td>$&lt;0.05^{c}$</td>
</tr>
<tr>
<td>Aortic function</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Aortic distensibility (mm$^2$/mmHg $\times 10^3$)</td>
<td>1.63 ± 1.44</td>
<td>1.83 ± 1.65</td>
<td>1.57 ± 1.05</td>
<td>1.01 ± 0.60</td>
<td>0.07$^a$</td>
</tr>
<tr>
<td>Aortoseptal angle (°)</td>
<td>122 ± 11</td>
<td>123 ± 12</td>
<td>125 ± 9</td>
<td>114 ± 10</td>
<td>$&lt;0.005^{ab}$</td>
</tr>
</tbody>
</table>

*a* Asymmetric wall thickening vs. no asymmetric wall thickening and no LVH, $P < 0.05$.

*b* Asymmetric wall thickening vs. no asymmetric wall thickening and LVH, $P < 0.05$.

*c* No asymmetric wall thickening and LVH vs. no asymmetric wall thickening and no LVH, $P < 0.05$.

**Figure 4** 16-segment American Heart Association bull’s eye plots demonstrating (A) the location of maximal wall thickness and (B) the magnitude (mean ± SD) of maximal wall thickness in ventricles with asymmetric wall thickness.
Limitations

There are several important limitations of this study. We were unable to investigate the influence of duration of hypertension due to the prolonged subclinical course of systemic hypertension. In addition, myocardial ischaemia was not formally excluded with anatomical or functional testing. As a result, this could confound the patterns of HHD observed. However, hypertension is a risk factor for coronary atheroma and hypertensive LVH itself is associated with myocardial ischaemia, so we feel that exclusion of such patients would not have been appropriate.

We have been unable to determine the prognostic implications of asymmetric HHD due to relatively low annual event rates and only short-term follow-up of the cohort to date. Asymmetric LVH has been demonstrated to be an important marker of adverse prognosis in aortic stenosis. Longitudinal outcome studies, or even retrospective re-analysis of previous CMR studies of HHD that did not account for asymmetry, are required to confirm or refute whether asymmetric HHD has significant prognostic implications.

Conclusions

Asymmetric HHD, morphologically overlapping with HCM, is common and occurs exclusively in the basal to mid-septum. Our results highlight that the diagnosis of HCM on the basis of wall thickness alone should be made with caution in the content of concomitant hypertension. More acute aortoseptal angulation and reduced aortic distensibility were observed in subjects with asymmetric HHD. However, increasing age and indexed LV mass were the only independent, significant predictors of asymmetric LV thickening in hypertension. Consequently, our results suggest that significant asymmetry in young hypertensive subjects is less likely to be related to their arterial hypertension.

Supplementary material

Supplementary material is available at European Journal of Echocardiography online.

Acknowledgements

NIHR Cardiovascular Biomedical Research Unit, Bristol Heart Institute.

Conflict of interest: The views expressed are those of the authors and not necessarily those of the National Health Service, National Institute for Health Research, or Department of Health.

Funding


References

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