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Abstract: Echocardiography-derived measurements of maximum left ventricular (LV) wall thickness are important for both the diagnosis and risk stratification of hypertrophic cardiomyopathy (HC). Cardiac Magnetic Resonance (CMR) imaging is increasingly being used in the assessment of HC however, little is known about the relationship between wall thickness measurements made by the 2 modalities. We sought to compare measurements made with echocardiography and CMR and to assess the impact of any differences on risk stratification using the current European Society of Cardiology (ESC) guidelines. Maximum LV wall thickness measurements were recorded on 50 consecutive patients with HC. 69% of LV wall thickness measurements were recorded with echocardiography, compared to 69% from CMR ($p<0.001$). There was poor agreement on the location of maximum LV wall thickness; weighted-Cohen's κ 0.14 (p 0.036) and maximum LV wall thicknesses were systematically higher with echocardiography than with CMR (mean 19.1±0.4mm vs 16.5±0.3mm, $p<0.01$ respectively), Bland-Altman bias 2.6mm (95% confidence interval -9.8 to 4.6). Inter-observer variability was lower for CMR (R2 0.67 echocardiography, R2 0.93 CMR). The mean difference in 5-year sudden cardiac death (SCD) risk between echocardiography and CMR was 0.49±0.45% (p=0.37). When classifying patients (low, intermediate or high risk), 6 patients were reclassified when CMR was used instead of echocardiography to assess maximum LV wall thickness. These findings suggest that CMR measures of maximum LV wall thickness can be cautiously used in the current ESC risk score calculations, although it is preferable to use wall measurements recorded by echocardiography.

Usefulness of Cardiac Magnetic Resonance imaging to measure Left Ventricular wall thickness for determining risk scores in patients with Hypertrophic Cardiomyopathy

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Running Title Measuring LV wall thickness in HC

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Abstract

Echocardiography-derived measurements of maximum left ventricular (LV) wall thickness are important for both the diagnosis and risk stratification of hypertrophic cardiomyopathy (HC). Cardiac Magnetic Resonance (CMR) imaging is increasingly being used in the assessment of HC however, little is known about the relationship between wall thickness measurements made by the 2 modalities. We sought to compare measurements made with echocardiography and CMR and to assess the impact of any differences on risk stratification using the current European Society of Cardiology (ESC) guidelines. Maximum LV wall thickness measurements were recorded on 50 consecutive patients with HC. 69% of LV wall thickness measurements were recorded with echocardiography, compared to 69% from CMR ($p<0.001$). There was poor agreement on the location of maximum LV wall thickness; weighted-Cohen's κ 0.14 (p 0.036) and maximum LV wall thicknesses were systematically higher with echocardiography than with CMR (mean 19.1±0.4mm vs 16.5±0.3mm, $p<0.01$ respectively), Bland-Altman bias 2.6mm (95% confidence interval -9.8 to 4.6). Inter-observer variability was lower for CMR (R^2 0.67 echocardiography, R^2 0.93 CMR). The mean difference in 5-year sudden cardiac death (SCD) risk between echocardiography and CMR was 0.49±0.45% (p=0.37). When classifying patients (low, intermediate or high risk), 6 patients were reclassified when CMR was used instead of echocardiography to assess maximum LV wall thickness. These findings suggest that CMR measures of maximum LV wall thickness can be cautiously used in the current ESC risk score calculations, although it is preferable to use wall measurements recorded by echocardiography.

Keywords Hypertrophic Cardiomyopathy (HC), sudden cardiac death (SCD), Maximum Left Ventricular wall thickness, Cardiovascular Magnetic Resonance (CMR)

Hypertrophic cardiomyopathy (HC) is defined by the presence of increased left ventricular (LV) wall thickness, not solely explained by abnormal loading conditions [1]. HC is the most common cause of sudden cardiac death (SCD) in those under the age of 35 years old [2,3]. However, only a small subset of patients are at increased risk and identifying these patients remains challenging. Maximum LV wall thickness as assessed by 2D-echocardiography has been found to be of prognostic value in risk stratification [2,4,5]. Recently a clinical risk prediction model has been developed incorporating echocardiography-derived measurements of maximum LV wall thickness [6] and one of the major strengths of this prediction model is that maximum LV wall thickness is assessed in a continuous method, rather than dichotomously as previously advocated [2,4,7,8]. Echocardiography and CMR are both commonly used to evaluate patients with HC and in clinical practice LV wall thickness measurements are frequently used interchangeably. However, echocardiography may overestimate LV wall thickness when compared to CMR [9] or underappreciate hypertrophy in some settings. The magnitude of any differences in measurements between the two modalities and their impact on risk stratification using the European Society of Cardiology (ESC) risk score is unknown. The main objective of this study was to assess the presence and extent of any systematic difference between LV wall thickness measurements by echocardiography and CMR and to determine the effect of using CMR-derived LV wall thickness measurements on ESC SCD risk score.

Methods

Maximum LV wall thickness measurements were recorded on 50 consecutive patients with HC referred for echocardiography and CMR at our institution between 2014 and 2015. Patients with implantable cardioverter defibrillators (ICDs), pacemakers or other contraindications to CMR were excluded. Written informed consent was obtained from all patients and the National Health Service ()NHS Health Research Authority ethics committee (REC Study number 15/NS/0030) approved the study.

Each patient underwent transthoracic echocardiography according to the protocol set by the British Society Echocardiography (BSE) guidelines by a trained sonographer[10]. Images were stored digitally and analyzed offline by an experienced BSE-accredited clinician blinded to the CMR measurements. Standard measures of cardiac dimensions were determined from the mean of 3 cardiac cycles. The end-diastolic thickness of the anterior, septal, inferior and lateral segments were recorded at the basal, mid and apical parasternal short-axis levels [11]. Twenty-five patients were analysed by two clinicians to assess inter-observer variability.

All patients underwent CMR imaging using either a 1.5-T or 3.0-T scanners (Philips Achieva, Philips Healthcare, Best, Netherlands) according to standard acquisition protocols set by the Society for Cardiovascular Magnetic Resonance (SCMR) [12]. ECG-gated, breath-hold steady-state free precession cine images were acquired in both the long-axis and the short-axis planes from the apex to the base of the left ventricle, with slice thicknesses of 8 mm. Only short axis stacks that were on axis with the mitral valve annulus were used for analysis. Images were analyzed offline using dedicated software (CVI42, Circle Cardiovascular Imaging Inc, Calgary, Alberta, Canada) by an experienced CMR clinician, blinded to the echo results. The same segments were recorded on CMR as described above on echocardiography, and similarly, 25 patients were analysed by two clinicians to assess inter-observer variability.

Hospital databases and records were used to confirm the other risk factors (history of syncope, family history SCD, LV outflow tract gradient, age, non-sustained ventricular tachycardia (NSVT), left atrial size) identified in the ESC SCD risk score calculation.

Categorical data are presented as frequencies and percentages. Comparisons between CMR and echocardiography group were made using McNemar's test. Continuous data were assessed for normality using the Shapiro-Wilks test. Data are presented as mean ± standard deviation (SD) or as median (interquartile range) depending on normality and were compared using Student's t-test or the Wilcoxon signed rank method as appropriate.

Classification agreement was assessed using weighted-Cohen's κ. Comparisons of wall-thickness measurements made by the two modalities together with interobserver variability assessments were made using the methods of Bland and Altman [13]. Interobserver variability was also quantified using the Pearson correlation. Differences in risk classification between the two modalities were evaluated using the Stuart-Maxwell test for marginal homogeneity. For all statistical analysis, two-tailed values of $p<0.05$ were considered significant.

Results

Patients' demographics are summarized in Table 1. Only 12 patients had all the twelve AHA segments recorded using echocardiography. For direct comparison only matched data was used (Table 2). Figure 1 shows echo and CMR images from the same patient illustrating how challenging it can be to measure LV wall thicknesses accurately with echocardiography due to poor endocardial definition. There was poor agreement on the location of maximum LV wall thickness using AHA segments with weighted-Cohen's κ calculated at 0.14 (p 0.036), table 3.

Overall, the 50 measures of myocardial maximum LV wall thickness were higher on echocardiography than CMR (mean 19.1±0.4mm vs 16.5±0.3mm, $p<0.01$ respectively, Figure 2). A Bland Altman plot was used, demonstrating a systematic difference of 2.6±3.7mm (95% confidence interval -9.8 to 4.6), Figure 3. The interclass correlation was 0.51 (95% confidence intervals 0.3-0.7).

There was less variation in inter-observer variability with CMR than echocardiography. R was calculated as 0.82 for the echocardiography measurements of LV wall thickness, with R^2 0.67 compared to R 0.96 for CMR measurements of LV wall thickness, with R^2 0.93. A Bland Altman plot has been used to show these differences in inter-observer variability for echocardiography and CMR, Figures 4A and 4B.

The SCD risk score was calculated for all 50 patients using both the maximum LV wall thicknesses from echocardiography and CMR; patients were classified as low risk of

SCD (5 year risk under 4%) intermediate risk (5 year risk 4-6%) or high risk (5 year risk over 6%). There was no statistically significant difference in 5-year SCD risk score between echocardiography and CMR (0.47±0.4% (p=0.37). However, the maximum LV wall measurements from CMR resulted in 6 patients allocated to lower risk categories: with echocardiography, 2 patients were classified as high risk, 8 as intermediate and 40 as low risk whereas, with CMR, 1 patient was classified as high risk 5 intermediate and 44 patients as low risk, Figure 5. The two patients with maximum LV wall thickness on CMR located in segments not measured by echocardiography were classified as low risk with both echocardiography and CMR risk calculations.

Discussion

In this cohort of patients with HC, we found that the disparity of 2.6mm between echocardiography and CMR measurements of maximum LV wall thickness did not translate to a statistical difference in the current SCD risk scores, although did result in fewer patients being identified as high risk.

This is in contrast to Rickers et al, who showed no significant difference in LV wall thickness between echocardiography and CMR in 48 patients with HC, although the authors did not perform a detailed comparison between the imaging modalities [14]. Previous work has shown greater discrepancy in LV wall thickness measurements with echocardiography, with increasing distance from the transducer [15].

There are many possible reasons for the discrepancy between the two imaging modalities. CMR has better spatial resolution in patients with suboptimal echocardiography images and in the apical and anterolateral segments [16]. Another possible reason for discrepancy is that the echocardiogram images recorded were analysed retrospectively and so we had to analyse the images that the sonographers have saved. In comparison, when we analysed the CMR we were able to choose the basal, mid and apical slices from the short axis stack, since the images were acquired with full LV coverage and without obliquity.

Moreover, with CMR the ventricular slices could always be acquired parallel to the mitral valve annulus, whereas with echocardiography the same degree of standardization was not possible. CMR has previously been shown to have superior inter-observer variability due to improved spatial resolution [9], and this was confirmed in this study.

From the echocardiographic data recorded it was not possible to record all LV wall thicknesses, particularly in the apical slices. This is important, as one of the benefits of CMR is the improved differentiation of the LV wall compared to the papillary muscles and surrounding trabeculation. Two patients had apical hypertrophy on CMR but no echocardiographic apical measurements, although had a diagnosis of HC made from the other segments on echocardiography. They were both eventually categorized at low risk.

Apart from previous sustained ventricular tachycardia, no one risk factor can be used in isolation to identify patients at high risk of SCD. HC remains one of the most frequent cause of SCD in the young, and in athletes under 35 years old in countries without systematic sports screening programmes [17]. The greatest number of SCD events occur in patients with LV wall thickness between 20 and 25mm[6]. On its own, a 2.6mm difference in LV wall thickness does not change the ESC recommendation or change the patient's individual risk, but the addition of more risk factors increases the chance of SCD.

The importance of echocardiography is clear in establishing the risk of sudden cardiac death, not least as half of the pre-specified predictor variables are assessed on echocardiography (maximum LV wall thickness, LA diameter, LV outflow tract obstruction and fractional shortening – although the latter was not included in the final risk model). In addition, most clinicians have better access to echocardiography than CMR. Despite this, CMR has a clear role in assessing if fibrosis is present and its extent, as well as confirming LV volumes and wall mass. In 2014, Elliott et al recommended that CMR should be considered in patients with HC at their baseline assessment if local resources and expertise permit [18]. A year later, Cardim et al, proposed a multimodality imaging approach to patients with HC [19]. The authors recommended echocardiography as the first line imaging modality

for family screening and for pre clinical diagnosis, but for CMR when image quality is suboptimal, in high-risk families when echocardiography is non-diagnostic and when a ‘more complete SCD risk assessment’ is required. The suggestion is that these LV wall measurements can be used interchangeably, although there is no previous evidence supporting this. Our study in 50 patients is the first to show a statistically significant difference between echocardiography and CMR maximum LV wall measurements. These differences did not translate into a statistically significant difference in SCD risk scores, although using CMR did result in 6 patients being reclassified into lower risk groups. As LV wall thickness is a continuous variable in the risk predictor model, we recommend that echocardiographic measurements be used if available and acceptable image quality, and CMR measurements can be used cautiously if appropriate. Studies such as the Hypertrophic CardioMyopathy Registry (HCMR) is currently recruiting, will further our understanding in this field, as the aim is to establish novel predictors of outcome in HC using CMR.

The limitations of this study are the small numbers of patients and that not all measurements were recorded using echocardiography. In addition, the left atrial dimensions were measured from echocardiography, but these might have changed when using CMR measurements, which might have further changed the ESC SCD score. Clinically in our institution, echocardiography is performed by a sonographer who measures LV wall thickness and acquires images for further reference; clinicians will verify or repeat the LV wall thickness measurements. This is in comparison to CMR where all the data is saved and is analysed and reported twice by at least two clinicians (although for this study, echocardiography measurements were measured by clinicians). It is likely that this is a similar set up to other hospitals in the United Kingdom, but these results may not applicable worldwide due to differing availability of CMR and differing levels of experience and expertise. Moreover, this study was not designed to address follow up, as current studies such as HCMR are actively recruiting and designed to address these outcomes.

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Figure Legends

Figure 1: Cardiac Magnetic Resonance images (A) and echocardiography (B) from the same patient showing difficulties visualizing accurately left ventricular myocardium

Figure 2: Maximum left ventricular wall thickness (mm) measurements using echocardiography and Cardiac Magnetic Resonance

Figure 3: Bland Altman Plot to show difference between echocardiography and Cardiac Magnetic Resonance for maximum left ventricular wall thickness measurements

Figure 4: Reproducibility of maximum left ventricular wall thickness measurements. Bland Altman Plot for LV wall thickness measurements measured by Cardiac Magnetic Resonance (4A) and echocardiography (4B)

Figure 5: Difference in Sudden Cardiac Death risk stratification classification using echocardiography and Cardiac Magnetic Resonance

Table 1

Table 1: Patients demographics (n=50)

Variable	Patients
Average age ± SD (years)	57 ± 16
Female	13 (26%)
Ethnicity: White	34 (68%)
Ethnicity: Asian	5 (10%)
Ethnicity: Black	11 (22%)
Family history sudden cardiac death (SCD)	7 (14%)
Unexplained syncope	4 (8%)
Non Sustained Ventricular Tachycardia (NSVT)	13 (26%)

Table 2

Table 2: Left Ventricular (LV) wall thickness measurements from Cardiac Magnetic Resonance (CMR) and echocardiography. Measurements of LV in mm ± SD

Location of wall thickness	LV wall thickness CMR	LV wall thickness echocardiography	P value	Number of Patients recorded CMR	Number of Patients recorded echocardiography
Total number of segments				600 (100%)	414 (69%)
Basal anterior	10.0 ± 4	15.4 ± 6	<0.0001	50 (100%)	44 (88%)
Basal septal	14.9 ± 4	16.5 ± 4	0.03	50 (100%)	48 (96%)
Basal inferior	8.2 ± 3	14.3 ± 4	<0.0001	50 (100%)	46 (92%)
Basal lateral	7.7 ± 3	12.1 ± 3	<0.0001	50 (100%)	45 (90%)
Mid anterior	9.6 ± 4	14.9 ± 5	<0.0001	50 (100%)	40 (80%)
Mid septal	13.5 ± 4	16.9 ± 5	0.0007	50 (100%)	42 (84%)
Mid inferior	10.1 ± 3	14.1 ± 4	<0.0001	50 (100%)	41 (82%)
Mid lateral	8.1 ± 3	12.2 ± 3	<0.0001	50 (100%)	39 (78%)
Apical anterior	9.0 ± 4	14.4 ± 5	<0.0001	50 (100%)	17 (34%)
Apical septal	8.4 ± 3	17.0 ± 6	<0.0001	50 (100%)	18 (36%)
Apical inferior	8.5 ± 3	13.2 ± 4	<0.0001	50 (100%)	17 (34%)
Apical lateral	8.9 ± 4	12.4 ± 3	0.0025	50 (100%)	17 (34%)

Table 3

Table 3: Number of patients (%) with maximum Left Ventricle (LV) wall thickness location by Cardiac Magnetic Resonance (CMR) and echocardiography.

Location of Wall Thickness	By CMR	By echocardiography
Basal anterior segment	4 (8%)	8 (16%)
Basal septal segment	25 (50%)	11 (22%)
Basal anterior/septal segments	*29 (58%)*	*19 (38%)*
Basal inferior segment	1 (2%)	3 (6%)
Basal lateral segment	-	1 (2%)
Mid anterior segment	2 (4%)	5 (10%)
Mid septal segment	14 (28%)	9 (18%)
Mid anterior/septal segments	*16 (32%)*	*14 (28%)*
Mid inferior segment	-	3 (6%)
Mid lateral segment	-	3 (6%)
Apical anterior segment	2 (4%)	3 (6)%
Apical septal segment	1 (2%)	4 (8%)
Apical anterior/septal segments	*3 (6%)*	*7 (14%)*
Apical inferior segment	1 (2%)	1 (2%)
Apical lateral segment	-	-

Figure 1

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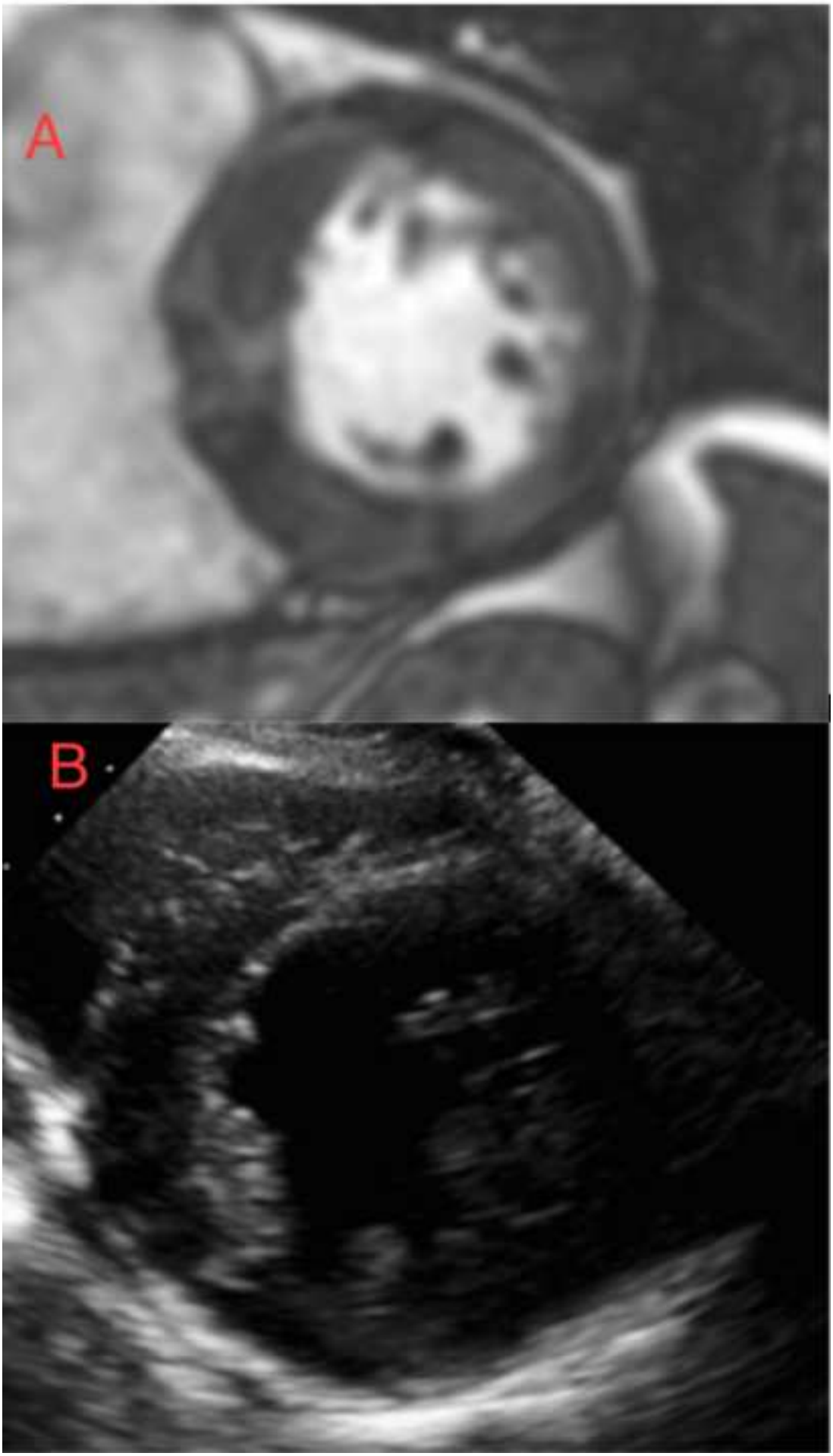

Figure 2

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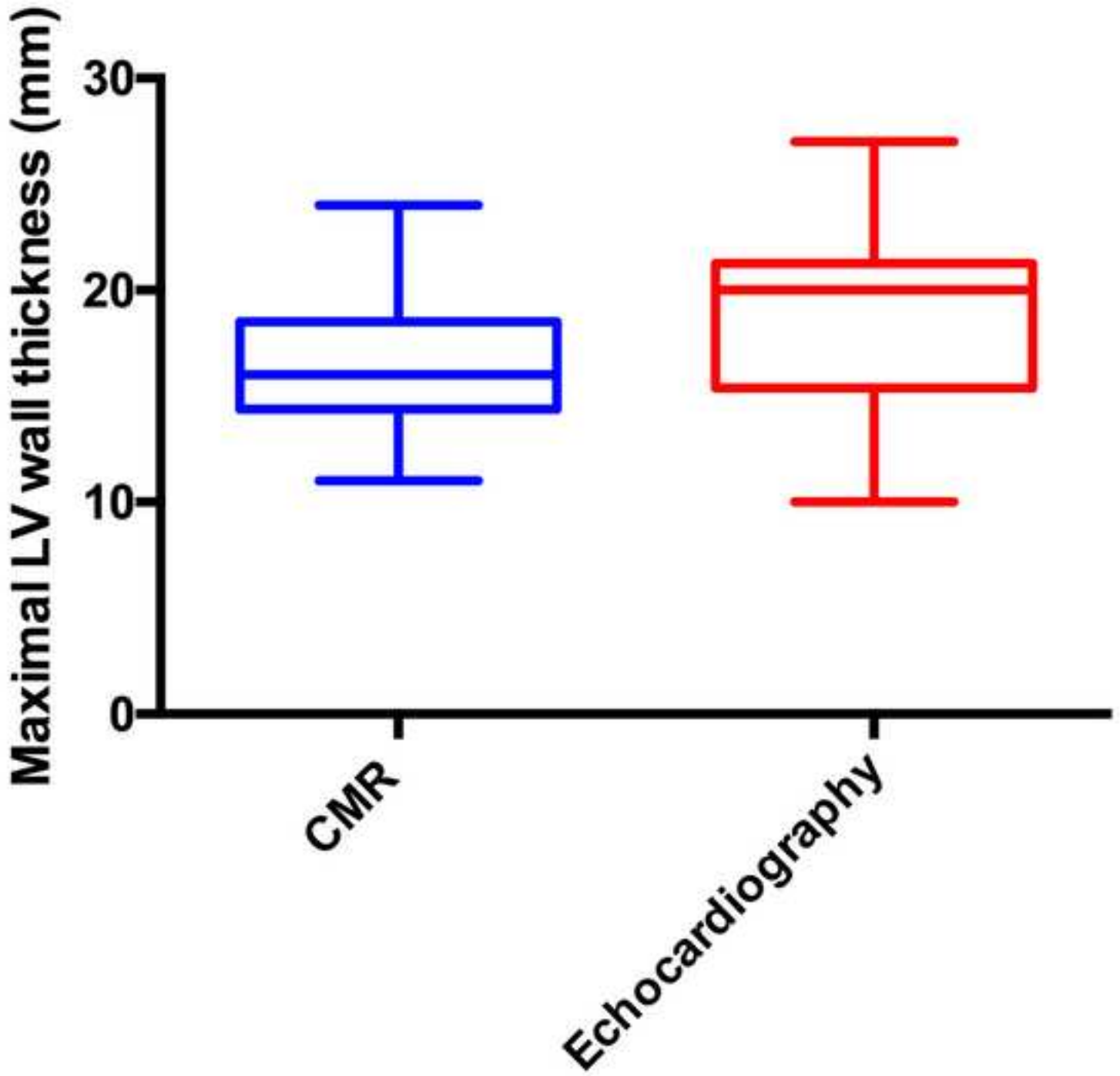

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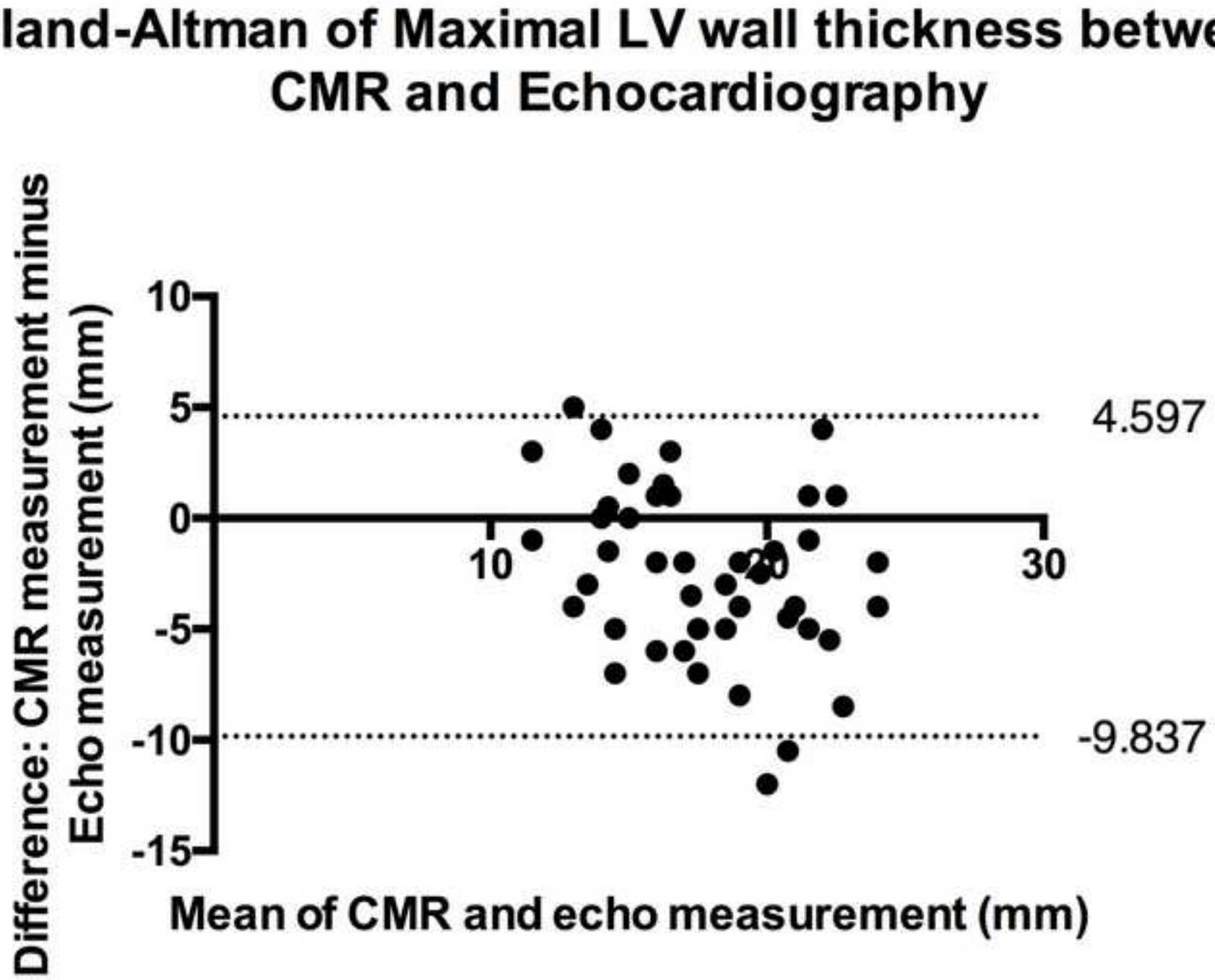

Figure 4A

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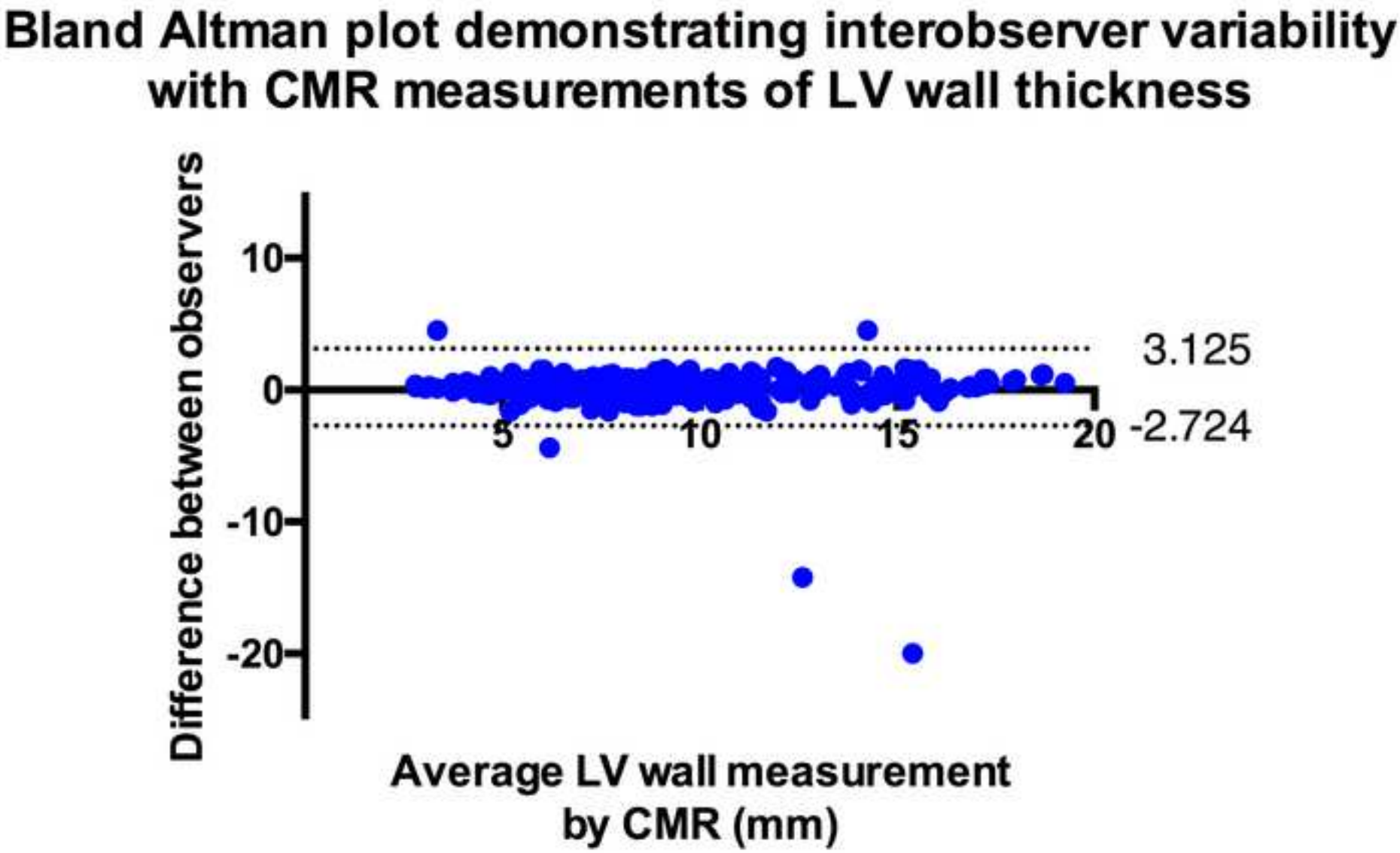

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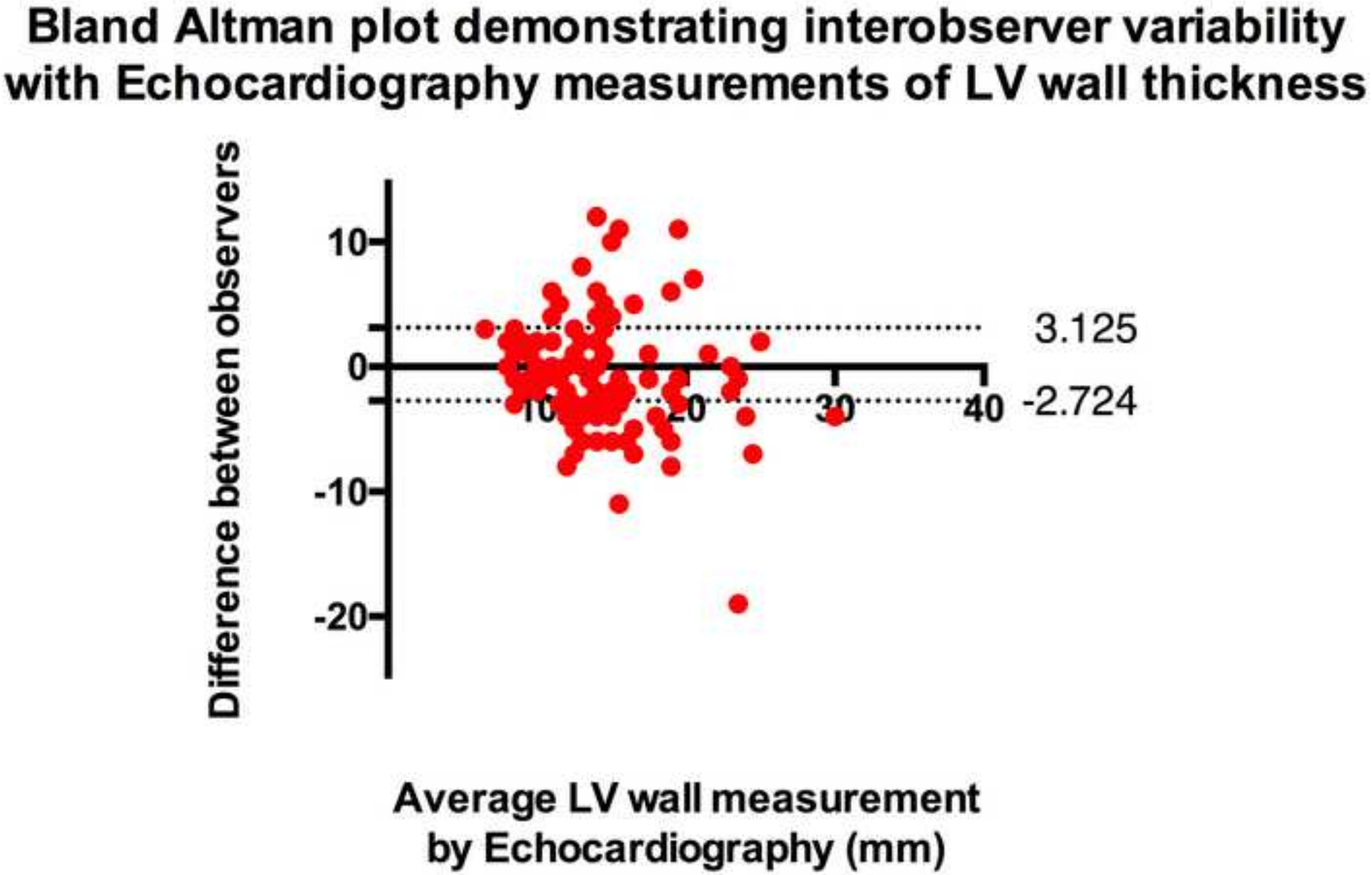

Figure 5

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