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Original Article

Left myocardial wall measurements on postmortem imaging compared to autopsy

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ABSTRACT

Purpose: The aims of this study were, firstly, to determine the relationship of left ventricular wall thickness (LVWT) measurements between postmortem computed tomography (PMCT) and postmortem magnetic resonance imaging (PMMR) and, secondly, to assess the utility of postmortem imaging for LVWT measurements compared to autopsy.
Materials and methods: All cases ≥18 years old, with postmortem interval ≤4 days, cardiac PMCT, PMMR, and full forensic autopsy, were reviewed in our database retrospectively. Exclusion criteria were gas accumulations in the myocardial wall and cardiac trauma. LVWT on PMCT and PMMR was assessed. The measurements were repeated by the same rater after 2 months. Autopsy reports were reviewed, and LVWT and pericardial fluid volume measured at autopsy were noted. Pericardial fluid volume >50 ml was determined positive for pericardial effusion.
Results: A total of 113 cases were included in the study. Twelve cases had pericardial effusion. Intrarater reliability for imaging based LVWT was excellent. LVWT (free wall) was significantly larger on PMCT (18.3 mm) compared to PMMR (17.6 mm), but these measurements correlated positively. LVWT (anterior wall) was significantly larger on PMMR (15 mm) than at autopsy (14 mm), and these measurements also correlated positively. Pericardial effusions led to larger differences between PMMR and autopsy measurements, however without statistical significance.
Discussion: There exist discrepancies between LVWT as measured on postmortem imaging and at autopsy. Specialists should be aware in order to not misinterpret imaging measurements.

1. Introduction

Natural causes of death, especially of cardiac etiology, are often encountered in the forensic pathologists' routine [1–5]. Cardiac hypertrophy is considered as the increase of heart weight and/or myocardial thickness or dilative changes of the heart [6]. Left ventricular hypertrophy may be associated with severe cardiac pathology and high risk of sudden death [4,7–8]. Thus, knowledge of underlying cardiac pathology is important in the forensic field, and cardiac assessment is an essential part of the forensic autopsy [7,9–10]. During the examination, heart weight, the heart valves' lengths, and myocardial wall thicknesses are determined. The measurements then may be compared with normal ranges' tables, taking into consideration the sex, body weight, height, body mass index, or the body surface area [11–16].

Postmortem imaging has gained an important role in the forensic routine, adding information to traditional autopsies or revealing relevant findings when autopsy is not conducted [17–19]. Postmortem computed tomography (PMCT) is used as a routine scan tool, while postmortem magnetic resonance imaging (PMMR) offers better visualization of the soft tissues and higher resolution for determining cardiac pathologies [20–29].

Winklhofer et al. [9] investigated the cardiothoracic ratio (CTR) on PMCT of 170 cases and stated that CTR threshold of 0.57 can identify cardiomegaly with high specificity. According to Jotterand et al. [30], CTR on PMCT is useful for determining cardiomegaly, whereas ventricular dilatation and body mass index may lead to increased CTR, based on a sample of 109 cases.

Hatch et al. [31] and Ruder et al. [4] showed that calculated heart weight based on the left ventricular circumferential area on PMCT of 50 [31] and PMMR of 53 cases [4], respectively, reflects the actual heart weight measured at autopsy. According to Ogawa et al. [32], in a sample of 33 cases, heart weight at autopsy could be accurately predicted by the left ventricular volume estimated on PMCT with the assumption that the left ventricle is an ellipse [32].

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Ampanozi et al. [33] compared the ventricular wall thicknesses and heart valves' PMMR measurements with the correspondent autopsy measurements in 25 cases and showed that PMMR and autopsy measurements correlate but that there are significant differences between them. Troxler et al. [34] compared postmortem computed tomography–angiography (PMCTA) and autopsy heart measurements of 50 cases and showed that PMCTA is not accurate to estimate autopsy measurements for cardiac wall thicknesses. Okuma et al. [27] compared the left ventricular wall thickness between PMCT and pathologoanatomical specimens of the heart of 57 cases and showed that PMCT measurements were significantly larger.

Postmortem cardiac imaging can accurately deliver useful information about cardiac disease. However, it seems unclear as to which frame of reference to use, seeing as if conceptual differences between the existing autopsy-based statistics and new postmortem-imaging-derived values become relevant. It can be expected that imaging-based measurements generally are useful within a framework of postmortem morphology that examines causes of death. In general, measurements of organ volumes, masses, or weights based on imaging positively correlate with ex vivo weights or histopathology masses according to previous studies [4,24,31,35–37].

Regarding cardiac measurements related to the diagnosis of hypertrophy or ventricular dilation in postmortem scanning, it is noteworthy that diverse cardiac pathologies, like infarction, edema, and scarring of the myocardial tissue, or compression caused by contiguous structures and related pathologies, like pericardial effusions, may affect the in situ radiological measurements and lead to misinterpretation and false-positive diagnoses of cardiac hypertrophy [30–31,33,38–39].

The aims of the current study were, firstly, to compare the measurements of the left ventricular wall thickness (LVWT) of the same subjects between PMCT and PMMR, and secondly, to investigate if there exist discrepancies between radiological and autopsy LVWT measurements in general and also with regard to presence or absence of pericardial effusion.

2. Materials and methods

2.1. Subjects

All consecutive cases with age $\geq$18 years and postmortem interval $\leq$4 days based on the estimation of time of death [1–3] that underwent preautopsy cardiac imaging (both PMCT and PMMR) and full forensic autopsy from January 1, 2014, to September 3, 2018, were reviewed in our database retrospectively. During radiological data evaluation, cases with moderate/extended gas accumulations in the myocardium according to Egger et al. [40] and/or cardiac trauma were excluded. Autopsy reports were reviewed and gender, age, LVWT (in mm), and the pericardial fluid volume (in ml) measured at autopsy were noted. According to protocol in our institute, LVWT is measured at the left ventricular outflow tract, about 1 cm below the aortic valve, after applying gentle stabilizing finger pressure and excluding any possibly present trabecular structures. From that, it is clear that no exclusion of papillary muscle for a true LVWT measurement is necessary at autopsy. Gender distribution, time intervals between PMCT and autopsy, PMMR and autopsy, and PMCT and PMMR examinations and LVWT measurements at autopsy were investigated for the whole sample.

2.2. Imaging protocol

PMCT was performed on a 128-slice scanner (SOMATOM Definition Flash, Siemens Healthineers, Erlangen, Germany), with the bodies in a supine position, using automatic dose modulation (CARE Dose 4D, Siemens Healthineers, Erlangen, Germany). Imaging parameters were [41] as follows: tube voltage 120 kVp and slice collimation 128$\times$0.6 mm. PMCT image reconstructions of thorax and abdomen were performed [41], with a slice thickness of 1.0 mm and an increment of 0.6 mm.

Cardiac PMMR imaging was performed on a 3-T MR scanner (Achieva 3.0 TX, Philips Healthcare, Best, the Netherlands). All images used for this study were acquired with turbo spin echo T2-weighted sequences. Short-axis images were acquired with a repetition time (TR) of 3860–4620 ms, echo time (TE) of 100 ms, and a slice thickness of 3.0 mm. Four-chamber views were acquired with a TR of 6170 ms, TE of 100 ms, and a slice thickness of 3.0 mm.

LVWT was radiologically measured by one forensic pathology trainee with 2 years' experience in forensic radiology at the time of image reviewing by usage of Syngo.via imaging software for multimodality reading (Syngo.Via, Siemens Healthineers, Erlangen, Germany). For comparing imaging and autopsy measurements, the *anterior* left ventricular wall was measured once in the whole sample in mm on PMMR short-axis views (T2-weighted turbo spin echo sequence) at the outflow tract approximately 1 cm below the aortic valve (Fig. 1). This level was chosen for better comparison with the autopsy measurements. The observer was blinded to the autopsy measurements. For comparing PMCT with PMMR, the *free* left ventricular wall was measured in mm on both PMCT and PMMR four-chamber views [42] (PMCT in soft tissue kernel and PMMR in T2-weighted turbo spin echo sequence). Free wall was chosen for this purpose considering the relatively low resolution of PMCT compared

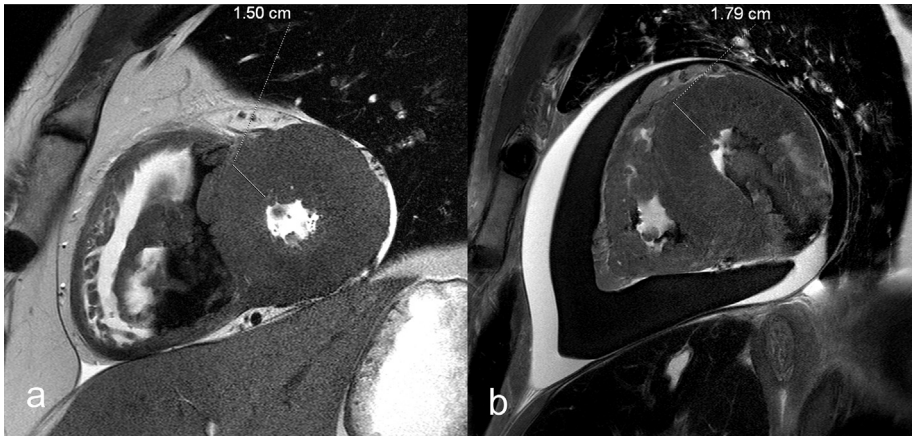

Fig. 1. Anterior left ventricular wall thickness measurements on PMMR for comparison with autopsy measurements.
Measurements of the anterior LVWT at the outflow tract in short-axis views on PMMR T2-weighted turbo echo sequence of a case without (a) and a case with (b) pericardial effusion.

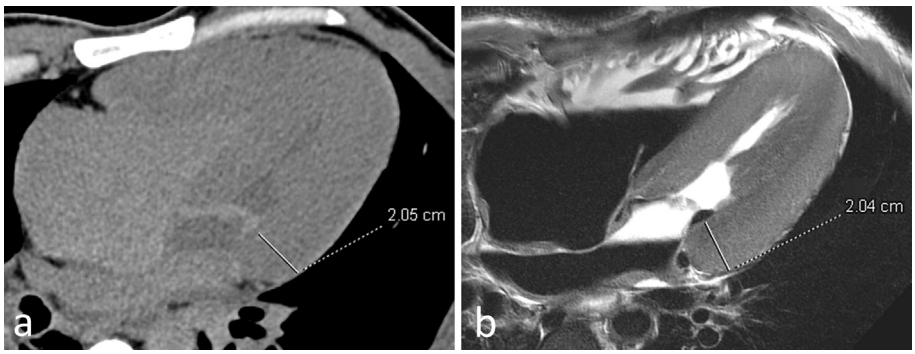

Fig. 2. Left ventricular free wall thickness measurements in postmortem imaging for comparison between PMCT and PMMR.
Four-chamber views of the same heart on PMCT soft tissue kernel (a) and PMMR T2-weighted turbo spin echo sequence (b). Measurements of the left ventricular wall thickness (free wall).

to the PMMR for differentiating and measuring the anterior wall in short-axis views. Four-chamber views according to Lu et al. [42] were applied for both PMCT and PMMR in order to avoid discrepancies caused by different planes. All measurements of the free wall were performed at the level of the mitral valve's largest diameter and approximately 1 cm distal (apical) to the mitral valve (Figs. 2 and 3). The level of 1 cm beneath the mitral valve was determined for better reproducibility of the study and because myocardial wall normally is thickest at the cardiac base and becomes thinner towards the apex [11,43]. Papillary muscles and epicardial fat were excluded from all radiological measurements. PMCT review took place first, and PMMR review took place 2 days later for the whole sample. The rater was blinded to the autopsy measurements during PMCT reviewing and to both the autopsy measurements and the PMCT measurements during PMMR reviewing. To increase accuracy, PMCT and PMMR measurements were repeated by the same observer in the same manner after a 2-month period [44–46]. During the repeat review, the case list was randomly reordered, and the observer was blinded to the measurements obtained by the first review. The intrarater agreement was determined, and the mean of both measurements (first and second review) of the LVWT on PMCT and on PMMR was calculated for every case. The mean LVWTs on PMCT and on PMMR for the whole sample were obtained [mean (M)±standard deviation (SD)] for statistical analysis.

2.3. Relationship between LVWT on PMCT and LVWT on PMMR (free wall)

Mean free LVWT on PMCT and mean free LVWT on PMMR were compared, and the correlation between them was investigated.

2.4. Pericardial effusion (anterior wall)

Cases with pericardial fluid volume >50 ml according to the autopsy reports were determined as positive for presence of pericardial effusion. Descriptive statistics were presented as absolute

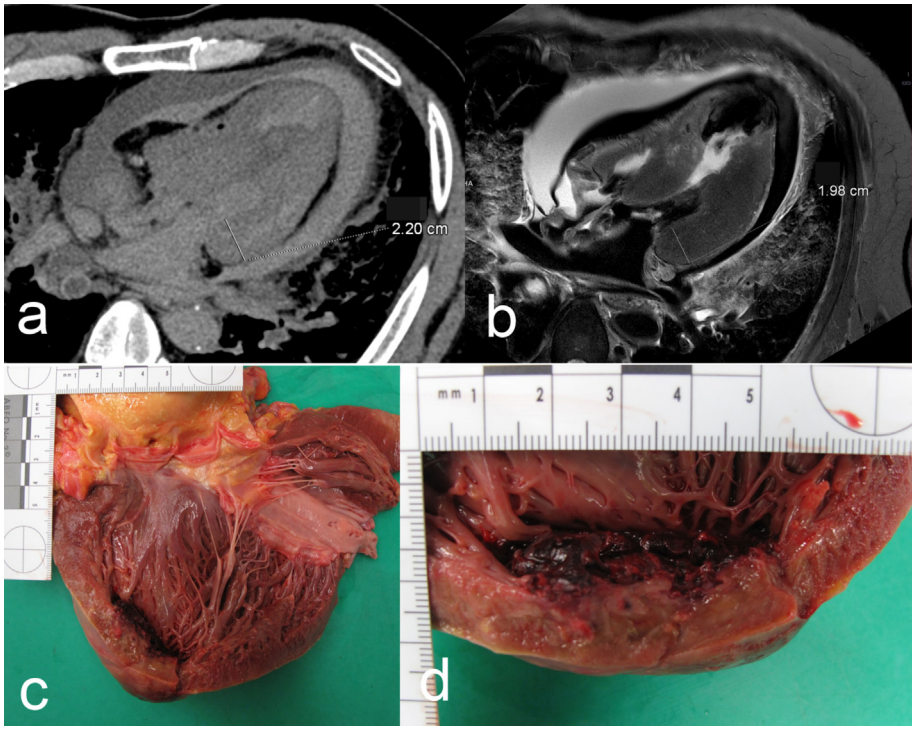

Fig. 3. Left ventricular free wall thickness measurements in postmortem imaging of a case with pericardial effusion for comparison between PMCT and PMMR.
Four-chamber views of the same heart on PMCT soft tissue kernel (a) and PMMR T2-weighted turbo spin echo sequence (b) for the measurements of the left ventricular wall. Note the presence of hemopericardium with the typical target sign revealed around the heart. Pericardial blood volume was 530 ml and anterior left ventricular wall thickness 13 mm at autopsy. Note also the changes of the wall at the apex of the heart indicative of cardiac wall rupture, which was confirmed during autopsy (c, d).

Table 1
Left ventricular wall thickness measurements of the whole sample (N=113)

	1st review				2nd review				Average			
LVWT	*M*	SD	Md	IQR	*M*	SD	Md	IQR	*M*	SD	Md	IQR
PMCT, free wall (mm)	**18.2**	**2.7**	18	4	**18.3**	**2.8**	18	3.8	**18.3**	**2.7**	17.8	4
PMMR, free wall (mm)	**17.6**	**2.9**	17.8	4.2	17.6	2.9	**18.1**	**3.8**	**17.6**	**2.9**	17.9	4
PMMR, anterior wall (mm)	**-**	**-**	-	-	-	-	**-**	**-**	15.1	2.2	**15**	**3**
Autopsy, anterior wall (mm)	-	-	-	-	-	-	-	-	14	2.2	**14**	**2**

Overview of the LVWT measurements (in mm) on PMCT, on PMMR, and at autopsy in the whole sample (N=113). *M* and SD are highlighted for the normally distributed (Shapiro–Wilk tests: P>.05), and Md and the IQR are highlighted for the not normally distributed values (Shapiro–Wilk test: P<.05).

Table 2
Left ventricular wall thickness measurements of the cases with pericardial effusion (N=12)

	1st review				2nd review				Average			
LVWT	*M*	SD	Md	IQR	*M*	SD	Md	IQR	*M*	SD	Md	IQR
PMCT, free wall (mm)	**18.9**	**2.6**	19.5	4.9	**18.9**	**2.8**	19	5.6	**18.9**	**2.7**	19.3	5.3
PMMR, free wall (mm)	18.5	3.2	**19.8**	**4.7**	18.5	3.1	**19.6**	**5**	18.5	3.2	**19.7**	**4.9**
PMMR, anterior wall (mm)	-	-	**-**	**-**	-	-	**-**	**-**	**15.3**	**2.6**	16	6
Autopsy, anterior wall (mm)	-	-	-	-	-	-	-	-	**13.3**	**2**	13	4

Overview of the LVWT measurements (in mm) on PMCT, on PMMR, and at autopsy in the cases with pericardial effusion (N=12). M and SD are highlighted for the normally distributed (Shapiro–Wilk tests: P>.05), and Md and IQR are highlighted for the not normally distributed values (Shapiro–Wilk test: P<.05).

numbers and percentages for the incidence of pericardial effusion in the sample. The difference of the *anterior* LVWT measured on PMMR and the LVWT at autopsy ($diff_{PMMR\text{-}aut}$ = LVWT on PMMR − LVWT at autopsy) was calculated for every case. This difference ($diff_{PMMR\text{-}aut}$) was compared between the cases with and without pericardial effusion.

2.5. Relationship of LVWT on PMMR and LVWT at autopsy (anterior wall)

All cases with pericardial effusion were excluded for this part of the study. *Anterior* LVWT on PMMR was compared to LVWT measured at autopsy, and the correlation between them was investigated. The correlation was expressed as linear regression with equation for the estimation of the LVWT based on PMMR. Calculated LVWT based on the regression equation and LVWT measured at autopsy were compared.

2.6. Statistical analysis

Statistical analysis was performed with the SPSS Statistical Package (IBM, SPSS 20, Chicago, IL, USA). Shapiro–Wilk tests were used for assessing normality of distributions. Levene's test was used for exploring the heterogeneity of variances between groups. Descriptive statistics were presented for the nominal variables as absolute numbers and percentages, for the continuous variables that were normally distributed as *M* and SD, and for the continuous variables that were not normally distributed as median (Md) and interquartile range (IQR). Intraclass correlation coefficient (ICC) estimates and their 95% confident intervals (CIs) were calculated for assessing intrarater reliability based on two-way mixed-effects model, a mean rating (k=2), and absolute agreement for intrarater reliability [47]. Paired-samples t tests were used to compare free LVWT on PMCT with free LVWT on PMMR, and its nonparametric alternative, Wilcoxon signed-rank test, was used to compare anterior LVWT on PMMR with the LVWT measured at autopsy. Pearson's correlation coefficient and its nonparametric alternative, Spearman Rho test, were used for exploring correlations for normally and not normally distributed variables, respectively. Mann–Whitney U test (nonparametric independent-samples t test) was used to compare $diff_{PMMR\text{-}aut}$ (anterior LVWT on PMMR − LVWT at autopsy) between the cases with and without pericardial effusion. A multiple regression model was used for the estimation of LVWT measured at autopsy based on the PMMR measurements. Wilcoxon signed-rank test was also used for comparing the calculated LVWT based on the regression equation with the actual LVWT measured at autopsy. P values less than 0.05 indicated statistical significance. Excel (Microsoft Excel, 2010, Microsoft Corporation, Redmond, WA, USA) was used for creating the graphs of this study.

3. Results

3.1. Subjects

One hundred eighteen cases were identified. During imaging evaluation, five cases were excluded because of gas and/or cardiac trauma in imaging. Thus, 113 cases (age: 51±17 years) were included in the study. Ninety concerned males (79.6%, age: 49±15 years) and 23 females (20.4%, age: 60±22 years). PMCT–autopsy time interval was 15±11 h, PMMR–autopsy time interval was 11±13 h and PMCT–PMMR absolute interval was 5±11 h (in only 4 cases PMMR scan was conducted prior to PMCT). For these 113 cases, median LVWT measured at autopsy was 14 mm (IQR: 2 mm) (Table 1).

3.2. Imaging

The results obtained by the two reviews for the measurements of the free wall are presented on Table 1. Intrarater reliability was

Table 3
Anterior wall measurement differences between PMMR and autopsy for the whole sample, for the cases with and the cases without pericardial effusion

	$diff_{PMMR\text{-}aut}$ = LVWT on PMMR − LVWT at autopsy (mm)			
Pericardial effusion	*M*	SD	Md	IQR
Present + absent (N=113)	1	2	**1**	**2**
Present (>50 ml) (N=12)	**1.9**	**2.5**	2	4
Absent (≤50 ml) (N=101)	1	1.9	**1**	**2**

Overview of the differences of the anterior LVWT on PMMR and the anterior LVWT measured at autopsy ($diff_{PMMR\text{-}aut}$) in the whole sample independent of presence of pericardial effusion (N=113), and for the cases with (pericardial fluid volume >50 ml, N=12) and the cases without pericardial effusion (pericardial fluid volume ≤50 ml, N=101). There were no statistically significant differences of the $diff_{PMMR\text{-}aut}$ between the two pericardial effusion groups (Mann–Whitney U test not significant: P=.1).

Table 4
Left ventricular wall thickness measurements of the cases without pericardial effusion (N=101)

	1st review				2nd review				Average			
LVWT	*M*	SD	Md	IQR	*M*	SD	Md	IQR	*M*	SD	Md	IQR
PMCT, free wall (mm)	**18.1**	**2.7**	17.7	3.8	**18.3**	**2.8**	17.9	3.6	**18.2**	**2.7**	17.7	3.7
PMMR, free wall (mm)	**17.4**	**2.9**	17.6	3.9	17.6	2.9	**17.5**	**3.7**	**17.5**	**2.9**	17.7	3.8
PMMR, anterior wall (mm)	-	-	-	-	-	-	-	-	15.1	2.2	**15**	**3**
Autopsy, anterior wall (mm)	-	-	-	-	-	-	-	-	14.1	2.2	**14**	**3**

Overview of the LVWT measurements (in mm) on PMCT, on PMMR, and at autopsy in the cases without pericardial effusion (N=101). M and SD are highlighted for the normally distributed (Shapiro–Wilk tests: P>.05), and Md and IQR are highlighted for the not normally distributed values (Shapiro–Wilk test: P<.05).

excellent for both modalities (PMCT: ICC=0.97, 95% CI=0.96–0.98, P<.001. PMMR: ICC=0.98, 95% CI=0.97–0.99, P<.001) [47]. In 42 cases (37.2%), free LVWT on PMCT was smaller than PMMR; in 69 (61%), it was larger; and in 2 (1.8%) cases, measurements were equal. Mean free LVWT was 18.3±2.7 mm on PMCT and 17.6±2.9 mm on PMMR. Median anterior LVWT on PMMR was 15 mm (IQR: 3 mm).

3.3. Relationship between LVWT on PMCT and LVWT on PMMR (free wall)

In all 113 cases (containing the cases both with and without pericardial effusion), statistically, *free* LVWT on PMCT was significantly larger than on PMMR (paired-samples t test: P=.001, mean absolute difference: 1.6±2.1 mm, 95% CI=0.28–1.07) with large effect size (Eta squared: 0.09). There was strong positive correlation between LVWT on PMCT and PMMR (Pearson correlation: r=0.72, P<.001).

3.4. Role of pericardial effusion (anterior wall)

Twelve cases (10.6%) had pericardial effusion (pericardial fluid volume: 284±200 ml, minimum: 70 ml, maximum: 600 ml). An overview of the LVWT measurements for the cases with pericardial effusion is given in Table 2. Median difference of the *anterior* wall measured on PMMR versus the wall measured at autopsy ($diff_{PMMR-aut}$) was 2 mm for the cases with pericardial effusion and 1 mm for the cases without pericardial effusion (Mann–Whitney U test: P=.1) (Table 3).

3.5. Relationship of LVWT on PMMR and LVWT measured at autopsy (anterior wall)

Cases with pericardial effusion (pericardial fluid >50 ml, N=12) were excluded from the following analyses, as presence of pericardial effusion led to larger (however, not statistically significant) differences between LVWT on PMMR and LVWT at autopsy.

For the 101 cases without effusion, mean free LVWT on PMCT was 18.2±2.7 mm, on PMMR: 17.5±2.9 mm, median anterior wall on PMMR: 15 mm (IQR: 3 mm) and median LVWT at autopsy: 14 mm (IQR: 3 mm) (Table 4).

In 17 cases (16.8%), *anterior* LVWT was smaller on PMMR than at autopsy. In 19 cases (18.8%), anterior LVWTs on PMMR and at autopsy were equal, and in the rest, 65 (64.4%) anterior LVWT measurements were larger on PMMR compared to autopsy. *Anterior* LVWT on PMMR was significantly larger than the LVWT measured at autopsy (Wilcoxon signed-rank test: P<.001). *Anterior* LVWT decreased from PMMR (15 mm) to autopsy (14 mm) (Table 4). $diff_{PMMR-aut}$ for the *anterior* wall measurements was 1 mm (Table 3, Fig. 4). Anterior LVWT on PMMR was strongly and positively correlated with the LVWT at autopsy (Spearman's Rho for anterior wall on PMMR and at autopsy: r=0.63, P<.001). The linear regression for this relationship is described by the following equation:

LVWT at autopsy (mm) = 0.6×LVWT on PMMR+4.8 (R^2=0.4, P<.001) (Fig. 5).

There were no significant differences between calculated LVWT based on the regression equation and LVWT at autopsy (Wilcoxon signed-rank test: P=.14). The mean absolute difference between LVWT at autopsy and calculated LVWT was 1.4 mm. A

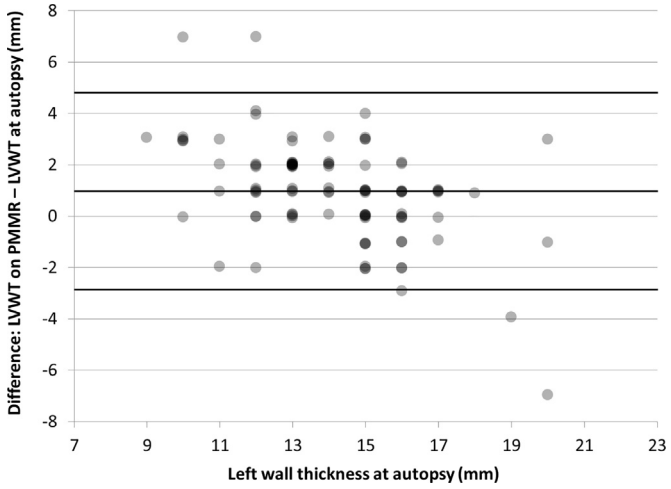

Fig. 4. Relationship between PMMR and autopsy measurements of the anterior LVWT. Bland–Altman plots depicting the difference of the anterior LVWT obtained by the two different methods (y-axis: LVWT on PMMR – LVWT at autopsy difference) versus the LVWT measurements at autopsy (golden standard, x-axis) of the 101 cases without pericardial effusion.

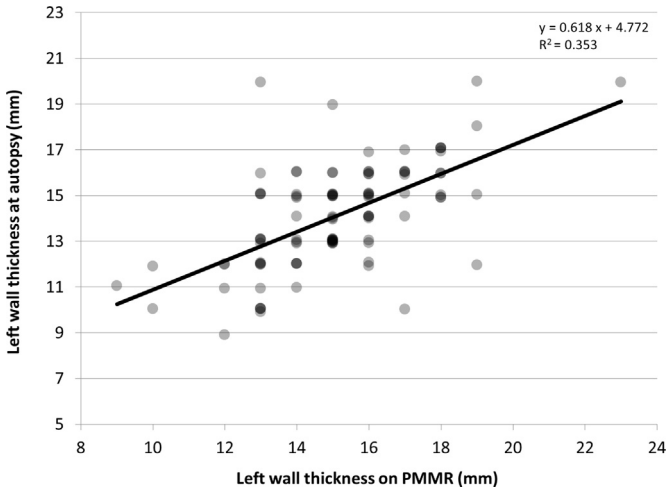

Fig. 5. Relationship between PMMR and autopsy measurements of the anterior LVWT. Graph of anterior LVWT on PMMR (x-axis) versus LVWT at autopsy (y-axis) and linear regression calculated from the data points. The linear regression line is indicated by the black line. All cases with pericardial effusion (>50 ml, N=12) are excluded.

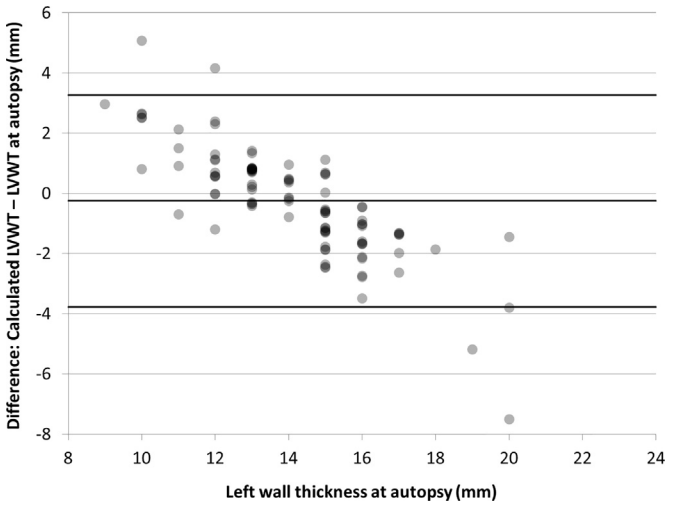

Fig. 6. Relationship between calculated LVWT based on PMMR and LVWT autopsy measurements.
Bland–Altman plot depicting LVWT at autopsy (*x*-axis) versus the calculated LVWT based on the regression equation - LVWT at autopsy difference (*y*-axis). All cases with pericardial effusion (>50 ml, *N*=12) are excluded. Small LVWTs are overestimated and large LVWTs are underestimated based on the regression equation.

Bland–Altman plot showed that the regression equation overestimates smaller LVWTs and underestimates larger LVWTs (Fig. 6).

4. Discussion

This study was based on a relatively large sample and showed that LVWT measurements significantly differ between PMCT and PMMR, as well as between PMMR and autopsy, but they exhibit positive correlations. LVWT measurements are larger on PMCT than on PMMR and on PMMR than at autopsy.

In this study, LVWT was measured on PMMR in two different locations: at the anterior wall on short-axis views for comparison with the autopsy measurements and at the free wall on four-chamber views for the comparisons with the PMCT of the same subjects. Anterior LVWT was approximately 15 mm for the whole sample, whereas free wall almost reached 18 mm. These results, even though obtained by different PMMR planes (short-axis vs. four-chamber views), are compatible with previous studies referring to left wall thickness in living [48–49] and deceased subjects [27] indicating that intrasubject variability of the left wall thickness exists.

Median LVWT at autopsy was 14 mm; this value is comparable to the LVWT autopsy measurements of the 25 cases reviewed by Ampanozi et al. (14.04±2.13 mm) [33] and of the 50 cases by Troxler et al. (13.8±2.8 mm) [34] and to the pathologoanatomical measurements of 57 autopsy hearts measured by Okuma et al. (13.9±3 mm) [27]. Mean LVWT on PMCT was 18.27±2.73 mm, which was compatible with the results by Okuma et al. (19.3±3.5 mm) [27]. LVWT on PMMR was 17.59±2.89 mm, a value comparable to the results of Ampanozi et al. (18.23±3.03 mm) [33].

PMCT and PMMR measurements of the free wall were significantly different though conducted at the same position and level. The relatively low resolution, contrast, and soft tissue density of PMCT, compared to those of PMMR that comprises the golden standard for cardiac examinations, are believed to be the main cause of this discrepancy [7,25,27,29]. Different modalities and imaging parameters may have had also an influence; however, four-chamber views were applied for both modalities. Kanza et al. compared LVWT measurements between multidetector-row CT and cine-MRI in 19 living subjects and revealed significant differences; however, larger values were observed for MRI probably because of the thicker MRI slice thickness [48]. PMMR measurements were larger than PMCT for the 37.2% of our sample as for the 61% LVWT on PMCT was larger than on PMMR. In our study, despite the also significant differences, there was strong correlation between PMCT and PMMR measurements, and this result is compatible with that of Kanza et al. [50].

Pericardial effusion was expected to affect the radiological measurements, as pericardial effusion and pericardial tamponade take up volume within the mostly nonelastic pericardial sac. This will invariably cause mostly concentric cardiac compression with consequential change of the heart shape and subsequently a pseudohypertrophy of the cardiac walls when measured in situ by echocardiography [38–39]. In this study, presence of pericardial effusion led to a certain overestimation of the anterior LVWT (approximately 1 mm), but this result was not statistically significant. This observation may be consistent with the assumption that increased in situ cardiac compression is reflected with pseudohypertrophy of the cardiac walls in situ [38–39], which may be released during autopsy as the pericardial sack is opened and the effusion is cleared away. To the best of our knowledge, this is the first attempt to investigate LVWT on postmortem imaging with distinction of the presence of pericardial effusion. However, the number of the cases with pericardial effusion in the current sample was small, and a larger number of cases with pericardial effusion may have led to more prominent differences and statistically significantly different values.

For some cases, LVWT was smaller on PMMR compared to autopsy. This may be explained by the data review processing and possible errors, case-dependent variables, data acquisition parameters, and imaging resolution. Additionally, Jotterand et al. suggested that dilatation of the heart due to perimortem acute cardiac failure may result in an overestimation of the postmortem cardiothoracic ratio [30]. Within the same concept, perimortem cardiac dilation may result in a pseudohypotrophy of the cardiac walls in situ.

Anterior LVWT was measured significantly larger on PMMR compared to autopsy. However, the measurements were correlated. These observations are consistent with the results of Ampanozi et al., who revealed significantly larger LVWT on PMMR than at autopsy in 25 cases after exclusion of cases with hemopericardium, however also with strong correlation [33]. Additionally, according to Okuma et al., PMCT measurements of the left cardiac wall (19.3±3.5 mm mm) were also significantly larger than the measurements of the pathologoanatomical specimens of the same 57 hearts (13.9±3 mm) after excluding the papillary muscles and the epicardial fat tissue [27].

Even in the absence of pathology or trauma, measurements of the myocardium differ significantly between imaging and autopsy. In situ, the heart is filled with blood and is mechanically compounded by the surrounding mediastinal structures, the diaphragm, and the pericardial sac. Thus, the heart has to appear slightly smaller and more compact, exposing thicker wall diameters in imaging than at autopsy, where the surrounding structures, including pericardial effusions, are removed, the cardiac cavities are dissected, the blood with its clots are cleared away, and the heart is manually stretched, causing the walls to appear more flattened. In addition, the exact location of measurement and any amount of gentle finger pressure applied to elicit what pathologists assume to be a true reflection of left myocardial thickness do not appear to be standardized. The overall process of arriving at LVWT at autopsy therefore appears to be both fundamentally and technically different from any in situ measurement due to various reasons [27,51–53].

After death, the heart may assume a postmortem middle position, halfway between full dilation and contraction. While the

diagnosis of hypertrophy during autopsy may be based on wall thickness and heart weight, diagnosing dilation, particularly when hypertrophy is present, does not seem quite as straightforward. A rounded shape or increased spherical appearance of the left chamber as well as a flabby consistency is described as criteria, but also, a slightly lower wall thickness than assumedly present given a certain heart weight may indicate dilation [54].

Stiffening of the cardiac musculature may play an additional role for discrepancies causing cardiac pseudohypertrophy. Under the assumption that a considerable number of the hearts may have already been affected by rigor mortis at the time of PMMR scan, the interval between PMMR scan and autopsy may have allowed partial or complete resolution of the rigor, causing the cardiac walls to appear larger at autopsy again [27,51–53]. The cadavers were preserved in low ambient temperatures, which also affect the rigor process. However, this comprises only an assumption; it neither can be proven nor was a purpose of the current study.

With that, imaging does not lack precision. The intrarater agreement for PMCT and PMMR was high, indicating very precise measurements. However, there exist discrepancies between imaging and autopsy LVWT measurements because of the different nature of the different methods. Thus, it is essential to not compare imaging measurements with the normal reference values proposed for autopsy for the assessment of cardiac hypertrophy. Normal ranges' tables for radiological postmortem measurements of the heart have to be established for an accurate postmortem radiological diagnosis of ventricular hypertrophy and dilation. For that, the in situ factors impacting the heart shape need to be considered. This is challenging in that a compressed heart may not easily reveal its ventricular geometry in situ as seen at autopsy. Conversely, the precision of autopsy LVWT measurements seems to be also technically constrained in a rather subjective aspect (gentle finger pressure).

This study has limitations. Firstly, there may exist selection bias considering that the bodies that underwent PMMR were mostly cardiac deaths; however, the aim of the current study was to evaluate possible presence of significant discrepancies between imaging and autopsy in general, independently of cardiac pathology and cause of death. Secondly, pericardial effusion was the only pathology investigated for having a potential effect on the LVWT measurements in situ in only a small number of cases. Thirdly, the measurements were conducted by one rater; however intrarater agreement was assessed for the measurements of the free wall on both PMCT and PMMR, and the authors believe additional raters would not lead to statistically significant discrepancies as simple measurement under the guidance of a specific study protocol is a relatively objective task [30–31]. Fourthly, autopsy measurements as recorded in the reports are rounded up, which may have led to more profound discrepancies. Last but not least, the R^2 value of the regression model was relatively low, indicating lack of precise predictions. Technically, precision at the level of mm may be considered difficult to be achieved under such variability.

5. Conclusion

Left wall thickness is measured significantly larger on PMCT compared to PMMR. Measurements of the left ventricular wall thickness on PMMR are, on average, significantly larger than at autopsy, with positive correlation. These discrepancies may become more prominent by the presence of pericardial effusion. It is of main importance to not compare the imaging measurements with the normal reference values proposed for autopsy for the assessment of cardiac hypertrophy. Normal ranges' tables for the diagnosis of cardiac hypertrophy and dilation based on postmortem imaging need to be established.

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Compliance with ethical standards

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Conflicts of interest

The authors declare no conflicts of interest.

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