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An exploratory study using left main and right coronary artery diameter and heart mass assessed on post-mortem computed tomography to identify ischaemic heart disease

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ABSTRACT

Coronary artery calcification is used to identify ischaemic heart disease (IHD) on post-mortem computed tomography (PMCT), but with inherent limitations. Using an alternative approach, this study explored coronary artery diameters and estimated heart mass to identify IHD on PMCT. A 6-year retrospective study was performed comparing the sum of the left main coronary artery and right coronary artery diameters to the power of four/heart mass (CAA – HM ratio) between control (92 subjects) and IHD (63 subjects) using PMCT. In control, only age was associated/correlated with the CAA – HM ratio, whereas in the IHD, no factors were associated/correlated with the CAA – HM ratio. A subsequent case-control study selecting subjects aged 50–75 (control: 32 subjects, IHD: 34 subjects) showed the CAA – HM ratio was significantly higher in IHD (t-test, $p < 0.05$). The area under the curve in the plotted receiver operating characteristics was 0.847 (specificity: 0.719, sensitivity of 0.853), with the optimal cut-off ratio being 0.202 (10^{-3} mm^4/g). This study showed that the CAA – HM ratio was higher and could differentiate IHD with reasonable accuracy from control aged 50–75 years. It may serve as an adjunct in identifying IHD on PMCT. Further validation and subsequent studies comparing and incorporating other PMCT parameters for IHD are recommended.

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Postmortem imaging; myocardial ischaemia; coronary artery disease; ischaemic heart disease

Introduction

Ischaemic heart disease (IHD) is commonly caused by coronary artery atherosclerosis [1]. It is the leading cause of death in adults and a common natural cause of death encountered in forensic pathology, especially chronic IHD [2]. Modern forensic pathology has integrated post-mortem computed tomography (PMCT) into routine practice as an adjunct to traditional post-mortem examination[3]. Currently, IHD can be identified indirectly using PMCT by assessing coronary arteries for calcification, a common feature

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of atherosclerosis[4]; however, this method only assesses the pathology of coronary artery atherosclerosis and does not necessarily consider the pathophysiology of IHD. In chronic IHD, the coronary artery increases in calibre to compensate for reduced perfusion from atherosclerotic narrowing or increased heart demand from cardiac remodelling or hypertrophy [5]. This exploratory study investigates whether assessing coronary artery diameters (left main and right coronary arteries to the power of four) and estimated heart mass using PMCT can differentiate IHD from normal/control.

Materials and methods

Case selection

The study was a 6-year retrospective observatory study (2019–2024) performed at a regional forensic pathology department in Australia, comparing the coronary artery diameter to heart mass ratio (measured and estimated) on PMCT between control and deaths from IHD. In all selected cases the age (years), sex (male or female), height (m), and weight (kg) were recorded from the post-mortem report. Adults aged >18 years were chosen for both groups because IHD is very rare among those aged <18 years. Only Caucasians were selected because of the potential confounding of race.

Control

The control subjects were deaths with clear non-natural and non-cardiac death, with the inclusion criteria being:

- Adults (aged >18 years) were selected as ischaemic heart disease seldom occurs in the paediatric age group.
- No evidence of IHD on medical record and PMCT scan (e.g. absence of significant coronary artery calcification).
- No major disruption of anatomy precluding exact PMCT and anthropometric measurements.
- Non-drug or alcohol-related deaths due to potential toxicological effects of these substances on the heart.

IHD

Adult (aged > 18 years) cases where the primary cause of death was IHD or coronary artery atherosclerosis in the same study period were selected. These deaths were thought to be due to chronic IHD, and the mechanism of death was thought to be due to fatal cardiac arrhythmia. Cases with acute plaque events were excluded because an acute plaque event resulting in acute coronary artery blockage would not enable sufficient time for the coronary artery to compensate and the heart to remodel.

Exclusion criteria

- All suspicious and homicide deaths due to potential legal implications.

- Cases with incomplete data set.
- Non-binary sex due to the small number of cases for analysis.
- Case showing decomposition or body disruption.
- Maternal deaths due to potential altered physiology.

Heart weight estimation, coronary artery diameter measurement on PMCT

All PMCT scans were performed using a 32-slice Siemens SOMATOM Scope (Siemens Healthineers, Erlangen, Germany), using 130 kV and 60mAs before the examination as standard practice. The PMCT scans were reconstructed using 'Siemens' syngo.via Frontier software package (Siemens Healthineers, Erlangen, Germany) then viewed, manipulated (Figure 1(a)), and measured using the standard in-built measurement tools (Figure 1(b,c)):

- On the coronial section, the crosshairs were moved to the base of the aortic valve, and the vertical crosshair was rotated to align with the apex of the heart.
- On the sagittal plane, the crosshair was moved to the aortic valve, and the vertical crosshair rotated and aligned with the apex.

All measurements (Left Ventricular (LV) width, LV depth, LV length, coronary artery diameters) were later done on the sagittal and transverse sections.

Heart mass estimation

The heart weight for all subjects was estimated using the heart volume generated by the PMCT using an ellipsoid model of the left ventricle [6]. The length of the left ventricle was measured on the sagittal plane, and the dimensions of the base of the left ventricle were measured on the transverse plane in centimetres (Figure 1(b)). The estimated heart mass (g) was calculated as $4/3 \times 3.14 \times \text{width}/2 \times \text{depth}/2 \times \text{length} \times 1.05$. The model proposed uses only the left ventricle volume and not the whole heart volume [6]. This was considered suitable for this study as the highest blood demand would be from the left ventricle. It is a quick and simple method (~10 sec) easily applied to daily routine forensic pathology practice.

Coronary artery diameter, and coronary artery to estimated heart mass ratio

The left main and right coronary artery external diameters were measured within 10 mm away from the ostia after the infundibulum tapers and before branching, and without calcification (Figure 1(c)); the proximal segment of the left anterior descending and left circumflex coronary artery diameter were not measured due to difficult visualization on non-contrast PMCT. Using Poiseuille's equation for closed pipe flow (incompressible viscous fluid), flow is proportional to the diameter/radius of the pipe to the power of four [7,8]. As such, the coronary artery to estimated heart mass ratio (CAA – HM ratio) was calculated (diameter left main^4 + diameter right coronary artery^4)/estimated heart mass, in $x10^{-3}$ mm^4/g).

The external coronary artery diameters were used instead of the internal diameter because routine PMCT in forensic pathology does not use contrast, and angiography in PMCT was not available at this centre. Coronary artery wall thickness is relatively uniform

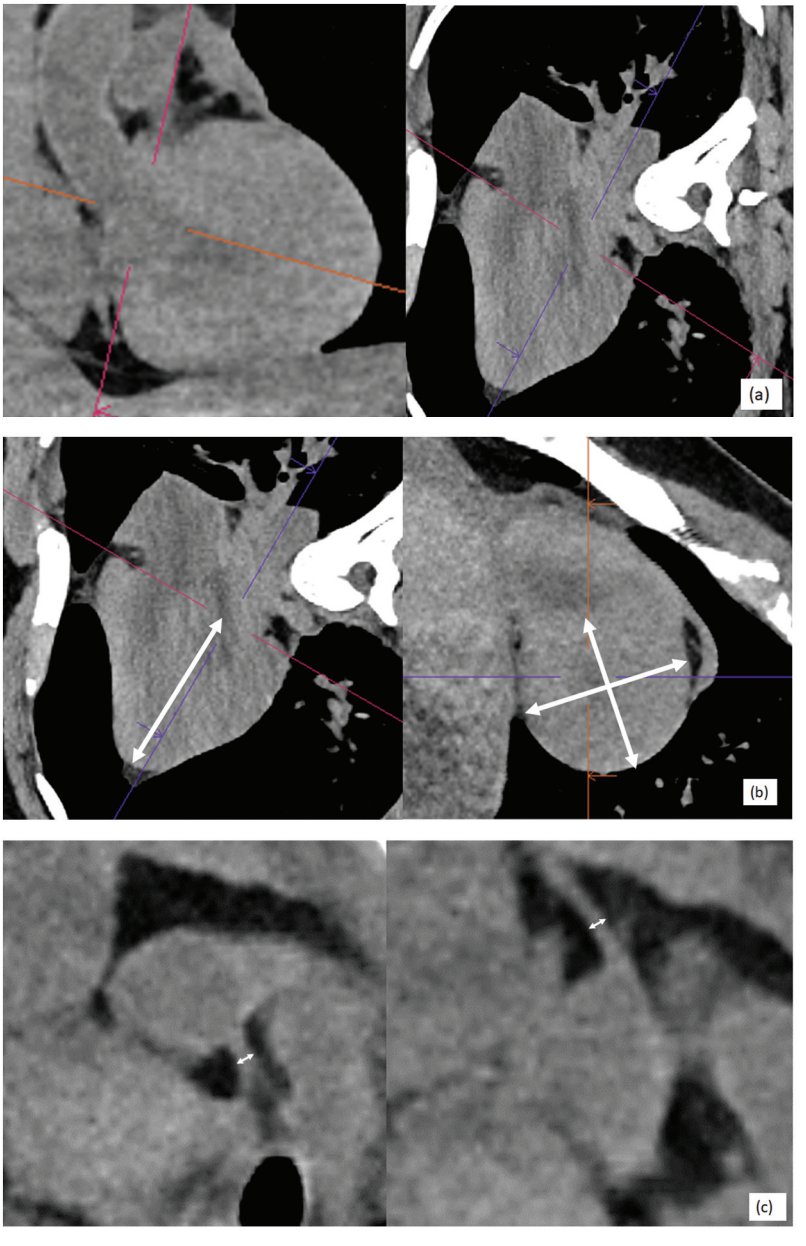

Figure 1. Representative PMCT images for (a) PMCT scan manipulation (details on step-by-step manipulation described in the text), (b) heart dimension measurement for heart mass estimation (arrowheads represent length on the left pane, and width and depth on the right) and (c) measurement of the diameter (arrowheads) of left (left pane) and right (right pane) coronary arteries.

in normal healthy adult individuals [9], as such, external coronary artery diameter can be a surrogate for internal diameters.

Statistical analysis

Statistical analysis was performed using IBM-Statistical Package for Social Science-29 (IBM SPSS Statistics 29, IBM Corporation, New York, United States of America) software and a p-value of <0.05 was considered significant. Continuous variables were described using means and standard deviations (SD). Medians, minimum, maximum, min and max, were also presented. Counts (percentages) were used to describe categorical variables. The Shapiro–Wilk test was used to check for normality. For each group, student t-tests and Pearson's correlation tests were used to investigate confounding factors associated with the CAA – HM ratio. Univariate and subsequent multivariate linear regression analysis was conducted to evaluate the relationship between the CAA – HM ratio and age, sex, height, and weight.

Student t-tests and chi-square tests were used to compare the differences between control and IHD. As the IHD group was shown to be significantly older and age was associated with the CAA – HM ratio in control, the potential confounding effect of age was controlled by selecting all subjects aged 50–75 years in establishing the best threshold to differentiate control and IHD. The best threshold was determined using receiver operation curve analysis (ROC) by choosing the cut point closest to the perfect predictor (sensitivity of '1' and false positive rate of '0'). The area under the curve (AUC, marker for accuracy) and the corresponding sensitivity and specificity for the threshold were determined.

Ethics approval

This study was approved by the Coronial and Public Health Sciences Human Ethics Committee (HEC 24–11, project title: Association between coronary artery diameter and heart weight on post-mortem computed tomography as an adjunct in assessing cardiac ischaemia in coronial casework).

Results

Control

A total of 92 subjects were identified and were exclusively deaths from neck compression. It had a mean age of 43.17 years (SD: 13.46, median: 45.50, min: 19, max: 75) and a male predominance [M: F = 50 (54%): 42 (46%)]. The mean weight and height were 72.84 kg (SD: 16.37, median: 71, min: 41, max: 112) and 1.69 m (SD: 0.10, median: 1.69, min: 1.40, max: 2.02) respectively. The mean left main coronary artery diameter, right coronary artery diameter, and estimated heart mass were 3.7 mm (SD: 0.8, median: 3.6, min: 2.0, max: 5.4), 3.3 mm (SD: 0.8, median: 3.1, min: 1.8, max: 5.8), and 251.92 g (SD: 70.45, median: 252.07, min: 132.42, max: 448.76) respectively. The mean CAA – HM ratio was 0.15×10^{-3} mm^4/g (SD: 0.10×10^{-3}, median: 0.12×10^{-3}, min: 0.02×10^{-3}, max: 0.45×10^{-3}).

The CAA – HM ratio was associated positively and significantly with age (Pearson's R – value = 0.426, $p < 0.05$), but not with height and weight ($p > 0.05$) (Table 1). There was no significant difference in the CAA – HM ratio between sexes (t-test, $p > 0.05$). Univariate and subsequent multivariate analysis between the CAA – HM ratio with sex, age, height, and weight showed only age is positively associated with the CAA-HM ratio significantly ($p < 0.05$; Table 2).

Table 1. Summary of the control only showing the correlation between CAA-HM ratio with age; and the IHD group showing no association/correlation between any demographic and anthropometric factors.

Variables	Test	Coefficient	*p*-value
Normal (Control)			
Age (Years)	Pearson's correlation	0.426	<0.05
Height (m)	Pearson's correlation	−0.166	>0.05
Weight (kg)	Pearson's correlation	−0.115	>0.05
Sex	t-test		>0.05
IHD (Case)	**Test**	**Coefficient**	***p*-value**
Age (Years)	Pearson's correlation	−0.063	>0.05
Height (m)	Pearson's correlation	0.116	>0.05
Weight (kg)	Pearson's correlation	0.010	>0.05
Sex	t-test		>0.05

Table 2. Univariate and multivariate linear regression analyses of the CAA-HM ratio with various independent factors among control and IHD showing that only age was associated/correlated with the CAA-HM ratio in control.

Univariate Linear Regression				Multivariate Linear Regression		
Dependent Variables	Coefficient (95%CI)	*p*-value	R^2	Coefficient (95%CI)	*p*-value	R^2
Control						
Age (years)	0.03 (0.002, 0.004)	<0.001	0.182	0.003 (0.002, 0.004)	<0.001	0.192
Sex	0.023 (−0.18, 0.063)	0.273	0.013	0.015 (−0.037, 0.066)	0.576	
Weight (kg)	0.001 (0.000, 0.002)	0.199	0.018	0.001 (−0.001, 0.002)	0.495	
Height (m)	0.059 (−0.155, 0.273)	0.586	0.003	−0.001 (−0.004, 0.002)	0.530	
IHD						
Age (years)	−0.001 (−0.004, 0.002)	0.571	0.005	−0.001 (−0.004, 0.003)	0.754	0.019
Sex	0.013 (−0.113, 0.140)	0.833	0.001	0.016 (−0.153, 0.184)	0.853	
Weight (kg)	0.001 (−0.001, 0.003)	0.350	0.014	0.001(−0.001, 0.003)	0.386	
Height (m)	0.026 (−0.408, 0.461)	0.903	0.000	−0.131 (−0.755, 0.492	0.675	

Ischaemic heart disease

A total of 63 cases were identified with a mean age of 71 years (SD: 13.19, median: 73, min: 36, max: 98) and a male predominance (M: F = 54:9). The mean weight and height were 86.66 kg (SD: 21.75, median: 85.40, min: 37.20, max: 127.40) and 1.71 m (SD: 0.10, median: 1.73, min: 1.48, max: 1.90) respectively. The mean left main coronary artery diameter, right coronary artery diameter, and estimated heart mass were 4.1 mm (SD: 0.7, median: 4.2, min: 2.7, max: 5.8), 4.9 mm (SD: 0.6, median: 4.9, min: 3.1, max: 6.3), and 269.58 g (SD: 42.77, median: 263.14, min: 183.95, max: 380.24) respectively. The mean CAA – HM ratio was 0.36×10^{-3} mm^4/g (SD: 0.16×10^{-3}, median: 0.34×10^{-3}, min: 0.07×10^{-3}, max: 0.95×10^{-3}).

The CAA – HM ratio was not associated with age, height, and weight ($p > 0.05$). No significant difference was found in the CAA – HM ratio between sexes (t-test, $p > 0.05$) (Table 1). Univariate and subsequent multivariate analyses of the CAA – HM ratio with sex, age, height, and weight had no significant coefficients (Table 2).

CAA-HM ratio between IHD and control

Comparing the two groups, the IHD was significantly older in age and heavier in body weight (t-test, $p < 0.05$) and had a higher proportion of males (Chi-square, $p < 0.05$). The

Table 3. Summary of different anthropometric variables showing that age, sex, weight, and the CAA-HM ratio are statistically significantly different between the IHD and Control groups in the study.

Variables	All Cases ($n = 155$)	IHD ($n = 63$)	Control ($n = 92$)	p-Value
	Mean (SD) Median (Min, Max)	Mean (SD) Median (Min, Max)	Mean (SD) Median (Min, Max)	
Age (years)	54.48 (19.11) 54.00 (19, 98)	71.00 (13.19) 73.00 (36, 98)	43.17 (13.46) 45.50 (19, 75)	<0.001
Sex n (%)	M:104 (67%) F:51 (33%)	M:54 (86%) F:9 (14%)	M:50 (54%) F:42 (46%)	<0.001
Weight (kg)	79.48 (22.36) 76.60 (37.20, 179.00)	89.18 (26.23) 86.00 (37.20, 179.00)	72.84 (16.37) 71.00 (41.00, 112.00)	<0.001
Height (m)	1.69 (1.34) 1.70 (0.73, 2.02)	1.69 (1.77) 1.72 (0.73, 1.90)	1.69 (0.6) 1.69 (1.40, 2.02)	0.864
CAA-HM ratio (10^{-3} mm^4/g)	0.24 (0.17) 0.19 (0.02, 0.95)	0.36 (0.17) 0.34 (0.07, 0.95)	0.15 (0.10) 0.12 (0.02, 0.45)	<0.001

CAA – HM ratio was significantly higher in the IHD (t-test, $p < 0.05$); otherwise, there was no difference in height (Table 3).

In the selected group aged 50–75 years, the CAA – HM ratio was significantly higher in the IHD (IHD: 0.35×10^{-3} mm^4/g, SD:0.17; Control: 0.18×10^{-3} mm^4/g, SD:0.09) (t-test, $p < 0.05$). The plotted ROC curve showed an AUC of 0.847 (Figure 2), and the optimised threshold was 0.202×10^{-3} mm^4/g with sensitivity of 0.853 and specificity of 0.719.

Discussion

This study explored whether the CAA – HM ratio can differentiate control and IHD. In control, the CAA – HM ratio was a robust parameter that only associated/correlated positively and significantly with age but not with other anthropometric and demographic factors. In the IHD, the CAA – HM ratio did not show any correlation or association with any anthropometric and demographic factors. CAA – HM ratio was significantly higher in IHD, before and after being controlled for age (50–75 years old).; It has an acceptable diagnostic accuracy (AUC = 0.847 in ROC), and a threshold of 0.202×10^{-3} mm^4/g yielded a reasonable sensitivity and specificity in differentiating IHD (0.719–0.853).

Coronary artery atherosclerosis causes over 90% of IHD[1], which is the most common natural cause of death encountered in forensic pathology, commonly presenting as chronic IHD resulting in sudden cardiac death[2]. Post-mortem examination of the heart in these deaths commonly shows coronary artery narrowing from atherosclerosis and cardiac hypertrophy (or old myocardial infarction) [10]. In modern forensic pathology practice, PMCT can be used to aid in diagnosing IHD to reduce the need (and cost) for internal post-mortem examination for these deaths [11–13]. Coronary artery calcification, a pathological feature of coronary artery atherosclerosis, is widely used to assist in identifying IHD in PMCT[4]; however, atherosclerosis may not calcify (i.e. soft atherosclerotic plaque) or may calcify but not result in occlusion/ischaemia, limiting its diagnostic ability[14]. This study used the CAA – HM ratio to assess the pathophysiological changes in IHD rather than the pathology of coronary artery atherosclerosis.

Cardiac ischaemia is due to insufficient blood supply to meet the demand from the heart muscle [15]. Generally, the blood supply to the heart depends on the coronary artery diameter, and the blood demand depends on the heart mass [16]. Coronary arteries can

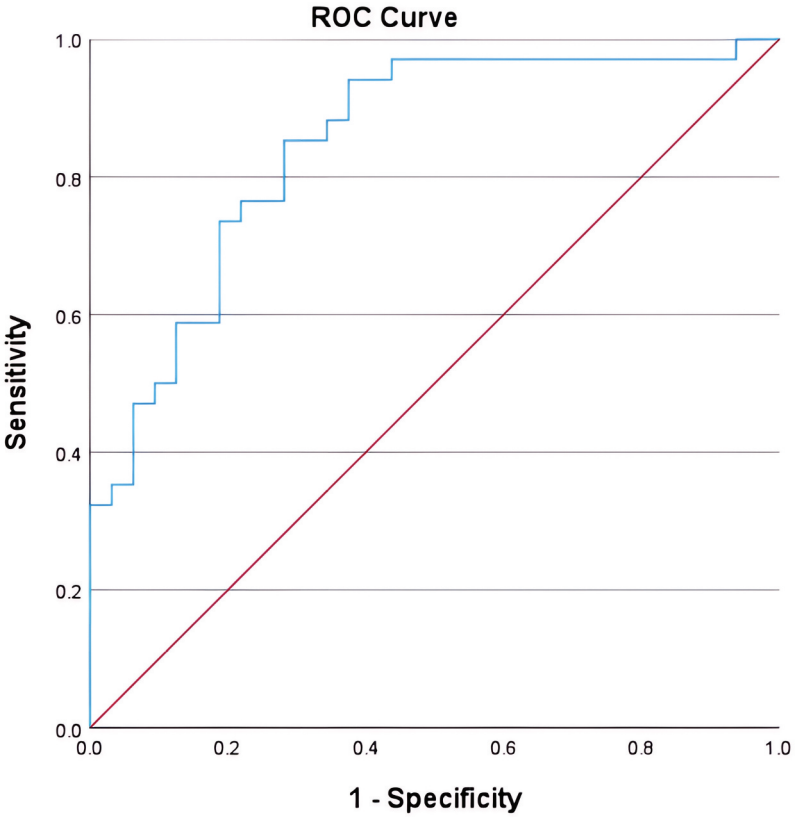

Figure 2. Receiver operating characteristic curve for CAA – HM ratio (the sum of the diameters of left main coronary artery^4 and right coronary artery^4/heart mass) between control and IHD with reasonable diagnostic ability.

compensate by increasing their diameter, to a certain extent, to enable increased demand from the heart in pathological or physiological states [5]. In chronic IHD, the coronary arteries may compensate disproportionally by increasing their diameter (upstream from the atherosclerotic narrowing) to maintain adequate perfusion to the diseased heart [17,18]. As such, it is assumed that there would be a normal ratio between coronary artery diameter and heart mass. This ratio would differ significantly in IHD, with the coronary artery diameter increasing disproportionally compared to the increased heart mass.

CAA-HM ratio characteristics in control and IHD

This study used the CAA – HM ratio, calculated by the sum of the left main and right coronary artery diameter to the power of four divided by heart mass, to assess IHD on PMCT. As the anatomy of the normal heart, particularly heart mass and coronary artery diameter is associated with demographic and anthropometric factors [19–21], the CAA – HM ratio was shown to be a robust parameter and independent of all demographic and anthropometric factors, apart from age.

In control, apart from age, the CAA – HM ratio was a robust parameter in terms of anthropometrics and demographics. The association/correlation between the CAA – HM ratio and age can be explained by the physiological changes in the ageing heart, such as myocardial hypertrophy, arterial stiffness, and endothelial dysfunction occur physiologically, which requires more blood perfusion [22–24]. Contrary to the control, the CAA – HM ratio in the IHD was independent of all anthropometric and demographic factors, even with age. The explanation for this difference may include the pathophysiological changes in IHD being more pronounced on the CAA-HM ratio than age and the study sample of this group being exclusively aged >50 years.

Comparison of CAA-HM ratio between control and IHD

With the association between age and CAA – HM ratio in control, a case-controlled comparison between the control and IHD was made to remove the confounding effects of age, comparing the CAA – HM ratio between control and IHD in the group aged 50–75 years. This comparison showed that the CAA – HM ratio was significantly higher in IHD. The increase in the CAA – HM ratio in the IHD can be explained by the coronary artery compensating disproportionately to perfuse the heart due to downstream coronary artery atherosclerosis narrowing in IHD[18]. In addition, it is also possible that with atherosclerosis and subsequent loss of compliance, the coronary artery may recoil less than normal after death [25,26]. Moreover, the increase of the coronary arteries diameter could be simply due to haemodynamic reasons: a vascular compensatory wall over-distention which is related to increased flow resistances induced by the impairment of collective diameter of the coronary tree in case of non-calcified coronary atherosclerosis or minimally calcified coronary arteries or calcified but non-occlusive appearing coronary arteries.

The overall diagnostic ability of the CAA – HM ratio to differentiate IHD and control was reasonable with a relatively high AUC on the plotted ROC curve, but strictly limited to the age range of 5–70 years. The reasons for only having a reasonable diagnostic ability may include the nature of the CAA – HM ratio, the post-mortem nature of the study, and the study design. The CAA – HM ratio was used as a parameter to assess the relationship between blood supply/flow and demand of the heart. Apart from coronary artery diameter, the blood supply/flow to the heart also depends on blood pressure and viscosity, and flow resistance of the coronary artery system (i.e. collective diameter, length, compliance, branching and collateral formations) [7,8], which cannot be assessed on routine PMCT and was therefore not examined in this study. Other reasons may include post-mortem artefacts on the heart affecting its dimensions. Although more sophisticated analysis and scanning modality may increase the accuracy, the CAA – HM ratio is simple to obtain on plain PMCT for routine forensic pathology practice and using a crude method to assess coronary artery dimensions and heart mass, and the rationale of this stdtyu was to use a quick and easy method without any specialized equipment. Despite this, the results of this exploratory study show potential utility in using the CAA – HM ratio as an adjunct in assessing IHD in PMCT.

The potential application may be in cases with non-convincing PMCT findings of IHD, such as cases with suspected non-calcified coronary artery atherosclerosis, minimally calcified coronary arteries, or calcified but non-occlusive appearing coronary arteries.

Limitations

This study was designed as an exploratory study performed in a regional forensic pathology department. This study only focused on one parameter in assessing a subgroup of IHD deaths (i.e. chronic IHD). Other features of IHD deaths on PMCT, such as coronary artery calcification and lung oedema/congestion, were not assessed or compared with the CAA – HM ratio as well as a comparison to gross post-mortem examination was not done. Further validation is needed with inter and intra-observer analysis for repeatability to apply to future routine practice. The threshold established together with the diagnostic characteristics is only used for illustrative purposes in the post-mortem setting and may not be translatable to other clinical or forensic pathology departments with different population mixes and computed tomography scan devices and configurations.

Conclusion

This exploratory study demonstrated that the CAA – HM ratio, when controlled with age, is a potential parameter in identifying IHD. In addition to the current parameters, the CAA – HM ratio may serve as an adjunct in recognizing IHD on PMCT. Further validation and subsequent studies comparing and incorporating other PMCT parameters for IHD are recommended.

Disclosure statement

No potential conflict of interest was reported by the author(s).

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