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CT findings of type A acute aortic dissection that did and did not result in prehospital death

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Abstract

The differences between the pathologies of cases of type A acute aortic dissection (AAD) that did and did not result in prehospital death (PHD) have not been fully elucidated.

This study aimed to compare the CT findings and clarify the differences between the pathologies of such cases.

Ninety four consecutive type A AAD patients between 2010 and 2020 were enrolled in this study. There were 47 males and 47 females (mean age: 69.0 ± 14.4 years). The patients were divided into those that did (n = 25, 27%) and did not (n = 69, 73%) suffer PHD. We retrospectively evaluated the CT or postmortem CT findings of each case and analyzed the relationships between clinical factors (CT findings and clinical characteristics) and PHD using logistic regression analysis.

Bloody pericardial effusion (96% vs 35%, $P < .0001$), bloody pleural effusion (40% vs 1%, $P < .0001$), and mediastinal hematomas (88% vs 14%, $P < .0001$) were significantly more common in the PHD group than in the no PHD group.

In the multivariate logistic regression analysis, bloody pericardial effusion and lung consolidation were found to be significant risk factors for PHD (odds ratio: 21.29 [95% confidence intervals {CI}: 1.19–248.29] and 13.72 [95% CI: 1.79–105.06], respectively; $P = .014$ and $P = .012$, respectively). AD affecting the abdominal aorta was identified as a significant negative risk factor for PHD (odds ratio: 0.02 [95% CI: 0.01–0.65]; $P = .0042$).

Most PHD due to type A AAD are associated with hemorrhaging. Bleeding into the pericardium and type A AAD confined to the thoracic aorta are significant risk factors for PHD. Secondary respiratory failure might contribute to PHD in such cases.

Abbreviations: AAD = acute aortic dissection, CI = confidence intervals, CPR = cardiopulmonary resuscitation, OHCPA = out-of-hospital cardiopulmonary arrest, OR = odds ratios, PHD = prehospital death, PMCT = postmortem computed tomography.

Keywords: acute aortic dissection, complication, CT, sudden death, type A

1. Introduction

Acute aortic dissection (AAD) is a life-threatening condition, which requires immediate diagnosis and treatment. In particular, Stanford type A AAD often causes out-of-hospital cardiopulmonary arrest (OHCPA), and most of these cases result in prehospital death (PHD).[1] However, the pathological differences between cases of type A AAD that do and do not result in PHD have not been fully elucidated.

Recently, autopsy imaging methods, such as post-mortem computed tomography (PMCT), have become increasingly popular because of their accessibility, noninvasiveness, and potential for detecting causes of death.[1–3] They have also been reported to be useful for assessing sudden death due to AAD.

The purpose of this study was to compare the CT findings of type A AAD cases that did and did not result in PHD and clarify the pathological differences between such cases.

2. Methods

2.1. Patients

The ethics committee of our hospital approved this study, and the need to obtain informed consent was waived. We retrospectively evaluated the cases of patients who were urgently transported to our hospital between 2010 and 2020 and were confirmed to have suffered type A AAD by CT. Cases that satisfied the following 2 conditions were examined: The onset time of chest pain and/or loss of consciousness was clear;[1] CT was performed within 4 hours of the onset time.[2]

The CT diagnostic criteria for type A AAD were defined based on previous reports.[4–7] A visible intimal flap (Fig. 1), inward shifting of a calcified intima, and/or an intramural hematoma (Fig. 2) in the ascending aorta (type A) were adopted as diagnostic

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The datasets generated during and/or analyzed during the current study are not publicly available, but are available from the corresponding author on reasonable request.

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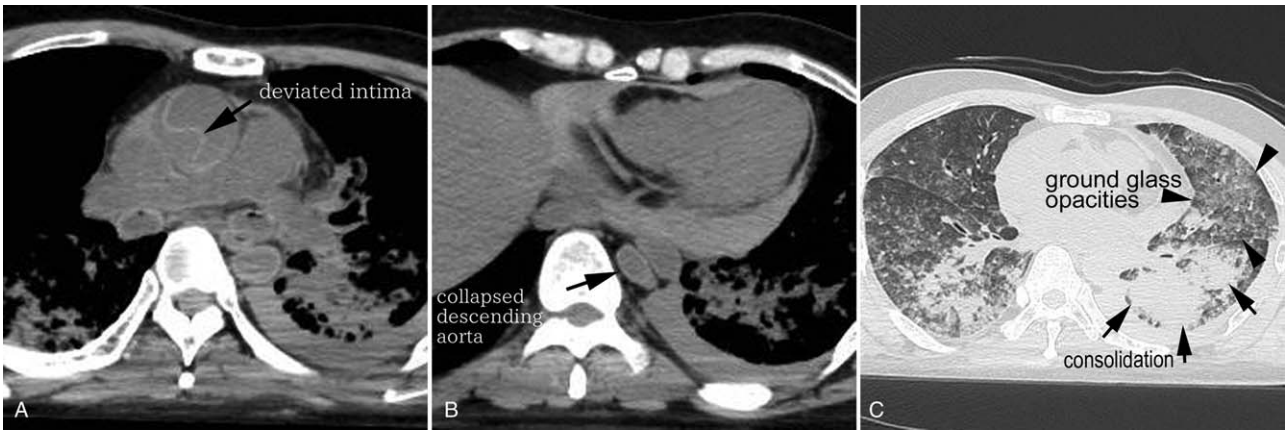

Figure 1. A 49-year-old male with type A AAD. A. PMCT image shows a deviated intima in the ascending aorta (arrow). The densities of the false lumen and true lumen are almost the same, indicating a type A aortic dissection with an open false lumen. There is no dissection of the descending aorta, and the dissection is confined to the ascending aorta. B. PMCT image showing bloody pericardial and left-sided bloody pleural effusion. The descending aorta has collapsed (arrow). C. PMCT image showing consolidation (arrows) and diffuse ground glass opacities (arrowheads) in the lung field.

criteria because these factors have been reported to be useful diagnostic findings in patients with AAD.[4–7]

Finally, 94 consecutive patients were enrolled in this study. There were 47 males and 47 females, who ranged in age from 36 to 101 years (mean age: 69.0 ± 14.4 years), and 87 patients (93%) had a history of hypertension. Two patients had Marfan syndrome.

In this study, OHCPA was defined as a state in which the patient was not conscious, was not breathing, and did not have a pulse at the time of the arrival of the emergency services. All patients that suffered OHCPA (n = 28) were subjected to cardiopulmonary resuscitation (CPR). PHD was defined as when spontaneous circulation was not observed from discovery until the declaration of death. Twenty five of 28 patients (89%) that suffered OHCPA progressed to PHD, and PMCT scans were performed in these cases.

On the other hand, CPR was not attempted in the patients that did not suffer OHCPA. Ordinary pre and postcontrast-enhanced CT scans were carried out in all of the cases that did not involve PHD.

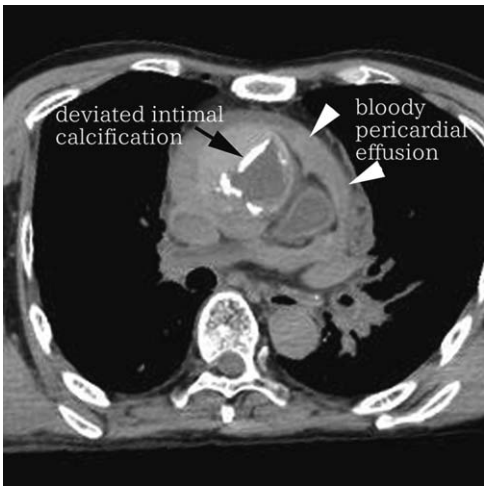

Figure 2. A 69-year-old male with type A AAD. PMCT images show deviated intimal calcification (arrow) and a high-density area within the enlarged ascending aortic wall. These findings are indicative of type A AAD with a closed false lumen. Bloody pericardial effusion can also be seen (arrowheads).

Finally, we divided the patients into 2 groups; that is, into those that did (n = 25, 27%) and did not (n = 69, 73%) suffer PHD. There were 2 cases of Marfan syndrome, 1 in each group.

2.2. Image acquisition protocol

In the PHD group, noncontrast-enhanced PMCT was performed after the declaration of death. The PMCT scans were conducted with a Somatom Definition or Definition Flash scanner (Siemens Medical Systems, Erlangen, Germany), generating axial images. The scanning parameters were as follows: tube voltage: 100 to 120 kVp, tube current: 140 to 200 mAs (effective). Reconstruction was performed using a 1-mm slice thickness and 1-mm intervals. All PMCT scans were carried out from the head to the pubic symphysis.

In the no PHD group, CT scans were conducted with and without contrast enhancement in all patients. The contrast-enhanced CT was carried out with nonionic contrast medium (300 mg/mL, iopromide; Bayer Schering, Berlin, Germany), which was delivered as a bolus injection (100 mL at 2–4 mL/sec) using a power injector (Dual Shot GX; Nemoto Kyorindo, Tokyo, Japan).

The CT scans were acquired with a Somatom Definition or Definition Flash scanner, generating axial images. The scans were commenced at 30 seconds and 120 seconds (2 phases) after the start of the injection of contrast medium. The scanning parameters were as follows: tube voltage: 100 to 120 kVp, tube current: 140 to 200 mAs (effective). Reconstruction was carried out using a 1-mm slice thickness and 1-mm intervals for both the unenhanced and contrast-enhanced images. Both the unenhanced and contrast-enhanced scans were performed from the head to the pubic symphysis. Electrocardiographic gating was not used.

2.3. Image evaluation

The CT data were evaluated by creating axial and longitudinal interactive multiplanar reformation images with a 1-mm slice thickness. The assessments of the CT findings were carried out independently by 2cardiovascular radiologists (E.S. and T.M.),

who had over 20 years of experience each, and particular attention was paid to the following items: (a) the presence/absence and location of AD; (b) the presence/absence of closed or open false lumens; (c) the maximum outer diameter of the affected part of the aorta (hereafter referred to as the maximum aortic diameter); and (d) the presence/absence, volume, and density of pericardial and/or pleural effusion. Bloody pericardial or pleural effusion was defined as fluid collection in the pericardial space that exhibited a CT value of ≥30 Hounsfield units.[1,8]

Pleural effusion, pericardial effusion, and aortic lumen density were measured at ≥5 locations, using regions of interest containing at least 10 pixels, and the mean value was used.

The volume of pericardial and the volume of pleural effusion were calculated by tracing each target area with a workstation using 3-mm-thick short-axis images.

In the no PHD group, a closed false lumen was defined as when no obvious contrast enhancement of a false lumen was seen from entry to re-entry on a contrast-enhanced CT scan. On PMCT, false lumens whose overall densities were 20 Hounsfield units higher (on average) than that of the respective true lumen were defined as closed false lumens.

In the non-PHD group, the aortic diameter was measured by automatically extracting the aorta from contrast-enhanced CT images using the Synapse Vincent software (version 5.3; Fujifilm, Tokyo, Japan). In the PHD group, aortic diameter was measured by manually tracing the aorta on PMCT images using the Synapse Vincent software. In cases in which PMCT showed that the aorta had collapsed, the entire circumference of the aorta was measured, and its diameter was calculated using the following formula: circumference ÷ π. PMCT aortic diameter measurements have been reported to be smaller than ante-mortem aortic diameter measurements. Therefore, in the present study the aortic diameter measurements were subjected to a correction of 20%.[9]

In this study, the aortic arch was defined as the segment between the brachiocephalic artery and the ligamentum arteriosum, and the descending aorta was defined as the segment between the ligamentum arteriosum and the aortic hiatus of the diaphragm.[10]

On CT, the presence or absence of a hematoma in the mediastinum, pulmonary consolidation, aortic arch branch extension, the extension of the dissection to the coronary artery orifice, and pulmonary artery dissection (hematoma) were also examined. Lung consolidation was defined as an area of increased opacification that completely obscured the underlying vascular structures (with a short diameter of ≥1 cm on the short-axis image).[11] Final decisions regarding the classification of lesions were made by the 2 observers reaching a consensus.

2.4. Statistical analysis

All statistical analyses were performed using JMP 15 (SAS Institute Inc., Cary, North Carolina). Categorical data are presented as frequencies and percentages, whereas continuous variables are shown as mean ± SD values. Categorical variables are described as absolute values and percentages, and were compared using the χ^2 test or Fisher exact test, as appropriate, while the Mann–Whitney *U* test was used for comparisons of continuous variables. The factors associated with PHD were analyzed using logistic regression models, and odds ratios (OR) and corresponding 95% confidence intervals (CI) were calculated. As a first step, each variable was subjected to univariate logistic regression analysis. Then, we developed a multivariate logistic regression model, using backward elimination based on likelihood ratios, and a *P* value of <.05 was used as a criterion for selecting variables for the model. Differences or associations that exhibited *P* values of <.05 were considered significant.

3. Results

3.1. Patients' characteristics and CT findings

The basic characteristics and CT findings of the 25 type A AAD patients in the PHD group and 69 type A AAD patients in the no PHD group are summarized in Table 1. A reduced hemoglobin level, the absence of aortic arch involvement, the absence of descending aorta involvement, the absence of abdominal aorta involvement, the absence of aortic arch branch involvement, the presence of bloody pericardial effusion, the presence of a hematoma in the mediastinum, the presence of bloody pleural effusion, the presence of lung consolidation (Figs. 1 and 3), and a reduction in the density of the aorta were found to be significantly more common among the patients in the PHD group than among those in the no PHD group.

The mean maximum aortic diameter was 40.9 ± 7.4 mm in the PHD group and 46.3 ± 6.5 mm in no PHD group. It was significantly smaller in the PHD group (*P* = .0047). However, after these values were subjected to correction, the mean maximum aortic diameter was significantly larger in the PHD group (51.2 ± 9.3 mm vs 46.3 ± 6.5 mm, respectively; *P* = .0428).

In the PHD group, 24 patients (96%) had bloody pericardial effusion. In the no PHD group, 24 patients (35%) had bloody pericardial effusion (*P* < .0001).

In the PHD group, 10 patients (40%) had bloody pleural effusion. In the no PHD group, 1 patient (1%) had bloody pleural effusion (*P* < .0001).

Comparisons of the amounts of bloody pericardial effusion and/or bloody pleural effusion were performed between the cases that exhibited these findings in each group. There was no significant difference in the amount of bloody pericardial effusion between the groups (PHD group [n = 24] vs no PHD group [n = 24]: 290.0 ± 241.2 mL vs 182.2 ± 108.1 mL, respectively; *P* = .1835). However, there was a significant difference in the total amount of bloody pericardial and pleural effusion between the groups (PHD group [n = 25] vs no PHD group [n = 24]: 967.2 ± 191.0 mL vs 189.8 ± 23.2 mL, respectively; *P* < .0001). The total amount of bloody pericardial effusion and bloody pleural effusion exceeded 2000 mL in 6 cases (24%) in the PHD group and 0 cases (0%) in the no PHD group.

3.2. Factors associated with PHD in type A AAD

Table 2 summarizes the results of the univariate and multivariate logistic regression analyses of the factors associated with PHD in type A AAD.

In the univariate analyses, a reduced hemoglobin level, bloody pericardial effusion, a hematoma in the mediastinum, bloody pleural effusion, lung consolidation, an increase in the maximum aortic diameter, and a reduction in the density of the aorta were identified as significant predictors of PHD. On the other hand, aortic arch involvement, descending aorta involvement, and abdominal aorta involvement were identified as significant negative risk factors for PHD.

Table 1

Comparison of patients' characteristics and CT findings between the prehospital death (PHD) and no PHD groups.

Parameter	PHD (n = 25)	No PHD (n = 69)	*P* value
Age, y (mean ± SD)	71.5 ± 12.9	68.0 ± 14.9	.2865
Female sex, n (%)	12 (48)	35 (51)	.8154
Laboratory data			
Hemoglobin (g/dL) (mean ± SD)	10.8 ± 2.4	12.1 ± 2.2	.0317
Platelet count <100,000/μL, n (%)	3 (12)	7 (10)	.7988
Creatinine (mg/dL) (mean ± SD)	1.3 ± 0.6	1.1 ± 0.6	.311
eGFR, n (%) (mean ± SD)	48.6 ± 17.9	55.9 ± 21.3	.1642
Clinical characteristics			
Hypertension, n (%)	23 (92)	68 (99)	.1399
Cerebrovascular disease, n (%)	2 (8)	9 (13)	.5015
Coronary heart disease, n (%)	1 (4)	6 (9)	.4163
Aortic aneurysm, n (%)	2 (8)	2 (3)	.3079
Hemodialysis n (%)	1 (4)	4 (6)	.7241
Diabetes mellitus, n (%)	3 (12)	7 (10)	.7548
Hyperlipidemia, n (%)	3 (12)	6 (9)	.6377
Current smoker, n (%)	9 (36)	16 (23)	.2142
History of malignancy, n (%)	3 (12)	7 (10)	.7548
Anticoagulants, n (%)	2 (8)	2 (3)	.3079
Hypertension drugs, n (%)	23 (92)	68 (99)	.1399
CT findings			
Closed false lumen, n (%)	9 (36)	16 (23)	.2142
Arch involvement, n (%)	19 (76)	66 (96)	.0042
Descending aorta involvement, n (%)	8 (32)	49 (71)	.0006
Abdominal aorta involvement, n (%)	1 (4)	48 (70)	.0006
Coronary artery orifice involvement, n (%)	2 (8)	4 (6)	.6995
Aortic arch branch involvement, n (%)	3 (12)	22 (32)	.0415
Corrected maximum diameter of affected aorta (mm)(mean ± SD)	51.2 ± 9.3	46.1 ± 6.5	.00428
Bloody pericardial effusion, n (%)	24 (96)	24 (35)	<.0001
Mediastinal hematoma, n (%)	22 (88)	10 (14)	<.0001
Bloody pleural effusion, n (%)	10 (40)	1 (1)	<.0001
Lung consolidation, n (%)	11 (44)	6 (9)	<.0001
Pulmonary artery dissection, n (%)	8 (32)	21 (30)	.8848
Density of the aorta (HU) (mean ± SD)	31.9 ± 5.5	42.1 ± 4.8	<.0001

eGFR = estimated glomerular filtration rate, HU = hounsfield units, SD = standard deviation.

In the multivariate analysis, bloody pericardial effusion and lung consolidation were identified as significant risk factors for PHD (OR: 21.29 [95% CI: 1.19–248.29] and 13.72 [95% CI: 1.79–105.06]; $P = .014$ and .012, respectively).

In addition, abdominal aorta involvement was identified as a significant negative risk factor for PHD (OR: 0.02 [95% CI: 0.01–0.65]; $P = .0042$).

4. Discussion

In the present study, bloody pericardial effusion, bloody pleural effusion, and mediastinal hematomas were significantly more common in the PHD group, particularly bloody pericardial effusion (96%). In the univariate analyses, these factors were identified as risk factors for PHD. In the multivariate analysis, bloody pericardial effusion was confirmed as a risk factor for PHD.

Therefore, the significantly lower hemoglobin levels seen in the PHD group might have been due to bleeding. In a comparison of the cases involving bloody pericardial and/or pleural effusion, the total amount of bloody pericardial and bloody pleural effusion was higher in the PHD group, which indicated that bleeding-related PHD might have occurred. Thus, it is suggested that cardiac tamponade and/or blood loss probably cause most PHD in cases of type A AAD.

In a previous study, PMCT revealed a high incidence of bloody pericardial effusion (91%) in patients with type A AAD who experienced OHCPA. In addition, bloody pericardial effusion was found to be significantly associated with OHCPA ($P < .01$).[1] In other studies, cardiac tamponade has been reported to occur as a complication in 19% to 42% of hospitalized type A AAD patients and has been observed in 79% to 87% of autopsy cases with type A AAD.[12,13] Similar to our results, these findings suggest that bleeding is a very important factor in PHD in cases of type A AAD.

The detection of bloody pleural effusion on PMCT does not necessarily indicate an intrathoracic rupture. Previous studies have shown that intrapericardial blood can pass through the pericardium and flow into the thoracic cavity after CPR.[14] Since

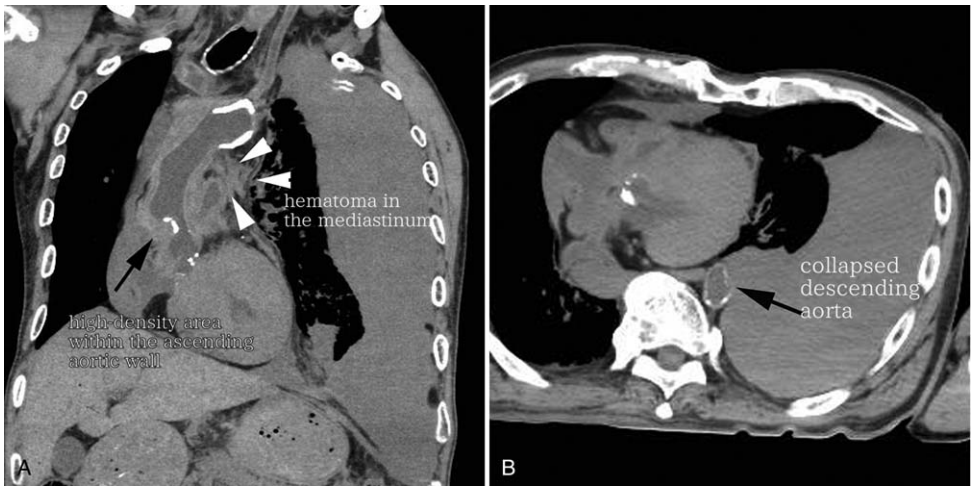

Figure 3. A 72-year-old male with type A AAD. A. PMCT images show a high-density area within the ascending aortic wall (arrow), indicating type A AAD with a closed false lumen. A large amount of left-sided bloody pleural effusion is seen. There is also a hematoma in the mediastinum (arrowheads) and a small amount of bloody pericardial effusion. These findings may suggest that intrapericardial blood has passed through the pericardium and flowed into the left thoracic cavity due to CPR. B. CT image shows bloody left-sided pleural effusion. The descending aorta has collapsed (arrow).

Table 2

Factors associated with prehospital death (PHD) in patients with type A acute aortic dissection (AAD).

Parameter	Univariate analysis		Multivariate analysis	
	OR (95% CI)	*P* value	OR (95% CI)	*P* value
Age (per year)	1.02 (0.98–1.05)	.2887		
Female sex	0.90 (0.35–2.24)	.8154		
Laboratory data				
Hemoglobin (per g/dL)	0.76 (0.61–0.96)	.0142		
Platelet count <100,000/μL, n (%)	1.21 (0.29–5.08)	.7988		
Creatinine (per mg/dL)	1.67 (0.79–3.53)	.1651		
eGFR (per percentage point)	0.98 (0.96–1.01)	.1174		
Clinical characteristics				
Hypertension, n (%)	0.17 (0.01–1.95)	.1399		
Cerebrovascular disease, n (%)	0.58 (0.12–2.89)	.4864		
Coronary heart disease, n (%)	0.44 (0.05–3.83)	.4163		
Aortic aneurysm, n (%)	2.91 (0.39–21.88)	.3079		
Hemodialysis, n (%)	0.68 (0.07–6.36)	.7241		
Diabetes mellitus, n (%)	0.77 (0.15–3.98)	.7507		
Hyperlipidemia, n (%)	1.43 (0.33–6.22)	.6377		
CT findings				
Closed false lumen	1.86 (0.69–5.01)	.223		
Arch involvement	0.14 (0.03–0.63)	.0077		
Descending aorta involvement	0.19 (0.07–0.52)	.0007		
Abdominal aorta involvement	0.02 (0.00–0.14)	<.0001	0.02 (0.01–0.65)	.0042
Coronary artery orifice involvement	1.41 (0.24–8.24)	.7057		
Aortic arch branch involvement	0.29 (0.08–1.08)	.0415	0.98 (0.12–7.70)	.9837
Corrected maximum diameter of affected aorta (per mm)	1.09 (1.02–1.16)	.0067	1.05 (0.95–1.16)	.2921
Bloody pericardial effusion	45.00 (5.73–353.36)	<.0001	21.29 (1.186–248.29)	0.014
Mediastinal hematoma	43.27 (10.89–171.98)	<.0001		
Bloody pleural effusion	44.33 (5.39–348.59)	<.0001		
Lung consolidation	12.77 (3.54–46.01)	<.0001	13.72 (1.79–105.06)	.012
Pulmonary artery dissection	1.08 (0.40–2.88)	.8848		
Density of the aorta (per HU)	0.71 (0.61–0.81)	<.0001		

eGFR = estimated glomerular filtration rate, HU = hounsfield units.

all of the patients in the PHD group underwent CPR, the detection of bloody pleural effusion in these cases does not necessarily suggest that an AD ruptured into the thoracic cavity.[14] This might explain why the amount of bloody pericardial effusion did not differ significantly between the 2 groups. However, the total amount of intracavitary and intrathoracic hemorrhaging exceeded 2000 mL in 6 of 25 cases (24%) in the PHD group, which could have been fatal.

The present study also showed that the extent of the dissection affected the risk of PHD. There are more aorta involvement in the no PHD group than PHD group. The further a dissection extended in the distal direction, the lower the risk of PHD. Previous studies that reviewed autopsy cases of sudden death involving type A AAD reported a high frequency of dissections confined to the ascending aorta and arch.[12] Presumably, if a dissection occurs in the ascending aorta and does not extend distally, there is more pressure on the ascending aorta than if it extends distally. Also, if a dissection is short, the number of re-entries, such as tears and affected aortic branch orifices, is reduced, which would presumably increase the pressure on the false lumen. It is suggested that rupturing is more likely to occur in cases involving short dissections that do not extend distally.

When the uncorrected aortic diameter data obtained in the present study were compared between the groups, the mean maximum aortic diameter was found to be smaller in the PHD group. However, after the data were corrected the mean maximum aortic diameter was significantly larger in the PHD group. In previous studies, comparisons between ante-mortem CT and postmortem CT demonstrated that the aorta usually loses tension and shrinks in diameter by 20% to 25% postmortem.[9] In the current study, even though we only corrected the aortic diameter values by 20%, they were significantly larger in the PHD group. Although further studies are needed to determine its validity, a large aortic diameter was identified as a risk factor for PHD in the univariate analysis. According to previous studies, in patients with AD a large aortic diameter is associated with a higher risk of aneurysms and rupture.[15] As our results demonstrated that rupturing was more common in the PHD group, aortic diameter might be an important contributor to PHD in type A AAD.

In the univariate and multivariate analyses, lung consolidation was also identified as a risk factor for PHD. A pulmonary consolidation is a region of normally compressible lung tissue that has filled with liquid instead of air. It is a radiological sign. The liquid can be pulmonary edema, inflammatory exudate, pus, inhaled water, or blood (from the bronchial tree or hemorrhaging from a pulmonary artery).[16]

According to a previous report, PMCT images of the lung change as the time after death increases due to the natural postmortem changes of the corpse. Early PMCT is more suitable than delayed PMCT for discerning cause of death. Delayed PMCT reflects the autopsy findings more accurately than early PMCT.[17] In this study, all PMCT were performed within 4 hours of onset, which is unlikely to reflect postmortem lung changes. Based on our results, we speculate that the patients in the PHD group might have suffered sudden heart failure due to cardiac tamponade and/or massive hemorrhaging, causing pulmonary edema. They might

also have experienced aspiration due to impaired consciousness. Secondary respiratory distress due to these factors might also be associated with PHD. Patients at risk for aortic dissection may also need to be aware of respiratory disease.

4.1. Limitation

This study had several limitations. First, autopsies were not carried out in all cases. Nevertheless, it was not considered necessary to perform autopsies in every case in this study because of the high diagnostic accuracy of CT for type A AAD.

Second, different CT imaging protocols were used in each group, and contrast-enhanced CT was not employed in the PHD group. Therefore, diagnostic performance might have differed between the groups. In addition, postmortem changes and the stage of decomposition, such as hypostasis, were considered when interpreting the PMCT images because these processes can be mistaken for pathological changes.[1,18,19] However, previous studies found that the high sensitivity (>90%) and specificity (84% to 100%) of non-contrast-enhanced CT were sufficient for diagnosing type A AAD.[1,7,20]

Finally, this study included a relatively small number of subjects; therefore, other unidentified factors might be associated with PHD in patients with type A AAD.

4.2. Future directions

Thus, this study showed that there are several pathological differences between patients with type A AAD that do and do not suffer PHD. Clinically, it is very important to prevent PHD caused by type A AAD. However, at present it is difficult to do this. Further research is needed to establish methods for preventing PHD due to type A AAD based on the pathological differences between cases of type A AAD that do and do not involve PHD.

In conclusion, most PHD in type A AAD are associated with hemorrhaging, and the amount of blood loss is higher in cases that result in PHD than in cases that do not result in PHD. In particular, bleeding into the pericardium is an important and significant risk factor for PHD. Secondary respiratory failure might also be involved in PHD caused by type A AAD. A type A AAD that is confined to the thoracic aorta is also a significant risk factor for PHD. Further research is needed to establish methods for preventing PHD.

Author contributions

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References

[1] Tanaka Y, Sakata K, Sakurai Y, et al. Prevalence of type A acute aortic dissection in patients with out-of-hospital cardiopulmonary arrest. Am J Cardiol 2016;117:1826–30.
[2] Roberts IS, Benamore RE, Benbow EW, et al. Post-mortem imaging as an alternative to autopsy in the diagnosis of adult deaths: a validation study. Lancet 2012;379:136–42.
[3] Levy AD, Harcke HT, Mallak CT. Postmortem imaging: MDCT features of postmortem change and decomposition. Am J Forensic Med Pathol 2010;31:12–7.
[4] Salvolini L, Renda P, Fiore D, Scaglione M, Piccoli G, Giovagnoni A. Acute aortic syndromes: role of multi-detector row CT. Eur J Radiol 2008;65:350–8.
[5] Sebastià C, Pallisa E, Quiroga S, Alvarez-Castells A, Dominguez R, Evangelista A. Aortic dissection: diagnosis and follow-up with helical CT. Radiographics 1999;19:45–60.
[6] Demos TC, Posniak HV, Churchill RJ. Detection of the intimal flap of aortic dissection on unenhanced CT images. AJR Am J Roentgenol 1986;146:601–3.
[7] Kurabayashi M, Okishige K, Ueshima D, et al. Diagnostic utility of unenhanced computed tomography for acute aortic syndrome. Circ J 2014;78:1928–34.
[8] Meziane MA, Fishman EK, Siegelman SS. CT diagnosis of hemopericardium in acute dissecting aneurysm of the thoracic aorta. J Comput Assist Tomogr 1984;8:10–4.
[9] Takahashi N, Higuchi T, Hirose Y, Yamanouchi H, Takatsuka H, Funayama K. Changes in aortic shape and diameters after death: comparison of early postmortem computed tomography with antemortem computed tomography. Forensic Sci Int 2013;225:27–31.
[10] Otsuka T, Sueyoshi E, Tasaki Y, Uetani M. Computed tomography findings and in-hospital mortality in patients with rupture of type B aortic dissection. Acta Radiol 2020;61:136–44.
[11] Maeyashiki T, Suzuki K, Hattori A, Matsunaga T, Takamochi K, Oh S. The size of consolidation on thin-section computed tomography is a better predictor of survival than the maximum tumour dimension in resectable lung cancer. Eur J Cardiothorac Surg 2013;43:915–8.
[12] Murai T. Aortic dissection and sudden death—statistical analysis on 1320 cases autopsied at Tokyo-to Medical Examiner Office. Nihon Hoigaku Zasshi 1988;42:564–77.
[13] Van Arsdell GS, David TE, Butany J. Autopsies in acute type A aortic dissection. Surgical implications. Circulation 1998;98:II299–302.
[14] Okuda T, Takanari H, Shiotani S, Hayakawa H, Ohno Y, Fowler DR. Pericardial tear as a consequence of cardiopulmonary resuscitation (CPR) involving chest compression: a report of two postmortem cases of acute type A aortic dissection with hemopericardium. Leg Med (Tokyo) 2015;17:201–4.
[15] Nienaber CA, Clough RE. Management of acute aortic dissection. Lancet 2015;385:800–11.
[16] Metlay JP, Kapoor WN, Fine MJ. Does this patient have community-acquired pneumonia? Diagnosing pneumonia by history and physical examination. JAMA 1997;278:1440–5.
[17] Shiotani S, Kobayashi T, Hayakawa H, Kikuchi K, Kohno M. Postmortem pulmonary edema: a comparison between immediate and delayed postmortem computed tomography. Leg Med (Tokyo) 2011;13:151–5.
[18] Ishida M, Gonoi W, Hagiwara K, et al. Hypostasis in the heart and great vessels of nontraumatic in-hospital death cases on postmortem computed tomography: relationship to antemortem blood tests. Leg Med (Tokyo) 2011;13:280–5.
[19] Shiotani S, Kohno M, Ohashi N, Yamazaki K, Itai Y. Postmortem intravascular high-density fluid level (hypostasis): CT findings. J Comput Assist Tomogr 2002;26:892–3.
[20] Lovy AJ, Rosenblum JK, Levsky JM, et al. Acute aortic syndromes: a second look at dual-phase CT. AJR Am J Roentgenol 2013;200:805–11.