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Characteristics of Inter-Arm Difference in Blood Pressure in Acute Aortic Dissection

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Background: An inter-arm difference in blood pressure (IADBP) is characteristic of acute aortic dissection (AAD), but the importance of which arm exhibits lower blood pressure (BP) and the mechanism underlying IADBP are not well understood.

Methods: We identified consecutive patients with chest and/or back pain and suspected acute cardiovascular disease whose BP had been measured in both arms. We retrospectively compared the characteristics of such patients with AAD (n=93) to those without AAD (non-AAD group, n=122). Additionally, we separately compared patients with type A AAD (TAAD group, n=58) or type B AAD (TBAD group, n=35) to the non-AAD group. The characteristics analyzed were patient background and IADBP-related factors, including systolic BP (SBP) in the right arm (R) and left arm (L), and R-L or L-R as IADBP. Computed tomography (CT) findings of AD extending to the brachiocephalic artery (BCA) and/or left subclavian artery (LSCA) were examined in patients with an IADBP.

Results: In a comparison of the TAAD group and non-AAD group, the prevalences of R <130 mm Hg (38% vs. 19%, p=0.009), L-R >15 mm Hg (19% vs. 8%, p=0.047), L-R >20 mm Hg (14% vs. 4%, p=0.029) were higher in the TAAD group. Multivariate analysis showed that L-R >15 mm Hg with R <130 mm Hg was independently associated with TAAD (OR 25.97, 95% CI 2.45-275.67, p=0.007). However, IADBP-related factors were not associated with TBAD. AAD patients with L-R >20 mm Hg all had TAAD, and all aortic dissection extended to the BCA just before the right common carotid artery on CT.

Conclusions: IADBP was characterized by R<L with low R in TAAD but was not associated with TBAD. (J Nippon Med Sch 2021; 88: 467–474)

Key words: aortic dissection, inter-arm difference in blood pressure, pulse deficit, characteristics, stanford type A

Introduction

Acute aortic dissection (AAD) is a cardiovascular condition with high mortality. Inter-arm difference in blood pressure (IADBP) and pulse deficit are well-known characteristics of AAD[1–3]. IADBP associated with aortic dissection (AD) is thought to be caused by decreased blood flow in the upper arm from an AD extending to the brachiocephalic artery (BCA) (**Fig. 1**) or left subclavian

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artery (LSCA). Stanford type A AAD (TAAD) is defined as AD involving the ascending aorta, and Stanford type B AAD (TBAD) is defined as AD involving the descending aorta but not the ascending aorta. According to this definition, in TBAD, AD is thought to extend to the BCA with difficulty and systolic blood pressure (SBP) in the right arm does not decrease.

Previous studies of patients with AAD and IADBP did not investigate which arm had the lower BP, whether Stanford type was associated with IADBP, or the mechanism of IADBP. This study attempted to identify the characteristics and mechanism of IADBP in patients with AAD and to identify which arm had the lower BP value.

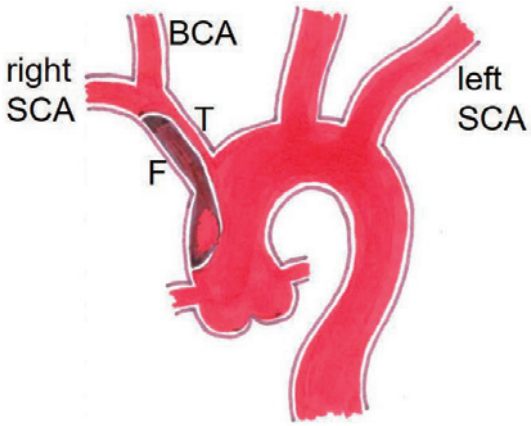


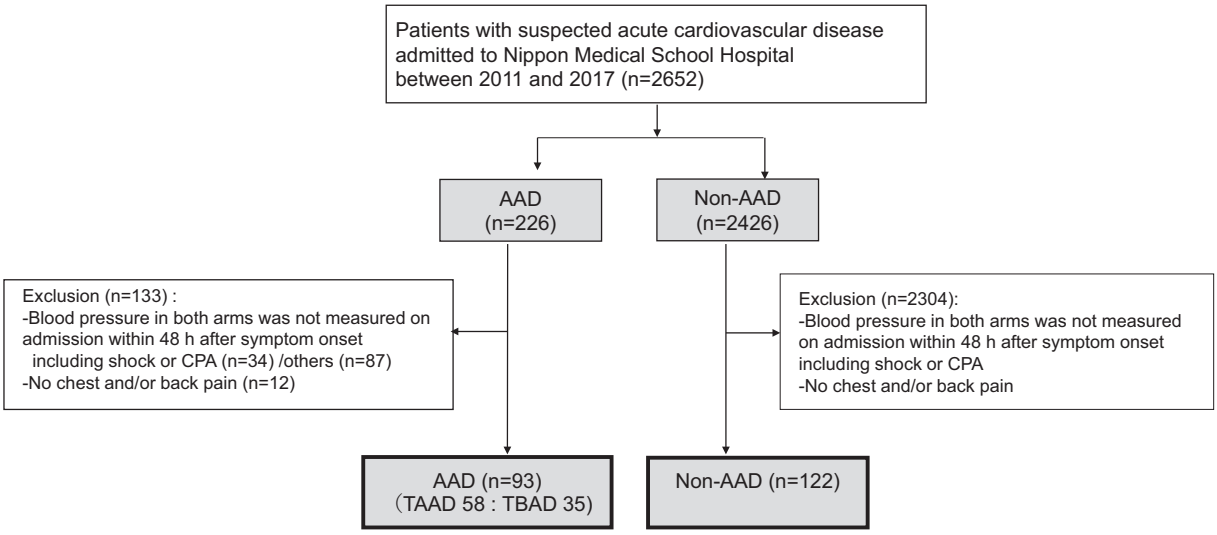
Fig. 1 Schema for aortic dissection causing decreased blood pressure (BP): Aortic dissection from the ascending aorta extends to the brachiocephalic artery (BCA). The true lumen is depressed by a dilated false lumen, resulting in decreased blood flow of the BCA, leading to decreased BP in the upper right arm.

Methods

This retrospective study analyzed clinical records from a single center. The Ethics Committee of the Nippon Medical School Council approved this study. Data collection was carried out by the opt-out method, as outlined on the Nippon Medical School website.

Patient Selection

We investigated 2,652 patients admitted to our hospital with suspected acute cardiovascular disease between January 2011 and March 2017. We classified patients as those with and without AAD. We excluded patients without chest and/or back pain whose blood pressure (BP) was not measured in both arms within 48 hours of symptom onset and those with cardiogenic shock or cardiopulmonary arrest (CPA). Among patients with AAD, BP was not measured in both arms because of cardiogenic shock or CPA in 34 patients and was measured in only one arm in 87 patients. Ultimately, the included patients were classified as having AAD (n=93) or not (n=122). AAD patients were further classified as having TAAD (n=58) or TBAD (n=35). Patients without AAD included those with acute myocardial infarction (n=60; 49%), unstable angina (n=25; 20%), other ischemic heart disease (n=10; 8%), pulmonary embolism (n=4; 3%), congestive heart failure (n=5; 4%), arrhythmia (n=2; 2%), and others (n=16; 13%). The patient selection flow chart is shown in **Figure 2**.



AAD, acute aortic dissection; CPA, cardiopulmonary arrest; TAAD, Stanford type A aortic dissection; TBAD, Stanford type B aortic dissection.

Fig. 2 Flow chart of patient selection.

Table 1 Characteristics of patients with type A aortic dissection (TAAD) and without acute aortic dissection (AAD) with suspected acute cardiovascular disease presenting as chest and/or back pain

Variable	TAAD (n=58)	Non-AAD (n=122)	*P* value
Age (years)	64±15	66±15	0.333
Male (n, %)	39 (67%)	86 (70%)	0.730
Interval from symptom onset to SBP measurement (hours)	5.6±6.3	9.9±9.8	0.003**
History of hypertension (n, %)	47/57 (82%)	92 (75%)	0.244
Diabetes mellitus (n, %)	9/49 (18%)	36/121 (30%)	0.179
Dyslipidemia (n, %)	24/56 (43%)	72 (59%)	0.053
Smoking (n, %)	41/53 (77%)	76/120 (63%)	0.079
R (mm Hg)	136±36	151±28	0.002**
<130 mm Hg (n, %)	22 (38%)	23 (19%)	0.009**
L (mmHg)	138±30	150±27	0.011*
<130 mm Hg (n, %)	22 (38%)	28 (23%)	0.050
R-L (mmHg)	-2±21	2±12	0.117
R<L: L-R>15 mm Hg (n, %)	11 (19%)	10 (8%)	0.047*
>20 mm Hg (n, %)	8 (14%)	5 (4%)	0.029*
R>L: R-L>15 mm Hg (n, %)	10 (17%)	11 (9%)	0.136
>20 mm Hg (n, %)	5 (9%)	6 (5%)	0.336
L-R>15 mm Hg with R<130 mm Hg (n, %)	8 (14%)	1 (1%)	0.001**
R-L>15 mm Hg with L<130 mm Hg (n, %)	5 (9%)	3 (2%)	0.114

AAD, acute aortic dissection; L, systolic blood pressure in left arm; R, systolic blood pressure in right arm; SBP, systolic blood pressure; *, $p<0.05$; **, $p<0.01$

Patient Characteristics

Baseline characteristics were compared in patients with AAD, TAAD, TBAD, and non-AAD. The characteristics analyzed were age, sex, interval from symptom onset to SBP measurement, history of hypertension, diabetes mellitus (DM), dyslipidemia (DL), smoking, factors related to SBP in the right arm (R) (mean values, prevalence of R < 130 mm Hg), factors related to SBP in the left arm (L) (mean values, prevalence of L <130 mm Hg), IADBP-related factors (R-L or L-R, prevalences of L-R >15 and > 20 mm Hg and R-L >15 and >20 mm Hg), and combined factors such as R-related or L-related factors with IADBP-related factors. Furthermore, we analyzed factors associated with TAAD and TBAD by univariate and multivariate logistic regression analyses.

A history of hypertension was defined as use of antihypertensive drugs. DM was defined as an HbA1c ≥ 6.5% or a history of active drug therapy for DM. DL was defined as a low-density lipoprotein cholesterol level ≥ 140 mg/dL, a high-density lipoprotein cholesterol level < 40 mg/dL, a serum triglyceride level ≥150 mg/dL, or a history of drug treatment for dyslipidemia. Smoking included being a current smoker or having a history of smoking. SBP values were measured by manual and automatic sphygmomanometers using a sequential approach.

Association of IADBP with AD Characteristics on Computed Tomography

Computed tomography (CT) findings showing whether AD extended to the BCA and/or LSCA were examined in patients with L-R >20 mm Hg or R-L >20 mm Hg.

Statistical Analysis

Continuous variables were expressed as mean ± SD. Differences in continuous variables between two groups were analyzed with the unpaired Student *t*-test. Categorical variables were expressed as numbers (%) and were compared with the Fisher exact test. Univariate and multivariate logistic regression analyses were performed to identify factors associated with TAAD and TBAD. A *p* value <0.05 was considered statistically significant. SPSS for Windows, version 16.0 (SPSS Inc., Chicago, IL, USA), was used for all statistical analyses.

Results

Characteristics of IADBP in Patients with TAAD and TBAD

Baseline characteristics in the TAAD group were compared with those in the non-AAD group (**Table 1**). Patients in the TAAD group had a shorter interval to SBP measurement (5.6 ± 6.3 vs. 9.9 ± 9.8 hours, $p = 0.003$) and a higher prevalence of R <130 mm Hg (38% vs. 19%, $p = 0.009$). They also had a higher prevalence of IADBP-

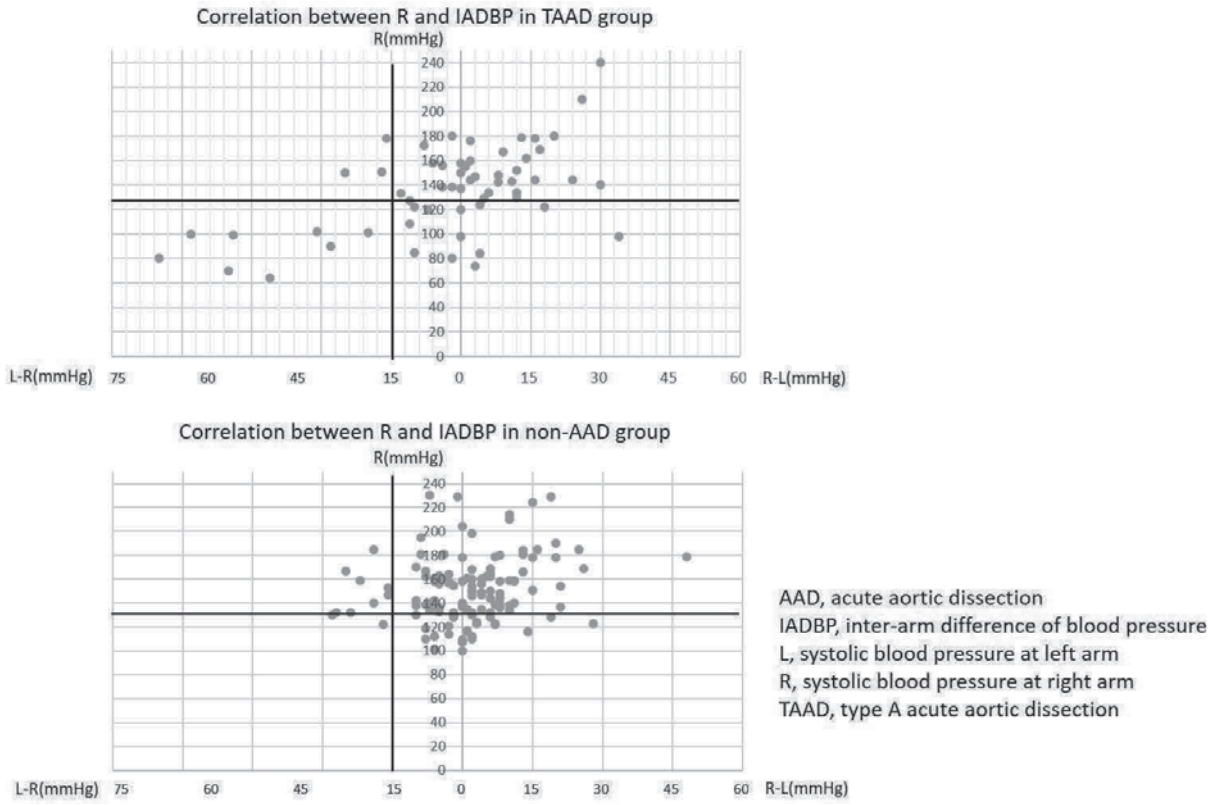


Fig. 3 Correlation of systolic blood pressure in the right arm and inter-arm differences in the TAAD group (upper) and non-AAD group (lower).

related factors with R<L, such as L-R >15 mm Hg (19% vs. 8%, p = 0.047) and L-R >20 mm Hg (14% vs. 4%, p = 0.029), and factors that combined R<L with R, such as L-R >15 mm Hg with R <130 mm Hg (14% vs. 1%, p = 0.001). Associations of IADBP and R in the TAAD and non-AAD group are shown in **Figure 3**. Multivariate logistic regression analysis showed that L-R >15 mm Hg with R <130 mm Hg was associated with TAAD (OR 25.97, 95% CI 2.45-275.67, p = 0.007) and that the OR was higher for the TAAD group than for the AAD group. DM and DL were not independently associated with AAD. Interval from symptom onset to SBP measurement was independently associated with TAAD (OR 0.929, 95% CI 0.887-0.984, p = 0.011) (**Table 2**).

The baseline characteristics of the TBAD group and non-AAD group are shown in **Table 3**. The characteristics of the TBAD group were similar to those of the non-AAD group. SBP in both arms tended to be higher than in the TBAD group, suggesting that blood flow in the left arm is not decreased in TBAD. There were no patients with L-R >20 mm Hg or R-L >20 mm Hg. No factor was independently associated with TBAD in multivariate logistic regression (**Supplementary Table 1**; https://doi.org/10.1272/jnms.JNMS.2021_88-605).

The AAD group included patients with TAAD and TBAD, for which the mechanisms responsible for IADBP are fundamentally different. For reference, the baseline characteristics of patients in the AAD group and non-AAD group are shown in **Supplementary Table 2** (https://doi.org/10.1272/jnms.JNMS.2021_88-605).

Association of IADBP with AD Characteristics on CT

CT findings showing whether AD extended to the BCA and/or LSCA were examined in the 27 patients with L-R >15 mm Hg or R-L >15 mm Hg. CT findings for the 13 patients with L-R >20 mm Hg or R-L >20 mm Hg are shown in **Table 4**. All patients had TAAD; none had TBAD. Characteristics CT findings are shown in **Figure 4**.

In eight patients with L-R >20 mm Hg, all AD extending to the BCA did so distally to at least just before the bifurcation of the right common carotid artery and right subclavian artery (* in **Table 4**; **Fig. 4-1**), and AD extending to the LSCA remained only in the basal segment. In contrast, in five patients with R-L >20 mm Hg, only one had AD extending to a distal segment of the LSCA (** in **Table 4**); however, this AD also extended to the BCA (case 11 in **Table 4**; **Fig. 4-2**). Case 13 was complicated by arterial stenosis in the upper left arm due to progressive systemic sclerosis.

Table 2 Factors associated with TAAD in patients with suspected acute cardiovascular disease presenting with chest and/or back pain

Variable	Univariate			Multivariate (Model 1)			Multivariate (Model 2)		
	OR	95% CI	*P* value	OR	95% CI	*P* value	OR	95% CI	*P* value
Interval from symptom onset to SBP measurement (hours)	0.999	0.998-1.000	0.010*	0.930	0.880-0.982	0.010*	0.929	0.887-0.984	0.011*
History of hypertension (n)	1.703	0.747-3.880	0.205						
Diabetes mellitus (n)	0.531	0.234-1.208	0.131	0.603	0.251-1.451	0.259	0.597	0.242-1.470	0.262
Dyslipidemia (n)	0.521	0.274-0.988	0.046*	0.620	0.296-1.298	0.204	0.586	0.279-1.230	0.158
Smoking (n)	1.978	0.941-4.157	0.072						
R <130 mmHg (n)	2.630	1.309-5.826	0.007**	2.305	1.035-5.135	0.041*			
L-R>15 mmHg (n)	2.621	1.043-6.588	0.040*	2.360	0.777-7.168	0.130			
L-R>15 mm Hg with R <130 mm Hg (n, %)	19.36	2.36-158.86	0.006**				25.974	2.447-275.67	0.007**

CI, confidence interval; OR, odds ratio; L, systolic blood pressure in left arm; R, systolic blood pressure in right arm; SBP, systolic blood pressure; TAAD, type A aortic dissection; *, $p<0.05$; **, $p<0.01$

Table 3. Characteristics of patients with type B acute aortic dissection (TBAD) and without acute aortic dissection (AAD) with suspected acute cardiovascular disease presenting with chest and/or back pain

Variable	TBAD (n=35)	Non-AAD (n=122)	*P* value
Age (years)	71±14	66±15	0.117
Male (n, %)	22 (63%)	86 (70%)	0.413
Interval from symptom onset to SBP measurement (hours)	7.6±9.2	9.9±9.8	0.181
History of hypertension (n, %)	29 (83%)	92 (75%)	0.494
Diabetes mellitus (n, %)	5 (14%)	36/121 (30%)	0.082
Dyslipidemia (n, %)	15 (43%)	72 (59%)	0.122
Smoking (n, %)	25/34 (74%)	76/120 (63%)	0.312
R (mm Hg)	161±28	151±28	0.075
<130 mm Hg (n, %)	6 (17%)	23 (19%)	1.000
L (mm Hg)	159±30	150±27	0.087
<130 mm Hg (n, %)	6 (17%)	28 (23%)	0.642
R-L (mm Hg)	2±10	2±12	0.889
R<L: L-R>15 mm Hg (n, %)	2 (6%)	10 (8%)	1
>20 mm Hg (n, %)	0	5 (4%)	0.588
R>L: R-L>15 mm Hg (n, %)	4 (11%)	11 (9%)	0.745
>20 mm Hg (n, %)	0	6 (5%)	0.339
L-R>15 mm Hg with R<130 mm Hg (n, %)	0	1 (1%)	1
R-L>15 mm Hg with L<130 mm Hg (n, %)	2 (6%)	3 (2%)	0.309

AAD, acute aortic dissection; L, systolic blood pressure in left arm; R, systolic blood pressure in right arm; SBP, systolic blood pressure; TBAD, Stanford type B acute aortic dissection; *, $p<0.05$; **, $p<0.01$

Discussion

The novel findings of this study of IADBP in patients with AAD are that R<L with low R was associated with TAAD, R>L was not associated with TAAD or TBAD, and IADBP-related factors were rarely associated with TBAD. CT findings for patients with R<L showed AD extending to the BCA to at least just before the right common carotid artery.

IADBP and Cardiovascular Disease

Previous studies defined significant IADBP as >10-20 mm Hg[4–7], and an IADBP >20 mm Hg was reported in 4.3% of patients with heterogeneous clinical characteristics in a systematic review[8]. Furthermore 7% of patients with suspected coronary artery disease had an IADBP > 15 mm Hg[9].

IADBP and AAD in Previous Reports

IADBP is a well-known characteristic of AAD[1–3], as is

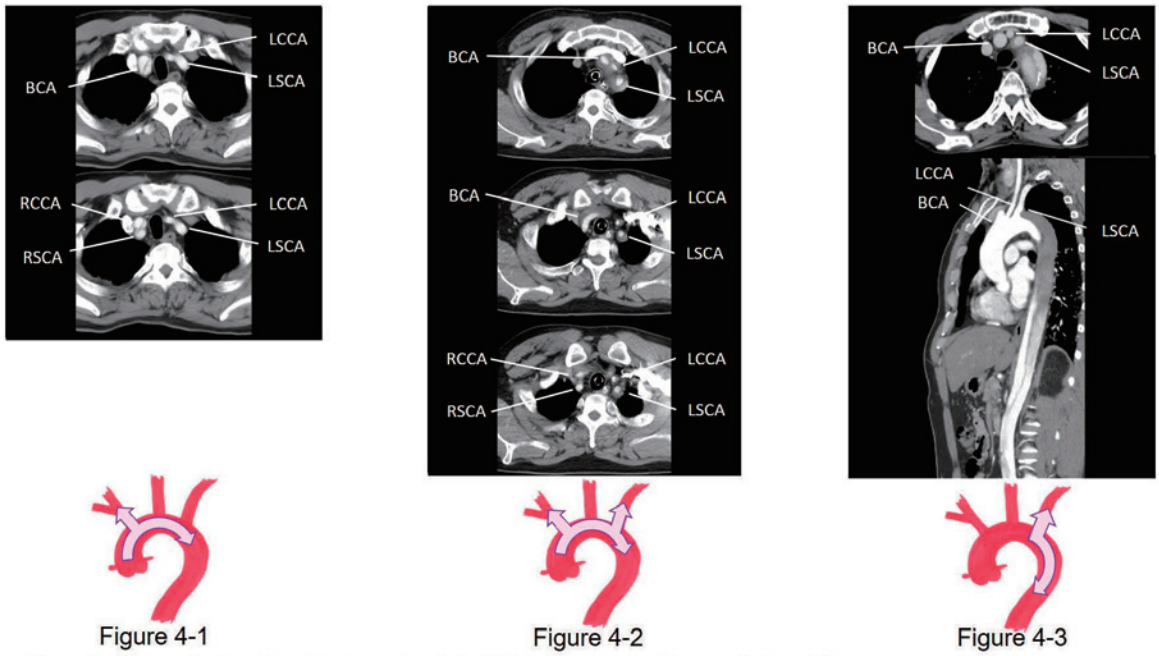


Fig. 4 Characteristic computed tomography (CT) findings for three types of aortic dissection extending to the brachiocephalic artery (BCA) and/or left subclavian artery (LSCA), and their schema. Fig. 4-1: Type A acute aortic dissection (TAAD) extends to the BCA at the distal segment just before the bifurcation of the right common carotid and right subclavian artery, with extension to the LSCA remaining only in the basal segment. Fig. 4-2: TAAD extends to the BCA at a distal segment, with concurrent extension to the LSCA at the distal segment. Fig. 4-3: Type B acute aortic dissection (TBAD) extends to the LSCA and is limited to a basal segment.

Table 4 Characteristics of patients with AAD and an IADBP>20 mm Hg

Case	IADBP (mm Hg)		R (mm Hg)	L (mm Hg)	Stanford type	CT findings
1	L-R>20	25	150	175	A	No extension
2		28	90	118	A	BCA* (Figure 4-1)
3		31	102	133	A	BCA*+LSCA
4		41	64	105	A	BCA*+LSCA
5		49	99	148	A	BCA*
6		50	70	120	A	BCA*
7		58	100	158	A	BCA*+LSCA
8		65	80	145	A	BCA*+LSCA**
9	R-L>20	34	98	64	A	LSCA+BCA
10		30	140	110	A	No extension
11		30	240	210	A	LSCA**+ BCA* (Figure 4-2)
12		26	210	184	A	LSCA+ BCA
13		24	144	120	A	BCA*

AAD, acute aortic dissection; BCA, brachiocephalic artery; CT, computed tomography; IADBP, inter-arm difference in blood pressure; L, systolic blood pressure in left arm; LSCA, left subclavian artery; R, systolic blood pressure in right arm; *, AD extension to the BCA distally just before bifurcation of the right common carotid artery and right subclavian artery; ** AD extension to a distal segment of the LSCA

pulse deficit. A pulse deficit, often defined by clinicians documenting a weak or absent carotid, brachial, or femoral pulse, was reported more frequently than IADBP in AAD[2,3,10,11]. IADBP could thus lead to more objective assessment than pulse deficit, as determined by clinicians. However, few studies have evaluated IADBP in patients

with AAD[1,12–15]. In addition, no previous study has attempted to determine which arm has the lower BP, the association of Stanford type with IADBP, and the mechanism of IADBP in patients with AAD presenting with IADBP.

In previous studies of AAD, a significant IADBP was defined as >20 mm Hg[1,12–15]. Using this definition, the prevalence of IADBP was reported to be 26% to 38% in patients with AAD[1,12,14,15] and 32% in TAAD patients[13]. In the present study, patients with an IADBP >20 mm Hg accounted for almost 14% (9% in L-R >20 mm Hg and 5% in R-L >20 mm Hg) of AAD patients overall and 23% (23% in L-R >20 mm Hg and 0% in R-L >20 mm Hg) of TAAD patients. These proportions are lower than those reported previously.

Mechanisms by which AAD Causes IADBP

IADBP associated with AAD is mainly attributable to AD extending to the BCA, LSCA, or both. R<L associated with AD is primarily caused by depression of the true lumen by a dilated false lumen in the BCA, which is caused by AD extending to at least a BCA mainly originating from the ascending aorta (**Fig. 4-1**). Hence, R<L is assumed to be strongly linked to TAAD only. In contrast, R>L associated with AD is considered to be caused by depression of the true lumen by a dilated false lumen in the LSCA, which is caused by AD extending to at least the LSCA. In such cases, AD is thought to originate from the ascending (**Fig. 4-2**) or descending (**Fig. 4-3**) aorta. R>L appears to be associated with TAAD or TBAD.

When AD extends to both the BCA and LSCA, IADBP would be determined by determining which blood flow is further decreased by AD extending to the BCA or LSCA.

Other Mechanisms of IADBP in Patients with AAD

There are several mechanisms other than AD that may be responsible for IADBP, for example, pre-existing stenosis at a distal portion of the subclavian arteries, caused by atherosclerosis. In the present study, atherosclerotic factors such as hypertension, DM, DL, and smoking were not strongly associated with TAAD or TBAD in multivariate logistic regression analyses. This suggests that associations of atherosclerotic factors with AAD would be similar to those with non-AAD. However, non-AAD was strongly associated with atherosclerotic factors in the present study. A previous study reported that LSCA stenosis, as confirmed by angiography, was present in 3.5% of patients undergoing cardiac catheterization[16]. BP fluctuation between measurements in the right and left arms might also explain IADBP, unless BP was not simultaneously measured in the right and left arms. In the hyper-acute period in AAD patients, BP is often very unstable because of the dynamic obstruction caused by AD. Thus, in addition to IADBP associated with AD extending to the BCA and/or LSCA, other mechanisms of IADBP are likely present.

IADBP and TAAD, and TBAD

First, in comparing TAAD and non-AAD, R<L with low R (L-R >15 mm Hg with R <130 mm Hg) was strongly associated with TAAD (OR 25.97) (**Table 2**), but R>L was not associated with TAAD. As discussed above, R<L is assumed to be associated with TAAD. In fact, the eight patients in this study with L-R >20 mm Hg all had TAAD. Furthermore, CT findings for almost all patients with L-R >20 mm Hg showed AD extension to at least the bifurcation of the right carotid and right subclavian arteries, which likely decreased blood flow in the right arm. Thus, CT findings also support an association of R<L plus low R with TAAD. However, R>L cannot be fully explained by AD extending to the LSCA on CT.

Second, in the comparison of TBAD and non-AD, IADBP-related factors were not associated with TBAD. As discussed above, TBAD is considered to be associated with R>L with decreased L. However, no patient with TBAD had R-L >20 mm Hg. Furthermore, only four patients with TBAD had R-L >15 mm Hg; CT findings from those patients showed AD extension to the LSCA was limited to the basal segment (**Fig. 4-3**), which might not create the substantial LSCA stenosis caused by AD. These results suggest that TBAD is rarely associated with IADBP.

Finally, we analyzed the interval from symptom onset to SBP measurement in patients with AAD, TAAD or TBAD, and non-AAD. This was shorter in the AAD group, possibly because symptoms were more serious in these patients, who may thus have contacted emergency services as soon as they noticed symptoms. Furthermore, multivariate analysis showed that the interval from symptom onset to SBP measurement was associated with TAAD, independent of IADBP-related factors. In other words, IADBP was present regardless of the interval to SBP measurement.

Therefore, R<L plus low R, but not R>L, was associated with TAAD. Furthermore, IADBP was not associated with TBAD. These results were supported by CT findings.

Study Limitations

The present study has several limitations. First, it is retrospective and analyzed a small patient cohort from a

single center. Second, in cases of hemodynamic collapse, such as cardiac tamponade or aortic rupture, blood pressure could not be measured and such cases were therefore excluded; hence, IADBP values in patients with very unstable conditions were not included in the present study. Third, whether BP was measured bilaterally or unilaterally might be subject to selection bias, especially in transferred cases. Some of these patients had established diagnoses before admission to our hospital. In that case, blood pressure measurement for both arms to obtain more diagnostic information on admission was deemed unnecessary. If bilateral BP measurement was less frequent for transferred patients with TAAD, those patients would have been excluded from the analysis, resulting in the impression that IADBP prevalence was lower. This is a possible reason why the prevalence of IADBP in patients with AAD and TAAD in the present study was lower than in previous reports. Fourth, we did not identify any pre-existing stenosis of the distal portion of the subclavian artery, which could have affected blood pressures measured in the upper arm, although such stenosis is not common. Finally, we measured BP in both arms once rather than multiple times, as was done in other studies. However, it is difficult to measure BP several times in patients admitted in an emergency setting.

Conclusions

The characteristics of IADBP in patients with AAD presenting with chest and/or back pain was R<L with low R in those with TAAD. CT findings showed that AD extended to the BCA at least as far as the point just before the right common carotid artery in patients with R<L. In addition, IADBP was not associated with TBAD.

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Conflict of Interest: The authors declare no conflicts of interest.

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