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DOI:

[10.1016/j.amjcard.2017.04.052](https://doi.org/10.1016/j.amjcard.2017.04.052)

Document Version

Peer reviewed version

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Citation for published version (APA):

Delsart, P., Ledieu, G. J., Ramdane, N., Sobocinski, J. P., Clough, R. E., Azzaoui, R. O., Mounier-vehier, C., Nienaber, C. A., & Haulon, S. (2017). Impact of the Management of Type B Aortic Dissection on the Long-Term Blood Pressure. *American Journal of Cardiology*. <https://doi.org/10.1016/j.amjcard.2017.04.052>

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Impact Of the Management of Type B Aortic Dissection on the Long-Term Blood Pressure

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PII: S0002-9149(17)30790-7

DOI: [10.1016/j.amjcard.2017.04.052](https://doi.org/10.1016/j.amjcard.2017.04.052)

Reference: AJC 22613

To appear in: *The American Journal of Cardiology*

Received Date: 1 February 2017

Revised Date: 11 April 2017

Accepted Date: 19 April 2017

Please cite this article as: Delsart P, Ledieu GJ, Ramdane N, Sobocinski JP, Clough RE, Azzaoui RO, Mounier-vehier C, Nienaber CA, Haulon S, Impact Of the Management of Type B Aortic Dissection on the Long-Term Blood Pressure, *The American Journal of Cardiology* (2017), doi: 10.1016/j.amjcard.2017.04.052.

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Title : Impact Of the Management of Type B Aortic Dissection on the Long-Term Blood Pressure

Short title: aortic dissection and blood pressure profile

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Running head: aortic dissection, blood pressure profile: aortic dissection, high blood pressure

Abstract

Ambulatory blood pressure (BP) measurement (ABPM) is recommended to assess optimal BP control, we studied its influence after an acute type B aortic dissection (ATBAD).

We retrospectively collected data from 111 patients with ATBAD from January 2004 to September 2014. Controlled BP group was defined according to a 24-hours BP under 130/80mmHg during chronic phase. The population consisted of 85 males, with a mean age of 61 $\pm$ 13 years and a mean body mass index of 28 $\pm$ 6 Kg/m². The median delay between ABPM and ATBAD was 2[0.3-4] months. The mean 24-hours BP of the entire population was 124/71 $\pm$ 15/8.8 mmHg. BP was not controlled in 41 patients (37%). The treatment score at discharge was 3.9 $\pm$ 1.4. The mean glomerular filtration rate was 83 $\pm$ 28 mL/mn/1.73m², with no difference between groups. Visceral stent implantation in the acute phase (OR=3.857 [1.199-12.406], p=0.023), higher left ventricular ejection fraction (OR=1.092 [1.005-1.187], p=0.038) and higher platelet count at discharge (OR=1.064 [1.018-1.112], p=0.006) were identified as predictors of good BP control by multivariate analysis. The analysis showed that night-time systolic BP was associated with aortic events during follow-up (HR=5.2 [1.01-27.2], p=0.049), particularly for a threshold of 124 mmHg or more (HR=1.967 [1.052-3.678], p=0.0341). Nighttime pulse pressure showed also its significance (HR 20.1 [1.4-282.7], p=0.026). In conclusion, subclinical renal malperfusion revascularization seems to improve BP control. A higher night-time systolic BP was associated with the risk of new aortic events during follow-up.

Keywords: Aortic dissection; blood pressure monitoring; renovascular hypertension; endovascular treatment.

Introduction

Acute type B aortic dissection (ATBAD) is a rare but potential life-threatening vascular emergency and a complex disease to manage. Recent European guidelines state that endovascular techniques are the first line strategy in cases with complications [1]. ATBAD often occurs in patients presenting with the association of atherosclerosis and an old and poorly controlled hypertension[2]. Current guidelines on hypertension management recommend the use of ambulatory blood pressure (BP) measurement (ABPM)[3]. Since, ABPM is a better predictor of cardiovascular outcome than office BP[4]. In this retrospective, single center study, we assessed the prevalence and the impact of ABPM in the chronic phase of type B aortic dissection, as this important aspect is still unknown.

Method

We retrospectively collected data of all patients admitted for a first ATBAD between January 2004 and September 2014 to cardiology intensive care unit. The acute nature of the aortic syndrome was defined by the onset of symptoms < 14 days to diagnosis. Penetrating aortic ulcer and iatrogenic or traumatic dissection were excluded from the study. Pre-hospital and in-hospital deaths were also excluded to assess only patients with a reasonable follow-up.

At admission, the BP target to reach was 120/70 mmHg as recommended by the European guidelines of 2001[5]. In cases with evidence of impending rupture or end-organ ischemia endovascular management was favoured. Ranging from aortic fenestration (scissor technique) to aortic stent graft implantation and/or visceral (renal, celiac, superior mesenteric arteries) stenting[6-7]. Malperfusion syndrome presented as bowel, renal and/or lower limb clinical acute ischemia. Blood investigations at admission and the day of discharge were incorporated into the statistical analysis.

The casual BP on the day of discharge was recorded. Then a 24 hours-ABPM (90207 monitor Spacelabs Healthcare, Snoqualmie, WA, USA) was performed. The cuff was placed on the right arm if the patient had an aortic stentgraft covering of the left subclavian artery. Daytime BP was defined between 6:00 am and 10:00 pm. “Dipper” status was defined as the presence of decreases in the BP values of more than 10% (compared with daytime values). Clinical BP was recorded at the time of the BP monitoring placement, and use of the various classes of antihypertensive medications taken at the time of the ABPM. A medication score (defined as the total number of antihypertensive medications taken by a patient) was recorded. The BP target to reach in the chronic phase was 135/80 mmHg at each consultation[1].

All patients underwent Computed Tomography angiogram (CTA) imaging from the apex of the lungs to the femoral heads before discharge. CTA analysis was performed on a dedicated 3D workstation (Aquarius, Terarecon, CA, USA). The true and false lumen diameters were measured at the level of the left pulmonary artery perpendicular to the line passing through the two insertion points of the intimal flap[8]. Left ventricular ejection fraction was estimated with Simpson’s rules. Left ventricle mass was measured according the American Society of Echocardiography and normalized against body surface area and against height 1.7[9].

Follow-up and long-term information was obtained in May 2015. The primary end point was defined as a new aortic event (defined as a new dissection, aortic rupture or need to perform surgical or endovascular procedure after discharge) after discharge. All cause of death was defined as secondary endpoints.

Quantitative variables are expressed as mean $\pm$ standard deviation in case of normal distribution or median (interquartile range) otherwise, and categorical variables are expressed as number (percentage). Normality of distributions was checked using the Shapiro-Wilk test. Patients were divided into two groups according to the level on ABPM at follow-up: controlled BP group (24-hour mean BP values $< 130/80$ mmHg).

Bivariate comparisons between groups were made using Chi-square test (or Fisher's exact test when the expected cell frequency was <5) for categorical variables and. In case of normal distribution Student t-test was used to compare quantitative parameters, otherwise Mann-Whitney U-test was used. Variables associated with BP control (at $p < 0.20$ in univariate analysis) were introduced in a multivariable logistic regression analysis; predictive power was evaluated using the c-statistics.

Overall survival and aortic event-free probabilities were estimated using the Kaplan-Meier method, and between-group comparisons were done using the log-rank test. Under random censoring assumption, loss of follow-up were censored at last follow-up; for aortic event-free probabilities, patients died from non-aortic disease were also treated as censored events at time of death. As recommended, the duration shown on the curves was stopped if less than 10% of the patients were still under follow-up[10]. Association of 24-BP values with all-cause mortality and aortic events were assessed in univariable Cox models; results are reported as hazard ratio (HR) and predictive power was evaluated using the harrell's c statistic. If a significant association was established, a threshold value to best predict aortic event-free probabilities was determined using the algorithm of maximization of hazard ratio.

Statistical testing was done at the two-tailed a level of 0.05 and data were analyzed using the SAS software package, release 9.4 (SAS Institute, Cary, NC).

Results

One hundred and fifty-three consecutive patients with first ATBAD were identified from hospital records. In-hospital mortality was 11% ($n=17$). Twenty five patients have no useful blood pressure monitoring data. Finally, our analysis on 111 patients with ABPM available for follow-up. The left ventricular ejection fraction was significantly higher in the BP controlled group ($60\% \pm 5.9$ vs $58\% \pm 5.1$, $p=0.0447$). Demographics data and the cardiovascular risk factors are described in Table 1.

The median delay between discharge and ABPM measurement was 2[0.3-4] months with no difference between the 2 groups. The average treatment score was 3.9 ± 1.4 drugs per day with no difference between groups. The average clinical BP in all patients at discharge was controlled ($128.8 \text{ mmHg} \pm 15.9$ for the systolic, and $73.4 \text{ mmHg} \pm 12.5$ for the diastolic). The systolic BP at discharge was significantly higher in the uncontrolled group compared with the controlled group ($133 \text{ mmHg} \pm 13.9$ vs $126.3 \text{ mmHg} \pm 16.5$, $p=0.036$). The clinical BP at the time of ABPM was $131.1 \pm 18.8 \text{ mmHg}$ for the systolic and $76.3 \pm 11.5 \text{ mmHg}$ for the diastolic. The details of the ABPM data are listed in Table 2.

The acute phase medical management is summarized in table 3. BP was better controlled in the group that received visceral/renal stent placement. Visceral stent implantation in the acute phase (OR=3.857 [1.199-12.406], $p=0.023$), higher LVEF (OR=1.092 [1.005-1.187], $p=0.038$) and higher platelet count at discharge (OR=1.064 [1.018-1.112], $p=0.006$) were demonstrated independently associated with successful BP control in multivariate analysis (Table 4) over a median follow-up period of 51[2-81] months. During follow-up, an aortic event occurred in 40 patients (38.7%) with a median delay of 23.5[9-51] months. In univariate Cox's regression analysis, night time systolic BP was identified as a predictor of aortic events during follow-up (HR=5.2 [1.01-27.2], $p=0.049$). A threshold of 124mmHg was identified the best cut-off value to predict these events (HR=1.967 [1.052-3.678], $p=0.0341$) (Figure 1). The nighttime pulse pressure has a strong impact (HR=20.1 [1.4-282.7], $p=0.026$) on outcomes. Overall all-cause mortality at follow-up was 21% ($n=22$) with a median delay of 41[22-75] months. There was no significant association between the different BP data (BP at discharge, BP at consultation time, and all data of the ABPM) and the risk of death during follow-up.

Discussion

Night time systolic BP is associated with an increased risk of aortic events during follow-up. The results show that endovascular revascularization of (visceral/renal) malperfusion syndrome is a strong predictor of BP control.

As guidelines are targeting a low normal BP, we could have demonstrated earlier that 4.3 drugs are necessary to reach the target BP[11]. The European Society of Hypertension define BP control as a mean BP of less than 130/80mmHg on the 24 hours ABPM. The ABPM thresholds were determined using the results of outcome studies such as the International Database of Ambulatory blood pressure in relation to Cardiovascular Outcome (IDACO), as the Ohasama study, as the PAMELA study and as the Dublin study[12-13-14-15]. The threshold of 130/80 mmHg on ABPM corresponds to a BP threshold of 140/90 mmHg at consultation. In the management of chronic aortic syndrome, the European Society of Cardiology suggests a threshold of 135/80mmHg on office measurement[5]. Thus, the ideal target to reach with ABPM should be lower.

Complications in the acute phase were recently defined by an expert consensus as malperfusion, hypertension associated with malperfusion or persisting despite full medical therapy, or increase in peri-aortic hematoma and hemorrhagic pleural effusion suggesting impending rupture[16]. In the special situation of resistant hypertension, early endovascular treatment has to be discussed. Indeed, data from IRAD suggest increased adverse outcomes in the acute phase for patients with resistant hypertension resulting in an increase in mortality mostly due to aortic rupture[17]. About 20 to 25% of individuals with ATBAS have a malperfusion syndrome with 10 to 15% of these involving renal arteries, which is an independent predictor of in-hospital mortality[18]. Renal malperfusion can be difficult to diagnose and can be underestimated[19]. Data from a series of spontaneous renal artery dissections presenting with severe hypertension report clinical success in treating hypertension by endovascular stent placement during long term follow-up[20]. The present

study cohort suggests a potential benefit of early endovascular treatment of malperfusion on successful blood pressure control in the chronic phase. Coagulation disorders have previously been described in the acute phase of aortic dissection and been shown to affect prognosis[21-22]. Our study also suggests a relationship between platelet count and blood pressure control which could be explained by chronic inflammation induced by the dissection and false lumen activity, such conceptual thoughts however need more solid foundation.

Finally, nighttime systolic BP appears to have a particularly important prognostic significance. Previous data suggest that night time systolic BP was the best predictor of cardiovascular events and mortality in patients with hypertension[12-13-23]. A higher systolic BP probably results from greater sympathetic activity[24] or greater stiffness of the peripheral arterial system. Considering the results, it is also logical to speculate that similar finding can be observed in type A dissection patient.

The limitations of this study include its retrospective design and single center data source, rendering it difficult to generalise our findings.

Disclosures

The authors have no conflict of interest to declare connecting with this manuscript.

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Figure Legends

Figure 1 : Prognostic significance of systolic nighttime blood pressure on aortic events.

Abbreviations : **NTSB** : *nighttime systolic blood pressure*

Variable	BP controlled			
	Overall n=111	Yes (n=70)	No (n=41)	p value
Age (years)	60.9 ± 12.9	60.1 ± 12.5	62.2 ± 13.6	0.361
Female sex	26 (23%)	17 (24%)	9 (22%)	0.779
Smoker	35 (31%)	22 (31%)	13 (32%)	0.976
Hypertension	80 (72%)	44 (63%)	36 (88%)	0.005
Dyslipidemia	27 (24%)	14 (20%)	13 (32%)	0.165
Diabetes mellitus	11 (10%)	10 (14%)	1 (2%)	0.051
Body mass index (Kg/m) ²	28.4 ± 6.2	28.6 ± 6.3	28.2 ± 6	0.839
Family history of CVD	12 (11%)	5 (7%)	7 (17%)	0.119
Prior atherosclerosis and cardiovascular disease	14 (13%)	8 (11%)	6 (15%)	0.623
	8 (7%)	7 (10%)	1 (2%)	0.254
Prior thoracic aortic aneurysm	3 (3%)	2 (3%)	1 (2%)	NA
Prior type A aortic dissection	10 (9%)	7 (10%)	3 (7%)	0.742
Prior cardiac surgery	9 (8%)	7 (10%)	2 (5%)	0.481
Atrial fibrillation	11 (10%)	8 (11%)	3 (7%)	0.744
Connective tissue disease	7 (6%)	6 (9%)	1 (2%)	1

Table 1 : Baseline characteristics of the patients.

Data are expressed by mean ± standard deviation.

Abbreviations : BP : blood pressure. CVD : cardiovascular disease. ECG : electrocardiogram. LVEF : left ventricular ejection fraction. LVM : left ventricular mass. NA : not applicable.

Prior cardiovascular disease : peripheral arterial disease, coronary artery disease or aortic aneurysm.

Tobacco use : current smoking or stopped from less than 3 years.

Variable	BP Controlled			p value
	Overall n=111	Yes (n=70)	NO (n=41)	
24-h SBP (mmHg)	124 ± 15	115 ± 9	140 ± 10	< 0.001
24-h DBP (mmHg)	71 ± 9	67 ± 6	78 ± 8	< 0.001
24-h PP (mmHg)	53 ± 11	48 ± 9	61 ± 10	< 0.001
24-h HR (bpm)	68 ± 10	66 ± 10	70 ± 10	0.052
Daytime SBP (mmHg)	125 ± 15	117 ± 10	140 ± 10	< 0.001
Daytime DBP (mmHg)	72 ± 9	68 ± 7	79 ± 8	< 0.001
Daytime PP (mmHg)	53 ± 11	48 ± 9	61 ± 10	< 0.001
Daytime HR (bpm)	69 ± 11	68 ± 11	72 ± 10	0.057
Nighttime SBP (mmHg)	121 ± 18	112 ± 12	138 ± 13	< 0.001
Nighttime DBP (mmHg)	69 ± 10	64 ± 7	77 ± 10	< 0.001
Nighttime PP (mmHg)	53 ± 12	48 ± 10	61 ± 11	< 0.001
Nighttime HR (bpm)	65 ± 11	64 ± 10	68 ± 10	0.042
Dipping status	33 (30%)	25 (36%)	8 (20%)	0.072

Table 2 : 24-hours blood pressure measurement data.

Data are expressed by mean ± standard deviation and median (interquartile range)

Abbreviations : ACE inhibitor : angiotensin-converting-enzyme inhibitor. ARB : angiotensin-receptor

blocker. BP : blood pressure. bpm : beats per minute. DBP : diastolic blood pressure. HR : heart rate.

SBP : systolic blood pressure. VKA : vitamin K antagonist. NA : not applicable.

Variable	BP Controlled			
	Overall n=111	Yes (n=70)	NO (n=41)	p value
Pharmacotherapy at discharge				
Beta-blocker	107 (96%)	67 (96%)	40 (98%)	NA
ACE inhibitor	78 (70%)	49 (70%)	29 (71%)	0.935
ARB	22 (20%)	12 (17%)	10 (24%)	0.355
Calcium-channel blocker	97 (87%)	60 (86%)	37 (90%)	0.488
Antialdosterone	32 (29%)	17 (24%)	15 (37%)	0.167
Thiazide diuretic	55 (49%)	31 (44%)	24 (58%)	0.147
Treatment score	3.9 ± 1.4	3.8 ± 1.4	4.1 ± 1.3	0.143
Endovascular procedure				
Aortic endograft	48 (43%)	34 (49%)	14 (34%)	0.14
Fenestration	31 (28%)	20 (28%)	11 (27%)	0.81
Visceral artery stenting	7 (6%)	5 (7%)	2 (5%)	NA
	26 (23%)	21 (30%)	5 (12%)	0.032
Early aortic complications				
Malperfusion syndrome	40 (36%)	26 (37%)	14 (34%)	0.75
Bowel ischemia	13 (12%)	10 (14%)	3 (7%)	0.37
Renal ischemia	24 (22%)	18 (26%)	6 (15%)	0.17
Lower limb ischemia	13 (12%)	7 (10%)	6 (15%)	0.55
Impending aortic rupture	11 (10%)	8 (11%)	3 (7%)	0.74
At discharge				
GFR (mL/mn/1.73m ²)	83.7 ± 28.2	83.6 ± 28.9	83.9 ± 27.2	0.95
Platelet count (/mm ³)	329 000	± 352 000 ± 116 000	289 000	± 0.006

	113 000		94 267	
LDL cholesterol (mg/dl)	104 ± 36	99 ± 39		0.002
			116 ± 30	
Ascending aorta diameter	36 ± 4.8	36.6 ± 4.7	35 ± 4.8	0.105
(mm)	37.3 ± 6.2	37.7 ± 6.4	36.7 ± 5.8	0.504
Thoracic descending aorta diameter (mm)	19.9 ± 7.8	20.1 ± 8.2	19.7 ± 7	0.814
False lumen diameter (mm)				
False lumen status				
Complete thrombosis	7 (6%)	4 (6%)	3 (7%)	NA
Partial thrombosis,	38 (34%)	22 (31%)	16 (39%)	
Patent	51 (46%)	34 (49%)	17 (42%)	
Not analysable	15 (14%)	10 (14%)	5 (12%)	

Table 3: In-hospital management, biological and morphological data at discharge.

Data are expressed by mean ± standard deviation.

Abbreviations : ACS : acute coronary syndrome. BP : blood pressure. GFR: glomerular filtration rate.

NA: not applicable

Variable	Units	OR [CI 95%]	p value
Visceral artery stenting	1	3.857 [1.199-12.406]	0.024
Platelet count at discharge	10 000	1.064 [1.018-1.112]	0.006
LVEF	1	1.092 [1.005-1.187]	0.038

Table 4: Multivariate analysis predicting good blood pressure control

Abbreviations : CI : confidence interval. LVEF : left ventricular ejection fraction. OR : odds ratio.

C-statistics (CI 95%)= 0.74 (0.64-0.83)

Figure 1: Kaplan-Meier curve for aortic events stratified by nighttime systolic blood pressure value

Abbreviations : NTSBP : nighttime systolic blood pressure