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ORIGINAL RESEARCH

Predictors of poor blood pressure control assessed by 24 hour monitoring in patients with type B acute aortic dissection

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Abstract: The chronic management of post-acute aortic dissection (AD) of the descending aorta (Type B) is based on optimal control of blood pressure (BP), with a target BP < 135/80 mmHg. The aim of our study was to determine and verify effective blood pressure control with an objective measurement method and to identify predicting factors.

Methods: We collected data from 26 patients hospitalized in the acute phase of a Type B AD between 2006 and 2009. Two groups were defined according to 24 hour BP monitoring results at follow-up. Group 1 consisted of patients with a controlled BP (<130/80 mmHg), and Group 2 consisted of patients with an uncontrolled BP.

Results: Thirty four percent of patients showed an uncontrolled BP at checkup. Vascular history before AD ($P = 0.06$), high baseline BP trend ($P = 0.01$ for systolic and $P = 0.08$ for diastolic), and greater diameter of the descending aorta ($P = 0.02$) were associated with poor BP control.

Conclusion: Prognosis after AD is associated with BP control. Therefore, 24 hour BP monitoring can be made.

Keywords: acute aortic syndrome, blood pressure monitoring, hypertension

Introduction

Aortic dissection (AD), a component of the acute aortic syndrome, is a rare disease with a poor prognosis. Its estimated incidence falls between 0.5 and 2.95 cases per 100,000 people per year.[1–4] While Type A AD treatment is based on emergency surgery, treatment of Type B AD is essentially clinical with the combination of endovascular techniques in case of organ malperfusion or aortic rupture threat. Nevertheless, short- and mid-term prognosis of Type B AD is poor, with 10% intrahospital deaths[5] and 25% mortality in the following 3 years, according to data from the International Registry of Aortic Dissection (IRAD).[6]

The management of these patients during the acute phase remains difficult, based primarily on blood pressure (BP) control and on treatment with analgesics. Chronic treatment is based on clinical and morphological monitoring of the aortic tree. Optimal BP control is recommended for patients who have a history of AD (Type A or B) with a target systolic BP lower than 135 mmHg and a target diastolic BP lower than 80 mmHg. However, these values were arbitrarily determined and require validation. The prognosis of these patients is associated with ectasia of the aorta and its implications. BP control seems to be the main issue after such a vascular event, even more so because it concerns severely hypertensive patients and is often overlooked. There are few data on antihypertensive treatment. Beta-blockers are the only type of drugs systematically used,[7,8]

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although a combination of four to five antihypertensive drugs appears to be necessary to reach BP control.[9] A recent update of the European recommendations on arterial hypertension stressed the importance of ambulatory measures.[10] These measures, including BP self-measurement and 24 hour monitoring, are common practice in the management of hypertension, but to our knowledge, no study has focused on these reliable methods of BP measuring in the particular context of AD.

The aim of this study was to ascertain the control of high BP in our population by an objective method of ambulatory BP measurement and to determine the predicting factors of poor BP control after a Type B AD.

Methods

Population

The data come from our local registry, comprising all patients hospitalized in the acute phase of an acute aortic dissection (AD [including intramural hematoma]) between 2006 and 2009, and monitored in our center.

The management of all patients was identical. All patients were admitted to the Emergency Department in cardiac intensive care, and after hemodynamic stabilization and management of any vascular or general emergencies, they were transferred to the department of vascular medicine and arterial hypertension.

From November 2006 through April 2009, 26 patients consecutively hospitalized and monitored in our center to support an AD of the descending aorta were included in the study. In these patients, the AD etiology was atherosclerotic, except in two patients, who were known to present elastopathy before hospitalization.

Dissections of traumatic origin were not included in our study. An AD of the descending aorta was defined as not involving the ascending aorta. The acute nature was defined among those patients who were hospitalized within 14 days of symptom onset.[11] We distinguished between intramural hematoma and aortic dissections with thrombosed false lumen by the absence of an intimal flap or individualized entry point in case of parietal hematoma.[12] No patient with a penetrating aortic ulcer was included in the study.

Treatment of the acute phase

Medical management in the acute phase was based on antihypertensive treatment with a target systolic BP of 120 mmHg, analgesic treatment, and management of vascular complications.

In cases of organ malperfusion, patients underwent endovascular revascularization such as aortic fenestration using the scissors technique[13] or aortic stent, with or without a peripheral stent.[14]

Clinical malperfusion was defined as limb ischemia or mesenteric ischemia, and renal malperfusion was defined as acute renal failure and arterial hypertension not controlled by optimal medical treatment where acute renovascular etiology by static or hemodynamic malperfusion was suspected.

Clinical data

Vascular history was assessed via coronary artery disease, peripheral arterial disease or stroke that had occurred before hospitalization. The history of aortic intervention was collected and included dissections and surgeries for aneurysms of the abdominal or thoracic aorta. Data on classical cardiovascular risk factors and waist circumference were also collected. Abdominal obesity was defined as waist circumference greater than 102 cm for men and 88 cm for women. Patients were considered active smokers if they were currently smoking or had stopped during the three years prior to hospitalization.

Metabolic syndrome was defined according to the criteria set by the IDF (International Diabetes Federation), the AHA (American Heart Association) and the NHLBI (National Heart, Lung and Blood Institute) in 2009.[15] Patients were considered to have metabolic syndrome if they presented at least three of the following five criteria: abdominal perimeter $\geq$102 cm for men or $\geq$88 cm for women; triglycerides $>$1.5 g/L or under treatment; HDL $<$0.4 in men and $<$0.5 in women; arterial pressure $\geq$130/80 mmHg or under treatment; and fasting glycemia $\geq$1.0 g/L.

The occurrence of an intrahospital cardiovascular event, such as myocardial infarction or stroke, was also recorded for each patient.

Blood pressure data

Upon admission to the intensive care unit, the first BP measure was recorded by the nurse. The BP at the patient's discharge was the casual oscillatory BP measured by the nurse in the department of vascular medicine and arterial hypertension, before discharge. After the acute phase, all patients received 24 hour BP monitoring with an adapted cuff. The equipment used was a Spacelabs Medical 90207 ambulatory blood pressure monitor (Spacelabs Medical Ltd, Issaquah, WA). A BP measure was performed every 15 minutes. Daytime BP was measured between 6:00 am and 10:00 pm, and nighttime BP was measured between 10:00 pm and 6:00 am.

The patients were divided into two groups according to the average 24 hour BP measured by monitoring. Group 1 consisted of patients who had a systolic BP strictly below 125 mmHg and a diastolic BP strictly below 80 mmHg. Group 2 consisted of patients who had a systolic BP greater than or equal to 125 mmHg and/or a diastolic BP greater than or equal to 80 mmHg.

Biological data

Renal function was assessed by estimating the glomerular filtration rate using the Modification of Diet in Renal Disease (MDRD) method, based on creatininemia measured on hospital discharge day.[16] Each patient underwent an exploration of lipid levels including LDL, HDL, and triglyceridemia.

Morphological data

Each patient had a CT or MRI angiography with injection before hospital discharge. The morphological data analyzed in this study was based on this reference examination performed at our center. The length of the ascending aorta was measured before the left pulmonary artery and perpendicular to the aortic arch. Measurement of the descending thoracic aorta was performed behind the right pulmonary artery. The aortic diameter was also measured at the diaphragm and subrenal aorta. Measurement of the false and true channel was done in a standardized manner, by taking the measure on a line perpendicular to the line of insertion of the intimal flap.[17] Finally, extension of the dissection into a peripheral artery (gastrointestinal, renal, or iliac) was noted. This was defined as the identification of an intimal flap and two distinct lumens in an iliac, renal, or gastrointestinal artery.

Discharge treatment

Antihypertensive drug classes used in discharge treatment were recorded for each patient. The six classes used were inhibitors of the renin-angiotensin system (inhibitors of the angiotensin-converting enzyme and angiotensin II receptor antagonists), beta-blockers, diuretics (intake of thiazides or anti-aldosteronic drugs or both), calcium channel blockers, alpha-blockers, and central antihypertensive drugs. The number of antihypertensive drugs used (from different classes) was also recorded, defining a treatment score.

Statistical methods

Statistical analyses were performed with SPSS software version 11.5 (IBM, Armonk, NY). Because the number of patients in each group was lower than 30, nonparametric tests were performed. Continuous variables were compared with the Mann–Whitney test, and categorical data were tested by Fisher's exact test. Continuous variables were expressed as mean ± standard deviation. Categorical data were expressed as percent. The significance threshold was set at 0.05.

Results

Nine patients (34% of all patients) had poor BP control. Among the initial characteristics, BP control did not correlate with the patient's age, gender, or body mass index. Only vascular history tended to be associated with poor BP control ($P = 0.06$). These results are summarized in Table 1.

BP measured upon admission did not affect future BP control, while BP measured casually at the patient's discharge was correlated with poor BP control in the chronic phase ($P = 0.017$ for systolic BP and $P = 0.088$ for diastolic BP on discharge). Patients with a high BP trend at discharge were more likely to be poorly controlled (Table 2).

However, we noticed that at follow-up, the group of patients not controlled by monitoring presented both systolic and diastolic BP within the recommended discharge target values (124.4 mmHg and 70.6 mmHg, respectively).

The data resulting from 24 hour monitoring is shown in Table 3. There was a statistically significant difference in all parameters of the 24 hour monitoring (Figure 1).

All patients received a beta-blocker and an inhibitor of the renin-angiotensin system, so this parameter could not be analyzed. The patients received at least three antihypertensive drugs on discharge.

There was no difference between the different therapeutic classes used in each group, and there was a trend to use a greater number of antihypertensive drugs in the less controlled patients group ($P = 0.07$) (Table 4).

Concerning the morphological data of Type B AD, patients who had a smaller diaphragmatic aortic diameter and subrenal aortic diameter were better controlled (respectively $P = 0.02$ and $P = 0.05$) (Table 5). Other parameters, such as the diameter of the ascending aorta or the diameter of the false lumen, did not affect BP control. Similarly, no statistically significant difference was noted between intramural hematomas and AD.

Discussion

The aim of our study was to assess BP control after a Type B AD with a reliable measurement method and to identify potential predicting factors of poor BP control. Thirty four percent of our population was not at the recommended BP goal, and a minimum of three to four antihypertensive

Table 1 Population characteristics

Clinical and demographic data	Overall population n = 26	Group 1 Controlled blood pressure (n = 17)	Group 2 Uncontrolled blood pressure (n = 9)	*P*-value
Age (years)	62.7 ± 11.9	63.6 ± 10.9	68.0 ± 12.1	0.66
Male/female	24 (92.3)	15 (88.2)	9 (100)	0.53
Vascular history (n, %)	6 (23.1)	2 (11.7)	4 (44.4)	0.06
Aortic history (n, %)	3 (11.5)	3 (17.6)	0 (0)	0.19
COPD (n, %)	3 (11.1)	1 (5.9)	2 (22.2)	0.22
Elastopathy (n, %)	2 (7.7)	2 (11.7)	0 (0)	0.29
Cardiovascular risk factors				
Hypertension (n, %)	19 (73.1)	11 (64.7)	8 (88.8)	0.19
Smoking (n, %)	13 (50)	7 (41.1)	6 (66.6)	0.22
BMI (Kg/m²)	28.8 ± 6.7	27.8 ± 6.6	29.9 ± 12.6	0.86
Diabetes (n, %)	2 (7.7)	2 (11.7)	0 (0)	0.29
Dyslipidemia (n, %)	11 (42.3)	6 (35.3)	5 (55.5)	0.33
Abdominal obesity ≥88 cm if ♀, ≥102 cm if ♂	17 (65.4)	10 (58.8)	7 (77.7)	0.34
Metabolic Syndrome (n, %) (IDF/AHA/NHLBI 2009)	15 (57.7)	8 (47.0)	7 (77.7)	0.13

Abbreviations: COPD, chronic obstructive pulmonary disease; BMI, body mass index.

treatments seemed necessary before discharge. The patients at a higher risk of poor BP control were those who had a trend of high systolic and diastolic BP readings upon discharge. BP control in patients with Type B AD correlated statistically to the situation: vascular patients before hospitalization were likely to be less well controlled. Conversely, patients with smaller diaphragmatic and subrenal aortic diameter showed a better-controlled BP.

BP control after acute AD

While the negative influence of a high BP in the acute phase of a Type B AD has been presumed for a long time, the IRAD registry has only recently confirmed this hypothesis. From a population of 365 patients with Type B AD, persistent (or recurrent) chest pain and arterial hypertension (not controlled according to European recommendations) were independent predictors of total intrahospital mortality (17.4% vs 4%).[18]

Table 2 Clinical and biological data, hospital care

	Overall population (n = 26)	Group 1 Controlled BP (n = 19)	Group 2 Uncontrolled patients (n = 7)	*P*-value
Clinical and biological data on admission				
Systolic blood pressure on admission (mmHg)	154.9 ± 29.4	155.5 ± 31.6	170.3 ± 33.6	0.24
Diastolic blood pressure on admission (mmHg)	71.7 ± 15.7	73.6 ± 18.9	78.9 ± 12.7	0.26
Heart rate on admission (bpm)	74.8 ± 12.2	76.2 ± 12.1	76.1 ± 15.5	0.11
MDRD clearance (Kg/ml/1.73 m²)	79.2 ± 23.0	72.3 ± 22.1	80.3 ± 18.7	0.48
HDL (g/L)	0.48 ± 0.15	0.44 ± 0.14	0.47 ± 0.11	0.74
LDL (g/L)	1.0 ± 0.38	1.11 ± 0.40	1.14 ± 0.33	0.91
Triglyceridemia (g/L)	1.1 ± 0.46	1.15 ± 0.56	1.00 ± 0.39	0.89
Intrahospital management				
Endovascular intervention (n, %)	9 (34.6)	7 (41.1)	2 (22.2)	0.34
Cardiovascular events (n, %)	3 (11.5)	2 (11.7)	1 (11.1)	0.96
Pleural effusions (n, %)	5 (19.2)	5 (29.43)	0 (0)	0.07
MDRD clearance on discharge (Kg/ml/1.73 m²)	78.7 ± 23.9	69.1 ± 20.9	79.9 ± 23.7	0.36
SBP on discharge (mmHg)	116.1 ± 15.0	112.2 ± 10.8	124.4 ± 18.0	0.017
DBP on discharge (mmHg)	65.9 ± 10.3	64.8 ± 10.7	70.6 ± 8.0	0.088
Pulse pressure on discharge (mmHg)	50.4 ± 9.9	48.1 ± 6.7	53.9 ± 11.8	0.087
Heart rate on discharge (bpm)	67.3 ± 12.4	65.7 ± 13.6	71.1 ± 11.6	0.18

Abbreviations: MDRD, modification of diet in renal disease; HDL, high density lipoprotein; LDL, low density lipoprotein; SBP, systolic blood pressure; DBP, diastolic blood pressure.

Table 3 Blood pressure monitoring

	Overall population n = 26	Group 1 Controlled blood pressure (n = 19)	Group 2 Uncontrolled blood pressure (n = 7)	*P*-value
24 hour SBP (mmHg)	123.0 ± 14.5	113.6 ± 8.6	141.0 ± 3.4	<0.0001
24 hour DBP (mmHg)	70.1 ± 7.6	65.8 ± 5.4	78.6 ± 4.9	<0.0001
24 hour PP (mmHg)	53.0 ± 9.3	47.9 ± 6.9	62.3 ± 5.8	<0.0001
Daytime SBP (mmHg)	124.4 ± 14.3	115.4 ± 8.1	141.4 ± 4.7	<0.0001
Daytime DBP (mmHg)	71.7 ± 7.7	67.7 ± 5.0	80.0 ± 5.6	<0.0001
Daytime PP (mmHg)	52.7 ± 9.3	47.7 ± 6.7	61.4 ± 6.2	<0.0001
Nighttime SBP (mmHg)	120.5 ± 16.3	110.6 ± 11.4	140.3 ± 7.4	<0.0001
Nighttime DBP (mmHg)	67.0 ± 8.9	62.3 ± 7.1	75.9 ± 6.2	0.002
Nighttime PP (mmHg)	54.3 ± 10.4	49.9 ± 9.4	64.7 ± 6.3	0.001

Abbreviations: SBP, systolic blood pressure; DBP, diastolic blood pressure; PP, pulse pressure.

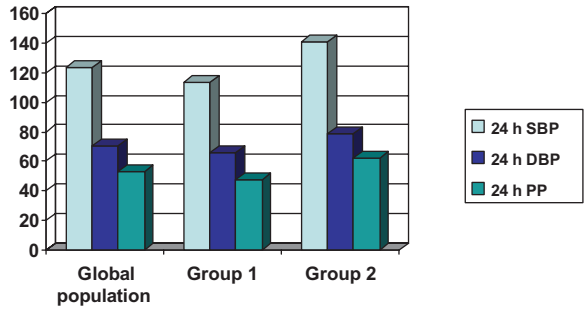

Figure 1 24 hour blood pressure difference between the two groups. Group 1: patients reach blood pressure target; Group 2: uncontrolled patients.
Abbreviations: SBP, systolic blood pressure; DBP, diastolic blood pressure; PP, pulse pressure.

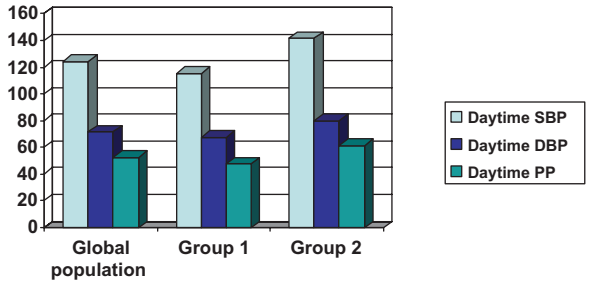

Figure 2 Daytime BP difference between the two groups. Group 1: patients reach blood pressure target; Group 2: uncontrolled patients.
Abbreviations: SBP, systolic blood pressure; DBP, diastolic blood pressure; PP, pulse pressure.

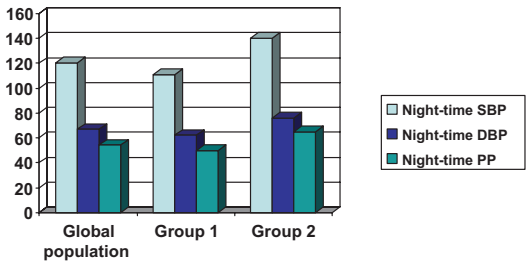

Figure 3 Night-time BP difference between the two groups. Group 1: patients reach blood pressure target; Group 2: uncontrolled patients.
Abbreviations: SBP, systolic blood pressure; DBP, diastolic blood pressure; PP, pulse pressure.

Strict BP control is an essential step in the management of these patients during the acute phase. However, little data has been reported on the proper control of BP during the chronic phase.

In our series, the diaphragmatic and subrenal aortic diameters were associated with BP control. Patients with a smaller diameter were better controlled. The benefits of BP decrease after dissection is assumed by the law of La Place, according to which the parietal tension of a vessel is the product of intravascular pressure and diameter. By lowering the systemic BP, the risk of aneurysmal evolution of the aorta would also decrease. Our results suggest that poorly controlled patients are at risk of developing an aneurysmal complication.

24 hour BP monitoring after AD

In 2005, Eggebrecht reported that poor BP control was associated with a younger age and a higher BMI in a population of 40 patients with chronic AD (38 patients suffered from Type B dissection, and two patients suffered from Type A dissection).[9] This work was based on measurements repeated on three consecutive days. In this series, the need for at least five antihypertensive drugs in 75% of the patients was already noted. Our study is based on a more reliable assessment of BP by using 24 hour monitoring. However, our results do not confirm previous published results. Indeed, in our series, the BMI and level of abdominal obesity of patients had no influence on BP control, and poorly controlled patients were on average older (NS).

The simple measure of BP at discharge was statistically associated with poor BP control in the chronic phase ($P = 0.01$ for systolic BP and 0.08 for diastolic BP). We noticed that the statistical significance was greater for systolic than for diastolic BP. Pulse pressure at discharge

Table 4 Therapeutics and number of antihypertensive treatments on discharge

Discharge treatment	Overall population (n = 26)	Group 1 Controlled blood pressure (n = 17)	Group 2 Uncontrolled blood pressure (n = 9)	*P*-value
PAA (n, %)	13 (50)	8 (47.0)	5 (55.5)	0.68
ACEi/ARA2 (n, %)	26 (100)	17 (100)	9 (100)	–
Beta-blockers	26 (100)	17 (100)	9 (100)	–
Calcium inhibitor (n, %)	23 (88.5)	15 (88.2)	8 (88.8)	0.96
Statin (n, %)	15 (57.7)	9 (52.9)	6 (66.6)	0.51
VKA (n, %)	5 (19.2)	4 (23.5)	1 (11.1)	0.45
Diuretic (n, %)	17 (55.4)	9 (52.9)	8 (88.8)	0.07
Number of antihypertensive drugs (different classes)	4.3 ± 0.9	4.2 ± 0.75	4.6 ± 0.96	0.07

Abbreviations: PAA, antiplatelet agents; ACEi, angiotensin-converting enzyme inhibitors; ARA2, antagonists of the angiotensin receptor Type 1 and Type 2; VKA, vitamin K antagonist.

was almost significantly higher, and pulse pressure during the 24 hour monitoring was also greater (Figures 2 and 3). These elements suggested that poorly controlled patients might have a greater arterial rigidity. This hypothesis is also supported by the fact that patients with vascular disease were already at risk of poor BP control. Arterial rigidity is known to be a risk marker for the development of cardiovascular diseases. This correlation underlines the importance of the cardiovascular field's intervention. The main etiology of the dissection of the descending aorta was atherosclerosis.

Measuring BP upon discharge is insufficient when trying to estimate a BP control after an AD. Twenty-four hour BP monitoring appears to be a critical tool for the monitoring of these patients. It allows avoiding "masked" high arterial BP and the "white coat" effect that are only diagnosed with ambulatory measures. It is difficult to identify because it is associated with a target therapeutic BP on consultation and pathological values of ambulatory BP, making it hard to determine whether the patient needs to be treated. Ambulatory measures are thus even more critical in this context, since poorly controlled patients had the target at-rest blood pressure before discharge. It seems legitimate to propose the ambulatory monitoring of BP, both to prevent the risk of a poor AD evolution (ectasia, evolution of the false lumen, extension of the dissection, aortic rupture) and for secondary cardiovascular prevention.

How to reach the blood pressure levels target

Thirty four percent of our population had an uncontrolled BP, despite antihypertensive treatment, with an average of five different antihypertensive classes used. This data is comparable to the Eggebrecht series of 2005,[9] in which 40% of patients had resistant hypertension despite the combination of at least five antihypertensive drugs. In 1995, on this same population, Grajek[19] showed that 75% of patients had resistant hypertension with an average grade 3, and those patients were then processed on average by 3.1 antihypertensive drugs, of which only 10% received more than five antihypertensive drugs. This combination of antihypertensive drugs incremented under monitoring as suggested by the current guidelines on hypertension, appear to be a worthy strategy. One hundred percent of our patients were treated

Table 5 Morphological data of Type B AD at discharge

Morphological data	Overall population (n = 26)	Group 1 Controlled blood pressure (n = 19)	Group 2 Uncontrolled blood pressure (n = 7)	*P*-value
Diameter of the ascending aorta (mm)	37.1 ± 5.3	33.8 ± 3.5	40.0 ± 6.4	0.39
Diameter of the descending aorta (mm)	39.4 ± 5.7	41.4 ± 6.3	37.4 ± 2.9	0.10
True lumen: descending (mm)	20.6 ± 6.8	21.7 ± 7.4	19.5 ± 7.1	0.34
False lumen: descending (mm)	18.3 ± 6.8	19.4 ± 6.9	17.0 ± 6.1	0.90
Diameter of the diaphragmatic aorta (mm)	35.2 ± 5.8	33.9 ± 3.5	40.0 ± 6.4	0.02
Subrenal aorta (mm)	26.2 ± 7.7	24.2 ± 4.4	30.2 ± 3.9	0.05
Dissected peripheral artery (n, %)	13 (50)	8 (47.0)	5 (55.5)	0.64

Abbreviation: AD, aortic dissection.

with beta-blockers and inhibitors of the renin-angiotensin system at hospital discharge and 88% of them were treated with a calcium channel blocker. Patients who presented with AD should be considered as patients with very high cardiovascular risk. The European recommendations state that these patients require at least an antihypertensive biotherapy (in addition to a specific beta-blocker therapy), and they advise to treat first with the combination of renin-angiotensin system blockers with dihydropyridine, ideally in the form of a fixed combination for better adherence. If a complementary therapy is required, a thiazide diuretic should be added to the combination.[20] In this regard, our data is consistent with the treatment strategy proposed by these latest recommendations and confirm that at least three antihypertensive drugs are needed to control BP in hypertensive patients at very high cardiovascular risk. Nighttime blood pressure is also much higher in the group of poorly controlled patients, whereas it remained under 120/70 mmHg in the group of controlled patients. This supra physiologic notion of nighttime blood pressure should be considered for systemic search of sleep apnea syndrome in these patients. The prevalence of abdominal obesity and metabolic syndrome in our population encourages this approach. The prevalence of obesity in our population, as in the population described by Eggebrecht et al, reinforces the idea of establishing strong educational supports in addition to the drug strategy. This notion remains prioritary in the management of blood pressure, even if uncomplicated, and the management of obstructive sleep apnea syndrome.

The results of our study present several limitations. The low prevalence of AD and poor short-term prognosis could explain the limited size of our monocentric series. Because of its observational nature, we can only propose possible prognosis benefits. Data on the pre-hospital phase, such as duration and severity of hypertension, have not been taken into account. This work focused on measuring BP and did not take into account other parameters such as adherence to treatment, nor did we screen for secondary causes, such as sleep apnea syndrome. However, all patients were regularly followed-up at our practice, specializing in hypertension and vascular disease.

Conclusion

BP control is critical after an AD. A high BP and a very high cardiovascular risk are predictors of poor BP control. The prescription of a combination of different antihypertensive drugs classes at discharge and the use of ambulatory measures could lead to an improvement of BP control and potentially improve the general and vascular prognosis of AD patients.

Disclosure

The authors report no conflicts of interest in this work.

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