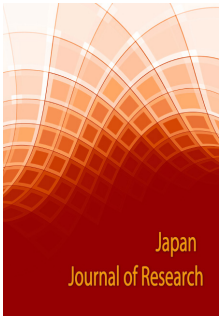
Japan Journal of Research

Case Report



Analysis of arterial stiffness: a further look at hypertension

Gilberto Campos Guimarães Filho[1,2*]; Pedro Guimarães Cunha[3,4,5]; Ana Luiza Lima Sousa[1,2]; Sayuri Inuzuka[1,2]; Adriana Camargo Oliveira[1,2]; Maria Emília Figueiredo Teixeira[1,2]; Priscila Valverde de Oliveira Vitorino[6]; Weimar Kunz Sebba Barroso[1,2]

[1]*Hypertension League, Federal University of Goias, Brazil*

[2]*Health Sciences in Postgraduate Program - Federal University of Goias, Brazil*

[3]*Center for the Research and Treatment of Arterial Hypertension and Cardiovascular Risk, Serviço de Medicina Interna do Hospital da Senhora da Oliveira, Guimarães; Brazil*

[4]*Life and Health Sciences Research Institute (ICVS), School of Medicine, University of Minho, Braga, Portugal;*

[5]*ICVS/3B's, PT Government Associate Laboratory, Braga/Guimarães, Portugal*

[6]*Pontifical Catholic University of Goiás, School of Social and Health Sciences, Brazil*

Correspondence

Gilberto Campos Guimarães Filho.

Professor of Cardiology, Faculty of Medicine, Federal University of Goiás, Brazil

E-mail: camposguimaraes@yahoo.com.br

- Received Date: 22 Feb 2021
- Accepted Date: 03 Mar 2021
- Publication Date: 10 Mar 2021

Introduction

An estimated 18 million deaths per year worldwide from cardiovascular disease (CVD), one third of all deaths. 10.4 million related to arterial hypertension (AH) [1]. AH is the main modifiable cause of cardiovascular morbidity and mortality in the adult population and a risk factor (RF) independent of CVD [1].

The use of methods that provide early identification of changes and cardiovascular needs can improve the treatment and control strategy of these patients [2].

The consolidation of the PWV (pulse wave velocity) measurement in the assessment of arterial stiffness has led several studies to demonstrate the association of this phenotype with the risk of developing different manifestations of CVD [3], constituting the gold standard due to reproducibility and confidence method and association, mainly, with cardiovascular risk in different groups [3].

The changes that an antihypertensive agent produces in arterial stiffness can be independent of pressure, directly affecting the arterial wall through remodeling of the elastic network and collagen fiber or dependent on pressure, occurring indirectly through the reduction of blood pressure (BP) and cardiovascular outcomes, when therapy is corrected early [3].

Case report

Sixty five years old, female, married, HA for 18 years, 4 years with Panic Syndrome, regular use of Candesartan 8mg, Fluoxetine 20mg and Alprazolan 0.5mg. She sought the cardiologist in June 2016, with tachycardia, palpitations, headache and ringing in the ear. Normal physical examination. BP = 172x104mmHg-right arm (MSD) and BP = 176x104mmHg-left arm (MSE) with an interval of 2 minutes between measurements, digital device, oscillometric, OMRON-M7. 69kg, 1.62m, body mass index (BMI) = 26.3kg/m^2, waist circumference (WC) = 82cm, 88bpm.

Electrocardiogram (ECG), Echocardiogram and Doppler of normal carotids. Normal biochemists, except LDLc = 142mg / dl and CRP-us = 16.

In front of the table, the dose of Candesartan was doubled to 16mg, Rosuvastatin 10mg added and a healthy lifestyle oriented. 1 month later, outpatient BP monitoring (ABPM) was performed, within the normal range (Means: Wake BP = 125x77mmHg; sleep BP = 113x70mmHg) with the proposed treatment.

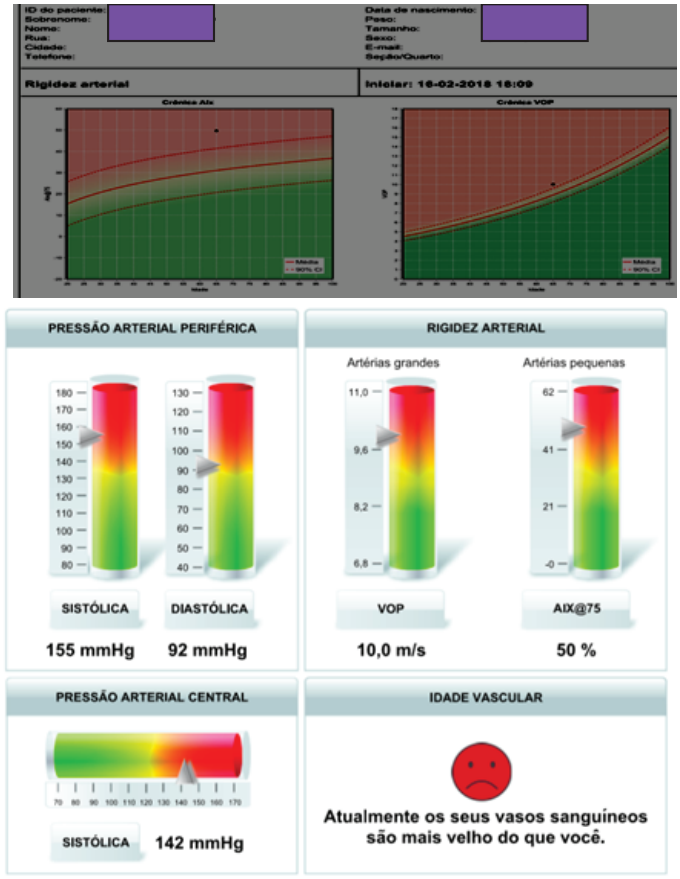
The patient returned in October 2017, presenting the same symptoms, physical and anthropometric exams as the first consultation. BP = 184x88mmHg-MSE. IN REGULAR USE: Candesartan 16mg/day. A new ABPM was performed (Mean: Wake BP = 118x72mmHg; Sleep BP = 108x66mmHg) and normal ECG. Suggested effect of the white coat or panic crisis, due to the suspension of Fluoxetine, reintroduced it and reinforced a healthy lifestyle.

In February 2018, he returned with maintenance of symptoms, normal physical examination, BP = 134x78mmHg. Echocardiogram with LV diastolic dysfunction grade I (altered ventricular relaxation pattern compared to the first exam), with regular use of medications. Measurement of the central arterial, oscillometric parameters, Mobil O'Graph model (Figure 1): central BP = 142mmHg, PWV = 10m / sec, augmentation index (AI) = 50%.

Due to the elevated central measures, he reduced Candesartan to 8mg and added Felodipino-2.5mg. Drug combination capable of reducing central parameters 4.

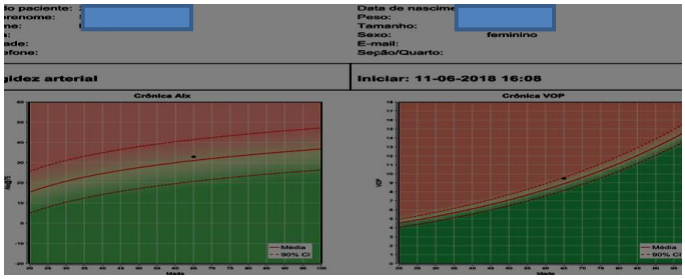
In order to rule out hypotension after a drug combination, a new ABPM was performed after 1.5 months, showing: Means = BP Wake = 114x74mmHg; BP Sleep period = 107x64mmHg.

Citation: Filho GCG, Cunha FG, Sousa ALL, et al. Analysis of arterial stiffness: a further look at hypertension. Japan J Res. 2021;2(1):1-3



AIX = augmentation index ; PWV pulse wave velocity

Figure 1. Mobil O'Graph-central parameters



AIX = augmentation index ; PWV pulse wave velocity

Figure 2. Mobil O'Graph-central parameters

Three months later, the patient returned asymptomatic, regularly using the medications and presenting new measurements of the central parameters within the normal range (Figure 2). There was a 34% reduction in AI and 9.9% in central BP. (Central BP = 128mmHg; PWV = 9.5m/sec; AI = 33%).

Discussion

When treating a patient with AH, optimal BP control, risk factors, changes in target organs and associated clinical diseases should be valued, stratifying cardiovascular risk and quantifying prognosis.

The treatment of AH depends on the choice of the drug, based on the effectiveness of BP reduction and cardiovascular morbidity and mortality [1].

There is now clear evidence indicating that central hemodynamic measures, by non-invasive methods, have a precise relationship with the pathophysiology of cardiovascular diseases, thus showing a good correlation between central parameters and the clinic, as they present less pressure variability and reflect the levels of tension in target organs, in addition to the benefit of reducing the use of antihypertensive drugs when the follow-up was performed by central BP compared to peripheral BP [5].

The case presented here reports an unusual phenomenon. Patient being treated with an angiotensin 2 AT1 receptor blocker, showing good pressure control evidenced, at 2 different times, through ABPM. However, the presence of subjective symptoms impaired her quality of life, leading her to repeated visits to the cardiologist and psychiatrist, under the suspicion of panic syndrome.

Studies suggest that central BP is the most important determinant of cardiovascular risk reduction than peripheral BP. In addition, it was observed that vasodilating drugs, such as angiotensin-converting enzyme inhibitors, angiotensin II receptor blockers and calcium channel blockers, could contribute to both the reduction of central systolic BP and the regression of left ventricular hypertrophy, mainly through reduction in the magnitude of wave reflections [6].

Studies comparing measurements of central parameters by oscillometry (Mobil O Graph® for example) with the tonometric method, showed agreement between the methods of central parameters, obtained by tonometry with oscillometry [7]. In another study with 89 patients, the authors also reported good reproducibility between methods and suggested that oscillometry be considered in everyday clinical practice, as it is an easy-to-perform, cost-effective test [8,9].

Based on information obtained from studies, such as the one mentioned above, the conducts were determined as well as the best therapeutic strategies for the patient. Thus, central hemodynamic measurements were performed that showed an increase in PWV, AI and central BP, even with the presence of ABPM within normal limits.

This information led us to use drugs that best respect the patient's metabolic profile, with pleiotropic action and rapid reduction in peripheral BP and central hemodynamic profile, offering greater protection in target organs.

Target organ damage caused by HA outside of targets is more precisely established in the heart, brain and kidney. The aggression to these organs occurs by the elevation of central BP [10]. The CAFE study showed the same, by showing equivalence in the reduction of peripheral BP in both groups (Anlodipino and Atenolol), however in the group treated with Atenolol there were greater cardio and cerebrovascular outcomes. by not reducing central BP [5].

In view of the situation, non-drug treatment was reinforced and Felodipino, a vasodilator was added with a significant reduction in the intensity and precocity of the reflected pulse wave and a consequent improvement in central hemodynamics. After 4 months, a new central measurement was performed, a significant reduction in central BP was observed (142 to 128mmHg), AI (50 to 33%) and normalization of PWV (<10m/sec), with improved quality of life.

Conclusion

AH is closely related to increased cardio and cerebrovascular events with reduced life expectancy [1]. The treatment of AH, based on the reduction of central parameters and peripheral BP, improves quality of life and survival, reducing CV morbidity and mortality and allowing for more dignified aging.

References

1. International Society of Hypertension Global Hypertension Practice Guidelines.Hypertension. 2020;75:1334–1357.
2. Pizzi OL, Brandão AA, Magalhães ME, Pozzan R, Brandão AP. Pulse wave velocity – methodology and prognostic implications in hypertension. Rev Bras Hipertensvol. 2006;13(1):59-62.
3. Vlachopoulos C, Xaplanteris P, Aboyans V, et al. The role of vascular biomarkers for primary and secondary prevention. A position paper from the European Society of Cardiology Working Group on peripheral circulation: Endorsed by the Association for Research into Arterial Structure and Physiology (ARTERY) Society. Atherosclerosis. 2015;241(2):507-32.
4. Williams B, Lacy PS, Thom SM, et al. CAFE Investigators; Anglo-Scandinavian Cardiac Outcomes Trial Investigators; CAFE Steering Committee and Writing Committee. Differential impact of blood pressure-lowering drugs on central aortic pressure and clinical outcomes: principal results of the Conduit Artery Function Evaluation (CAFE) study. Circulation. 2006;113(9):1213-25.
5. Gurovich AN, Braith RW. Pulse wave analysis and pulse wave velocity techniques: Are they ready for the clinic? Hypertens Res. 2011;34:166-9.
6. Matsui Y, Kario K. Differential Impacts of Antihypertensive Drugs on Central Blood Pressure and Their Clinical Significance. Current Hypertension Reviews. 2012; 8:114-119.
7. Schultz M, Qasem A, Peng X, et al. P7.16: Validation of na oscillometric brachial cuff method to derive central blood pressure using diferente calibration modes. Artery Research. 2015; 12:C, 33–33.
8. Barroso WKS, Gonçalves CF, Berigó JAC, Melo MA, Arantes AC, Lelis ES. Tonometric and Oscillometric Methods for Measurement of Central Blood Pressure Parameters: a Comparison in Patients with Borderline Hypertension or Stage 1 Hypertension. ijcs. 2020; 33(2):145-150.
9. Reshetnik A, Gohlisch C, Tölle M, Zidek W, Van Der Giet M. Oscillometric assessment of arterial stiffness in everyday clinical practice. Hypertens Res. 2016;40(2):1-6.
10. Protogerou AD, Argyris AA, Papaioannou TG, et al. Left-ventricular hypertrophy is associated better with 24-h aortic pressure than 24-h brachial pressure in hypertensive patients: the SAFAR study. J Hypertens. 2014;32(9):1805-14.