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Research Article

Onset of hypertension during pregnancy is associated with long-term worse blood pressure control and adverse cardiac remodeling

Roberto F. Mesquita, MD, MSc[a,1], Muriel Reis, MD[a,1], Ana Paula Beppler, MD[a], Vera Regina Bellinazzi, MD, PhD[a], Sandra S. Mattos, MD, PhD[b], José L. Lima-Filho, MD, PhD[b], José A. Cipolli, MD, PhD[a], Otavio R. Coelho-Filho, MD, PhD[a], José A. Pio-Magalhães, PhD[a], Andrei C. Sposito, MD, PhD[a], José R. Matos-Souza, MD, PhD[a], and Wilson Nadruz Jr., MD, PhD[a,b,*]

[a]*Department of Internal Medicine, University of Campinas, Campinas, SP, Brazil; and*
[b]*Laboratory of Immunopathology Keizo Asami, Federal University of Pernambuco, Recife, PE, Brazil*

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Abstract

Up to 20% of women with hypertensive pregnancy disorders might persist with chronic hypertension. This study compared clinical and echocardiographic features between women whose hypertension began as hypertensive pregnancy disorders (PH group) and women whose diagnosis of hypertension did not occur during pregnancy (NPH group). Fifty PH and 100 NPH women were cross–sectionally evaluated by clinical, laboratory, and echocardiography analysis, and the groups were matched by duration of hypertension. PH exhibited lower age (46.6 ± 1.4 vs. 65.3 ± 1.1 years; $P < .001$), but higher systolic (159.8 ± 3.9 vs. 148.0 ± 2.5 mm Hg; $P = .009$) and diastolic (97.1 ± 2.4 vs. 80.9 ± 1.3 mm Hg; $P < .001$) blood pressure than NPH, although used more antihypertensive classes (3.4 ± 0.2 vs. 2.6 ± 0.1; $P < .001$). Furthermore, PH showed higher left ventricular wall thickness and increased prevalence of concentric hypertrophy than NPH after adjusting for age and blood pressure. In conclusion, this study showed that PH may exhibit worse blood pressure control and adverse left ventricular remodeling compared with NPH. J Am Soc Hypertens 2014;8(11):827–831.

Keywords: Echocardiography; concentric hypertrophy; hypertensive pregnancy disorders; left ventricular.

Introduction

Hypertensive pregnancy disorders, namely gestational hypertension and pre–eclampsia, occur in 5%–7% of all pregnancies and are recognized causes of maternal and fetal complications.[1] Gestational hypertension is defined as the development of hypertension after 20 weeks of gestation, while women with the same pattern of elevated blood pressure (BP) and proteinuria (>300 mg protein/24-hour urine collection) are considered to present pre–eclampsia.[2]

Epidemiologic data have shown that women with a history of pregnancy–induced hypertension are at increased risk of developing hypertension and metabolic alterations later in life in comparison with women without a history of pregnancy–induced hypertension.[3–5] However, up to 20% of women with hypertensive pregnancy disorders might persist with high BP levels after 6 weeks postpartum, thus developing chronic hypertension.[6–8] Hitherto it remains unknown whether women who developed chronic hypertension starting as hypertensive pregnancy disorders present adverse long–term cardiovascular features among hypertensive women. In this study, we enrolled a sample

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*Corresponding author: Wilson Nadruz Jr., MD, PhD, Departamento de Clínica Médica, Faculdade de Ciências Médicas, Universidade Estadual de Campinas, Cidade Universitária "Zeferino Vaz", 13081-970 Campinas, SP, Brasil. Tel/Fax: (55) (19) 3521 7836.

E-mail: wilnj@fcm.unicamp.br

[1] Both authors contributed equally to this study.

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of women with chronic hypertension referred to a university hospital outpatient clinic and evaluated whether those whose hypertension began as hypertensive pregnancy disorders and persisted afterwards (PH group) exhibited clinical and echocardiographic differences in comparison with those whose diagnosis of hypertension did not occur during pregnancy (NPH group).

Methods

Study Population

Between 2005 and 2011, we evaluated a total of 638 hypertensive women who were referred to the hypertension unit of the University Hospital of the State University of Campinas, which is an outpatient clinic aimed to evaluate and treat subjects with end–organ damage and uncontrolled hypertension. All subjects were systematically asked about the onset of hypertension using questionnaires and were cross–sectionally evaluated by clinical, laboratory, and echocardiography analysis. Inclusion criteria in the study were age >18 years and no evidence of moderate or severe cardiac valve disease, hypertrophic cardiomyopathy, previous myocardial infarction, neoplastic disease, and secondary cause of hypertension. Fifty women stated that hypertension began after 20 weeks of pregnancy and persisted afterwards (PH group). Seven other women reported that hypertension onset took place before 20 weeks of pregnancy and therefore were not included in the study. Among PH women, 14 declared that hypertension began with the diagnosis of pre–eclampsia/eclampsia, 30 stated that hypertension did not begin with pre–eclampsia/eclampsia, and 6 did not know whether the onset of hypertension was associated with pre–eclampsia/eclampsia. The NPH group comprised hypertensive women who stated that hypertension onset did not occur during pregnancy or within 1 year after the delivery. Each PH woman was compared with two NPH women, and the PH and NPH groups were matched by duration of hypertension. Among NPH women, 93% stated that they had at least one pregnancy. Written informed consent was obtained from each patient, and the study protocol conforms to the ethical guidelines of the 1975 Declaration of Helsinki. This study was approved by the Human Research Ethics Committee of the University of Campinas.

BP was measured using a validated digital oscillometric device (Omron HEM-705CP, Omron Healthcare, Kyoto, Japan) with appropriate cuff sizes. Two readings were averaged, and, if they differed by more than 5 mm Hg, one additional measurement was performed and then averaged. Body mass index was calculated as body weight divided by height squared. Blood samples were obtained in the morning after 12 hours of fasting for analysis of cholesterol fractions, triglycerides, glucose, C-reactive protein, and creatinine levels. Albuminuria was evaluated in patients by measuring the albumin–creatinine ratio in morning urine samples. Hypertension was defined as systolic BP ≥140 mm Hg or diastolic BP ≥90 mm Hg or current antihypertensive medication use. Diabetes mellitus was diagnosed if fasting blood glucose was ≥126 mg/dL or when participants were taking hypoglycemic medications.

Echocardiography

Echocardiography studies were conducted on each subject at rest in the left lateral decubitus position using a Vivid 3 Pro (General Electric, Milwaukee, WI, USA) apparatus equipped with a 2.5–MHz transducer as previously described.[9,10] Aortic root and left atrial diameter as well as left ventricular (LV) dimensions were measured in accordance with the American Society of Echocardiography guidelines.[11] All recordings were made by the same physician, who was unaware of other data relating to the subjects. LV mass index was calculated as LV mass divided by body surface area. Body surface area was estimated by the DuBois formula. LV hypertrophy was defined with the use of cutoff point LV mass index >110 g/m^2,[12] whereas concentric geometry was considered if the relative wall thickness was ≥0.45.[13] The reproducibility of both acquiring and measuring LV mass was determined in recordings obtained from 10 subjects. Intraobserver LV mass variability was <8%.

Statistical Analysis

Data were analyzed using SPSS 15.0. Continuous normal and non–normal variables are presented as mean ± standard error and median (25th–75th percentile), respectively. The Kolmogorov–Smirnov test was used to test for normal distribution of the variables. Chi–squared test was used to compare categorical variables, whereas the unpaired *t*–test and Mann–Whitney test compared normal and non–normal continuous variables, respectively. Two–way analysis of covariance was used to assess intergroup difference in low–density lipoprotein cholesterol levels adjusted for statins use. Linear and logistic multivariate analyses were used to assess intergroup differences in echocardiographic parameters adjusted for age and diastolic blood pressure. A *P*–value <.05 was considered significant.

Results

Clinical and laboratory features of PH and NPH groups are presented in Table 1. In comparison with the NPH group, PH women exhibited lower age, but presented higher systolic and diastolic BP levels, although they used a higher number of antihypertensive classes. The PH group also exhibited higher low–density lipoprotein cholesterol levels than the NPH one, although this difference did not remain statistically significant after adjustment for statin use.

Echocardiographic features of the studied women are presented in Table 2. Unadjusted comparisons showed