

# Perinatal outcomes, blood pressure patterns and risk assessment of superimposed preeclampsia in mild chronic hypertensive pregnancy

Stefano Raffaele Giannubilo\*, Bernardo Dell'Uomo, Andrea L. Tranquilli

Department of Obstetrics and Gynecology, Marche Polytechnic University at Salesi Hospital, Via Corridoni 11, 60123 Ancona, Italy

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## Abstract

**Objective:** Assessment of perinatal outcomes, blood pressure (BP) patterns and risk of superimposed preeclampsia in a population with mild chronic hypertension.

**Study design:** We investigated 223 pregnant women with mild chronic hypertension and 200 controls. Twenty-four-hour BP monitoring longitudinally in pregnancy and Doppler assessment of uterine arteries at 24 weeks' gestation were performed. Perinatal outcomes were recorded.

**Results:** Superimposed preeclampsia occurred in 28.4% of hypertensive women, with an increased rate of small-for-gestational age babies (30.7% versus 8.9%), a lower birth weight ( $2587.75 \pm 832.97$  versus  $3167.35 \pm 536.3$ ;  $p < 0.001$ ) and a higher rate of caesarean sections (69.2% versus 35.5%) than controls. According to the ROC curve, the mean 24-h blood pressure (diastolic 78 mmHg [S.E.: 0.95; SP: 0.89] and systolic 121 mmHg [S.E.: 0.88; SP: 0.92]) and the mean resistance index of the uterine arteries of 0.52 (S.E.: 0.69; SP: 0.87) are better prognostic values for predicting superimposed preeclampsia.

**Conclusions:** In women with chronic hypertension in the second trimester 24-h blood pressure monitoring and Doppler velocimetry of the uterine artery are able to detect those at risk of superimposed preeclampsia. In women with circulatory adaptation to pregnancy, a good perinatal outcome is expected with proper obstetric care.

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**Keywords:** Pregnancy; Chronic hypertension; Preeclampsia; Doppler

## 1. Introduction

Chronic hypertension in pregnancy is associated with serious maternal and foetal complications, including premature birth, foetal growth restriction (FGR), foetal demise, placental abruption and caesarean delivery [1]. These complications have a substantial economic impact, including costs of monitoring and treating sick mothers and neonates.

According to the National High Blood Pressure Program Working Group on High Blood Pressure in Pregnancy, chronic hypertension is defined as hypertension present

before the 20th week of pregnancy or hypertension present before pregnancy [2]. Chronic hypertension in pregnancy can be classified as mild or severe; cut-offs commonly used to define "severe" are blood pressure (BP) of 160/110 mmHg or higher in the American literature and 170/110 mmHg or higher in the European literature [3].

The incidence of chronic hypertension in pregnancy ranges from 0.5 to 5%, depending on the populations studied and the diagnostic criteria used. At the University of Tennessee, Memphis, where the referral area is large and 80% of the obstetric patients are indigent Blacks, the incidence is 3.4% [2,4].

It is often difficult to diagnose chronic hypertension in pregnant women who do not have documented prepregnancy blood pressure recordings. In these cases, the presence of

\* Corresponding author. Tel.: +39 0715962214; fax: +39 07136575.

E-mail address: [s.giannubilo@univpm.it](mailto:s.giannubilo@univpm.it) (S.R. Giannubilo).

elevated blood pressure before 20 weeks' gestation establishes the diagnosis and hypertension should be documented on more than one occasion and at Korotkoff phase V [2]. Nevertheless, the ambulatory blood pressure monitoring for 24 h (ABPM 24-h) is considered to be a useful clinical method for defining more precisely BP elevation by the elaboration of several parameters such as mean, mesor and circadian rhythm.

Several physiologic changes occur in pregnancy that may modify the natural history of chronic hypertension, such as the increase in blood volume or the increased renal function, but the most important occurrence in pregnancy is the trophoblastic invasion of the spiral arteries at 16–18 weeks, which leads to a physiologic blood pressure decrease in normal pregnancy. This phenomenon of maternal “adaptation” to pregnancy may influence the natural course of both hypertension and pregnancy. Chesley and Annitto [5] observed that chronic hypertensives demonstrate greater decreases in blood pressure during pregnancy than normotensives, and that blood pressure was within the normal range in the second trimester in women who were severely hypertensive before pregnancy. In addition, Sibai et al. [6] found that blood pressure was within the normal range during the second trimester in 49% of 211 women who had mild chronic hypertension during pregnancy. The majority of these women subsequently developed increased blood pressure during the third trimester, and thus may be erroneously diagnosed as having pregnancy-induced hypertension. This change can mask either the course or the detection of chronic hypertension in early pregnancy [1]. On the other hand, superimposed preeclampsia in pregnancy complicated by chronic hypertension may be associated with worsening or malignant hypertension, central nervous system haemorrhage, cardiac decompensation and renal failure. The reported incidence of superimposed preeclampsia in patients with chronic hypertension ranges from 4.7 to 52% [5,7,8].

On the basis of evaluation before conception, women with chronic hypertension can be divided into “high-risk” or “low-risk” by the presence or absence of organ involvement. The management of low-risk chronic hypertension is still controversial and the role of antihypertensive therapy in pregnancy outcomes is particularly uncertain.

The primary outcome of this study was to determine the perinatal outcomes in a population of pregnant women with low-risk chronic hypertension; the secondary aim was to investigate the parameters useful in identifying chronic hypertension pregnancies at risk of superimposed preeclampsia and adverse perinatal outcomes.

## 2. Materials and methods

We investigated two groups of singleton pregnancies: 223 patients with mild chronic hypertension (study group) consecutively seen at the Department of Obstetrics and

Gynecology of Marche Polytechnic University, Ancona (Italy), over a four-year period (1999–2003), and all normotensive women with normal singleton pregnancies were enrolled consecutively in the same period, and matched for maternal age and parity. In this group, 200 women were selected who were and remained normotensive and had a physiologic pregnancy until delivery (control group). The inclusion criteria for the study group were: White race and systolic blood pressure  $\geq 140$  mmHg or diastolic blood pressure  $\geq 90$  mmHg (Korotkoff phase V), but not higher than 160/110 mmHg, on at least two occasions measured at least 4 h apart, in a woman known to suffer from chronic hypertension before pregnancy, but without organ damage. The blood pressure was assessed at the first post-conceptional examination (6–12 weeks), taken with the patient sitting up and the arm at the level of the heart, using standard mercury sphygmomanometers with appropriately sized cuffs. In the chronic hypertensive group, the antihypertensive therapy was begun, or modified if present, at the first visit: labetalol (100 mg  $\times$  2 per die p.o.) was the first choice and calcium antagonist (nifedipine 4 mg  $\times$  2 per die p.o.) was added when necessary. In all the hypertensive patients, the same therapeutical profile was adopted in terms of therapy, diet and clinical assistance by internal protocol for chronic hypertension in pregnancy.

All the women included were investigated using 24-h ambulatory blood pressure monitoring during pregnancy (first, second and third trimesters), post-partum and 6 weeks after; Doppler velocimetry of the uterine arteries was performed at 24 weeks' gestation.

Twenty-four-hour blood pressure was recorded at 30-min intervals by oscillometry (SpaceLabs 90207; SpaceLabs, Inc., Redmond, WA, USA), the cuff size was adapted to the upper arm circumference before starting the system, and patients were asked to continue normal daily activities during the following 24 h. The instrument is self-calibrating, so that measurements with artefacts are repeated after 2 min. Through this control, the percentage of correct readings is constantly above 96%. The mean 24-h diastolic and systolic blood pressure measurements were chosen to describe the whole maternal pressure regimen.

Flow velocity waveforms of the right and left uterine arteries were obtained at the 24th week of gestation with an Aloka SSD 1700 instrument, using a 3.5-MHz convex probe and a 100-Hz filter. The resistance index (RI) was calculated for both arteries; when greater than 0.58, the average was considered abnormal. The examinations were carried out by the same operator (S.R.G.) who was masked to the subject's study group status.

For each woman, the following data were recorded: mother's age, parity, gravidity, obstetric history, weight and cause of the hypertension (essential or other); data related to the current pregnancy—smoking habits, superimposed preeclampsia (defined, according to National High Blood Pressure Education Program (NHBPEP) criteria [2], by new onset proteinuria of 300 mg or greater in a 24-h specimen, or

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