

Effects of Air Pollution on Blood Pressure of Pregnant Women

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Abstract

The aim of our study is to evaluate effects on blood pressure of pregnant women exposed to air pollution. The subjects were 654 pregnant women, aged 25-40 years, living for more than five years in the same home. The exposed group of pregnant women (n=348) live in an urban area with a high level of air pollution, while the pregnant women (n=306) in the comparison group, designed as a non-exposed group, live in an area with a lower level of air pollution. The air concentrations of black smoke, sulfur dioxide, and lead in sediment matter were determined in the period from 2004 to 2008. The diagnosis of high blood pressure is made using pre-defined criteria. No effect of air pollution exposure on pregnancy-induced hypertension was found in multivariate logistic regression analysis ($p > 0.05$; 95% CI: 0.937-2.219). However, further research using more rigorous measures of exposure to air pollution as well as additional measures of blood pressure in pregnancy are needed to confirm the relationships.

Keywords: air pollution, pregnant women, blood pressure

Introduction

Long-term and short-term air pollution exposure can cause numerous symptoms and diseases. Studies conducted in a wide range of cities with contrasting levels of air pollution have reported such effects as increased mortality and morbidity from different causes and in various age groups [1-3]. There are also indications that these effects are higher on sensitive groups of the population, such as children, women in the generative age, the elderly, and persons with chronic diseases.

More recently, many epidemiological studies suggest possible links between acute or chronic exposure to air pollutants and cardiovascular disease [4-6]. Although the mechanism of air pollutants' influence on the cardiovascular system has not yet been fully explained, there is some evidence that exposure to air pollution can cause hypertension and arrhythmia, and also increase hospitalization due

to ischemic heart disease. The putative biological mechanisms linking air pollution to heart disease involve pulmonary oxidative stress, inflammation, and disturbance of cardiac autonomic control [7, 8].

Pregnancy-induced hypertension is a major cause of maternal mortality and morbidities, prenatal deaths, preterm birth, and intrauterine growth restriction [9, 10].

The aim of this study is to evaluate any difference in the prevalence of high blood pressure and any possible effects on the cardiovascular system in two groups of pregnant women exposed to different levels of air pollution.

Subjects and Methods

Study Area

The study was conducted in the city of Niš, in southern Serbia. In 2008 Niš had a population of 381.757 inhabitants in an area of 596.71 m². The dominant air pollution source is traffic.

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Subjects

The subjects are 654 non-smoking pregnant women living in two areas in Niš with different levels of air pollution. All subjects have lived more than five years in the same location, about 25 km from an air monitoring station and they are not professionally exposed to air pollution.

They are separated in two groups: exposed and non-exposed. The exposed group of pregnant women (n=348) live in an area of Niš with a high level of air pollution, while the pregnant women from the non-exposed group (n=306) live in an area with a lower level of air pollution: Niška Banja.

All of these pregnant women are in early pregnancy (gestational age <10 weeks). Data on pregnancy is collected on the basis of physical examinations, fetal ultrasounds, and hospital registrations.

The subjects are of the same ethnicity and are not alcohol consumers.

Data on age, educational level, parity, passive smoking, and genetic predisposition is collected by an interview. Passive smoking is defined as the average number of hours per week the women are exposed to cigarette smoke from their husbands or anyone else inside their house. Genetic predisposition to high blood pressure is defined as a positive parental history of hypertension. Pregnant women in both groups do not have symptoms of any cardiovascular or pulmonary diseases. They are informed about the aims of the study, the performance, and the expected results of the study.

Blood Pressure

Blood pressure is measured at each trimester of pregnancy in the Gynecological and Obstetrics Clinic, Niš (Serbia). Pregnancy-induced hypertension is defined according to criteria described by the International Society for the Study of Hypertension in Pregnancy (ISSHP): development of systolic blood pressure ≥ 140 mmHg and/or diastolic blood pressure ≥ 90 mmHg without proteinuria after 20 weeks of gestation in previously normotensive women [11].

Air Pollution Exposure Measures

The pollutants are measured daily, during the whole year in and out of heating season, at the two measuring points. One measuring point is in the urban area of the town at the Square Knjeginje Ljubica, and the second measuring point is in Niska Banja in the peripheral area of the town.

Outdoor air pollutants are monitored during the 5-year period. The concentrations of black smoke (BS), sulfur dioxide (SO₂), and lead (Pb) in sediment matter are determined in 24-hour samples of air. This data is used to determine exposure for all subjects before pregnancy from January 2004 to December 2007, and during the pregnancy in 2008. The exposure is assigned to each woman as the average value for each year in which the air pollution is measured, four years before the pregnancy from 2004 to 2007, and then the year of their pregnancy in 2008.

Sampling equipment was placed 1.5 m above floor level at two sampling sites. The sampling sites are selected to ensure diversity regarding the outdoor environment.

The ambient level of black smoke concentrations is measured by reflectance. The sampling is performed by means of a pump operating with flow rate of 1 L/min through Whatman N°1 paper filters. At the same time, the air concentration of sulfur dioxide is measured. The measured volume of air is bubbled through a solution of potassium tetrachloromercurate. The sulfur dioxide present in the air stream reacts with the solution to form a stable monochlorosulfonatomercurate complex. During subsequent analysis, this complex is brought into reaction with acid-bleached pararosaniline dye and formaldehyde, yielding intensely colored pararosaniline methyl sulfonic acid. The optical density of this species is determined spectrophotometrically at 548 nm and is directly related to the amount of sulfur dioxide collected. The total volume of the air sample is determined from the flow rate and the sampling time. The concentration of sulfur dioxide in the ambient air is calculated and expressed in $\mu\text{g}/\text{m}^3$. The lowest limit of detection is $1.7 \mu\text{g}/\text{m}^3$.

Lead in sediment matter is collected with absorbed solution of sulfur acid and is detected by graphic furnace atomic absorption spectrometry. The lowest limit of detection was $0.5 \mu\text{g}/\text{m}^3$.

Statistical Analysis

Statistical analysis is performed on a computer, according to the standard principles of descriptive and analytical statistical methodology, using standard programs for processing data such as Excel and SPSS software package in version 10.0. Comparison between the frequency of modality and occurrence of the tested characteristics is performed by the nonparametric Pearson chi-squared test. Comparison of average values is done by t-test. Testing of correlation of individual characteristics is performed by regression analysis (SPSS), which establishes type and strength between the tested characteristics.

Univariate and multivariate regression analysis were used to investigate relationships between exposure of air pollution and pregnancy-induced hypertension, adjusting for potential confounding factors (age, educational level, parity, passive smoking, genetic predisposition).

Results

Table 1 shows baseline characteristics of the study population. There are no statistically significant differences in age, education level, passive smoking, genetic predisposition to high blood pressure, and parity between the two groups.

All concentrations of the air pollutants measured during the period 2004-08 at the location in Knjeginje Ljubica Square were higher when compared to the concentrations of the same pollutants measured at Niška Banja location. This difference is statistically significant (Mann-Whitney U test: $P < 0.05$).

Table 1. Subject characteristics.

Subject characteristics	Exposed (n=348)	Non-exposed (n=306)	Significance
Age, year ^a (mean±SD)	36.82±1.32	36.54±1.56	n.s.
Education level ^b			
Elementary	16%	21%	n.s.
Above elementary	84%	79%	
Passive smoking ^b			
YES	44%	46%	n.s.
Genetic predisposition to high blood pressure ^b			
YES	13%	19%	n.s.
Parity ^b			n.s.
Nulliparous	31%	24%	
Multiparous	69%	76%	

^a t-test.^b chi-square test.

n.s.– no statistically significant

The univariate regression analysis of all parameters are made in compared with pregnancy-induced hypertension (age, educational level, parity, passive smoking, genetic predisposition), and it is found that age and parity have a significant impact on pregnancy-induced hypertension (Table 2).

Inserting the multivariate model of variables that show significance, a coefficient of determination model is obtained (Cox & Snell R Square = 0.241), and a significant predictive effect of variables isn't obtained for age

($p=0.746$; OR=0.982, CI 0.583 <OR <1.654) and parity ($p=0.549$; OR=1.171, CI 0.699 <OR <1.962) (Table 3).

Discussion

Air pollution has become a major concern worldwide because of its influence on human health [12, 13]. However, a lot of studies have investigated exposure to this environmental risk factor in association with birth and pregnancy outcomes [14-16].

This investigation has not shown that long-term and short-term exposure to air pollution positively corresponds to the occurrence of high blood pressure among pregnant women. This is the first study to describe air pollution levels and variables in pregnancy-induced hypertension among Serbian women in the peer reviewed literature. The results obtained are a baseline for further analytic epidemiological research.

Current knowledge about the pathophysiology of hypertension induced in pregnancy support the theory that reduced placental perfusion and the maternal syndrome are key components of pregnancy-induced hypertension [17, 18]. Pregnancy-induced hypertension is also characterized by activation of the coagulation cascade and immunologic component [19, 20].

The study has several limitations. First, we do not examine the relationship between pregravid body mass index (BMI) and the development of hypertensive disorders of pregnancy. Women who are overweight (BMI 25-30) and obese (BMI ≥ 30) are at greater risk of adverse reproductive health outcomes compared to women of normal weight status (BMI 19.8-25) [21]. Pregnancy-induced hypertension and pre-eclampsia [22] have been associated with maternal overweight.

Table 2. Univariate logistic regression for pregnancy-induced hypertension.

Characteristics	B	Standard error (SE)	p value	Odds ratio (OR) [95% confidence interval (CI)]
Age	0.046	0.022	<0.05	1.047 (1.01-1.09)
Education level	0.216	0.128	n.s.	1.241 (0.96-1.59)
Passive smoking	0.113	0.233	n.s.	1.120 (0.71-1.77)
Genetic predisposition	0.034	0.221	n.s.	0.966 (0.63-1.49)
Parity	0.032	0.012	<0.05	1.021 (1.01-1.04)
Exposed/non-exposed	0.366	0.220	n.s.	1.442 (0.94-2.24)

n.s.– not statistically significant

Table 3. Multivariate logistic regression for pregnancy-induced hypertension.

Characteristics	B	Standard error (SE)	p value	Odds ratio (OR) [95% confidence interval (CI)]
Age	0.182	0.266	n.s.	0.98 (0.58–1.65)
Parity	0.158	0.264	n.s.	1.17 (0.69–1.96)

n.s.– not statistically significant

Second, our study cannot control lipid levels of pregnant women. Reducing blood lipid levels can decrease the incidence of pre-eclampsia and preterm labor significantly. According to the data of another study [23], serum levels of Lp(a), total cholesterol, triglycerides (TG), and low-density lipoprotein cholesterol (LDL-C) are significantly higher, and high-density lipoprotein cholesterol (HDL-C) and Apo A-I levels are significantly lower in severely preeclamptic and mildly preeclamptic women than in normal pregnant women. Serum level of Lp(a) is positively correlated with body mass index in severely preeclamptic women ($r=0.489$, $p=0.008$). However, lipoprotein concentrations may be better biochemical markers of dislipidemia in the preeclamptic state than the corresponding apolipoproteins [24].

Throughout the study, it is not examined how other factors such as population density, household income, and life styles in two different locations affect our two examined groups. Another limitation is that there is the possibility of different concentrations of air pollutants, even in the same location. For example, the examined women living in the Niš close to a road may experience high air pollution levels, whereas women living further away may have low levels of air pollution.

Other factors may interfere with the evaluation of a relationship between high blood pressure in pregnancy and exposure to air pollution. Parity is one of those factors. Nulliparous women have lower cardiovascular disease prevalence compared with parous women [25]. A study in a rural community in Zimbabwe reports that nulliparous and high parity women have an elevated risk of hypertensive complications RR 1.62 (95% CI 1.37-1.92) and RR 1.64 (95% CI 1.29-2.07), respectively [26]. In our study, the results of univariate regression analysis have shown that parity has a significant impact on pregnancy-induced hypertension (95%CI 1.01-1.04).

It is difficult to determine whether one of the measured pollutants, alone or in combination, is responsible for the observed health effects of pregnant women. It is, also, less clear which pollutants are most responsible for pregnancy-induced hypertension. Little is known about the possible adverse effects of exposure to complex mixtures of chemicals. Although the concentration of sulfur dioxide and black smoke have been significantly decreasing worldwide in the last 10 years, the present air concentration in the city of Niš is still an important threat to pregnant women's health.

Data from a prospective cohort study in the Netherlands [27] has shown that PM₁₀ exposure, but not NO₂ exposure, was associated with an increased risk of pregnancy-induced hypertension (odds ratio 1.72 [95% CI 1.12 to 2.63] per 10- $\mu\text{g}/\text{m}^3$ increase). Concerning carbon monoxide, a study in California [28] (after adjustment for maternal characteristics) found adjusted odds ratios of 1.08 [95% confidence interval (CI), 1.02-1.14] for high CO exposure (>75th percentile) among mothers during their entire pregnancy who were residing within 10 kilometers of a monitor. An elevated odds ratio of 1.14 [95% confidence interval (CI), 1.05-1.23] for high SO₂ exposure (>75th percentile) also was observed. Analysis of data from acute impact of PM expo-

sure on preeclampsia complicating delivery in the United States [29] showed no significant associations between 6-week average PM₁₀ or PM_{2.5} and risk of preeclampsia. A study in southern California [30] has examined effects of residential exposure to local traffic-generated air pollution on preeclampsia. The risk of preeclampsia increases 33% and 42% for the highest NO(x) and PM(2.5) exposure quartiles, respectively.

Other studies have suggested that environmental lead exposure increases the risk of pregnancy-induced hypertension. The risk of pregnancy-induced hypertension increases by 4% per 0.05 $\mu\text{g}/\text{m}^3$ increase in seasonal average lead levels at conception and birth, in both smokers and nonsmokers [31]. The EDEN cohort study [32] reports that lead levels are significantly higher in pregnancy-induced hypertension cases than in normotensive patients.

Pregnancy-induced hypertension is at greater risk of cardiovascular and cerebrovascular events and those women have a less favorable overall risk profile for CVD years after the affected pregnancies [33], and if the hypertensive disorder occurs in more than one pregnancy, or in a relatively late pregnancy, the associations with later cardiovascular risk factors are substantially stronger [34]. Furthermore, preeclampsia is associated with a substantially higher risk of developing diabetes [35].

The fetoplacenta barrier is very sensitive to the impact of air pollution. It is speculated that air pollutants present in the environment reduce the amount of soft chorion villus sampling and circulation, resulting in involutive changes in placenta. Further study is needed to disentangle this interaction and the underlying mechanisms.

Conclusion

Our results suggest that exposure after and during pregnancy to relatively low levels of some air pollutants are not associated with pregnancy-induced hypertension. However, monitoring the influence of air pollution on the occurrence of pregnancy-induced hypertension and its outcome should be intensively pursued in the future.

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