

OBSTETRICS

A population-based study of associations between preeclampsia and later cardiovascular risk factors

Ingvild V. Alsnes, MD; Imre Janszky, MD, PhD; Michele R. Forman, MSc, PhD;
Lars J. Vatten, MD, PhD; Inger Økland, MD, PhD

OBJECTIVE: Women with a history of preeclampsia are at increased lifetime risk for cardiovascular disease. Their offspring may carry similar risks. The aim was to study cardiovascular and metabolic risk factors 11 years after the delivery among women who were diagnosed with mild, moderate, or severe preeclampsia, and their offspring, compared with women without preeclampsia and their offspring.

STUDY DESIGN: In a follow-up 11 years after a nested case-control study at birth, we studied 611 mother-offspring dyads, including 228 dyads with preeclampsia in the index pregnancy and 383 dyads without preeclampsia. Cardiovascular and metabolic risk profiles were assessed by serum lipids (total cholesterol, high-density lipoprotein [HDL] cholesterol, non-HDL cholesterol), insulin-related factors (glucose, insulin, and homeostasis assessment model for insulin resistance) and blood pressure in mothers and children.

RESULTS: Among mothers with mild or moderate preeclampsia, levels of glucose, insulin, and homeostasis assessment model

for insulin resistance were higher than in the nonpreeclampsia group and also higher compared with mothers with severe preeclampsia (all $P < .05$). HDL cholesterol was lower in mothers with mild or moderate preeclampsia (all $P < .05$), but other lipids did not substantially differ between the groups. Body mass index and blood pressure (systolic and diastolic) were also higher in the mild and moderate preeclampsia group compared with mothers without preeclampsia (all $P < .05$). Among the offspring, we found no clear differences in any blood analytes between the groups.

CONCLUSION: Women with a previous diagnosis of mild or moderate, but not severe, preeclampsia may have an adverse metabolic and cardiovascular risk profile 11 years after the delivery.

Key words: cardiovascular disease, cardiovascular risk factors, epidemiology, insulin resistance, preeclampsia

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Preeclampsia is characterized by hypertension and proteinuria in pregnancy and is diagnosed in about 3-4% of all pregnancies. It is a leading cause of maternal and perinatal morbidity,

and the most severe form (eclampsia) is associated with maternal morbidity worldwide.¹⁻³ There is a growing body of evidence that preeclampsia may increase the risk of cardiovascular disease later in life, but the underlying mechanisms are not known. A metaanalysis of 3.5 million women showed that having a history of preeclampsia is associated with an increased lifetime risk of hypertension, ischemic heart disease, stroke, and venous thromboembolism and that total mortality may be higher in women with a history of preeclampsia.⁴

Preeclampsia is a disease of unknown etiology that may develop through different pathways, as indicated by varying phenotypes that are classified as early or late at gestational age of onset and by the clinical severity of preeclampsia.^{5,6} Indeed, the diverse phenotypic expressions of preeclampsia may be differentially associated with later cardiovascular risk,

both among mothers and offspring. For example, increasing severity of hypertensive disorders in pregnancy may be related to increased risk of future cardiovascular events.^{7,8} Therefore, it is conceivable that mothers with a history of mild, moderate, or severe preeclampsia may exhibit different cardiovascular risk factor profiles⁹ and that similar corresponding patterns may be present among their offspring.

In a follow-up study of mothers and children 11 years after the delivery, we examined cardiovascular and metabolic factors in 2 groups: (1) the mothers who developed preeclampsia were compared with mothers without preeclampsia in the index pregnancy, and (2) their offspring were compared according to maternal preeclampsia status. The main aim was to examine whether the preeclampsia dyads had an adverse cardiovascular risk profile compared with the dyads without preeclampsia.

From the Departments of Obstetrics and Gynecology (Drs Alsnes and Økland) and Research (Dr Vatten), Stavanger University Hospital, Stavanger, and Department of Public Health, Faculty of Medicine, Norwegian University of Science and Technology, Trondheim (Drs Janszky and Vatten), Norway, and Department of Nutritional Sciences, University of Texas at Austin, Austin, TX (Dr Forman).

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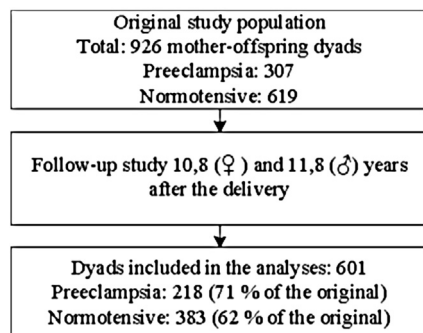
Corresponding author: Ingvild V. Alsnes, MD.
ingvild.vatten.alsnes@sus.no

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FIGURE

Flow chart of original and follow-up participants

Flow chart of the original participants and those who attended the follow-up study and were included in the analyses of the present study.

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A second aim was to assess whether the risk profile may differ between mothers with mild, moderate, or severe preeclampsia and between the corresponding offspring. From the same cohort, Ogland et al¹⁰⁻¹² have previously studied body mass index (BMI), waist and hip circumference, blood pressure, and clinical signs of puberty in the offspring.

MATERIALS AND METHODS

Study population

Mothers and offspring who participated in a population-based, nested, case-control study of births over a 3-year period (1993-1995) at Stavanger University Hospital (Stavanger, Norway) were followed up when the offspring were 10-11 years old. At baseline (birth), mothers and offspring of this study were identified using 3 separate procedures. First, all women who delivered at Stavanger University Hospital who were registered with preeclampsia in the Medical Birth Registry of Norway were identified as potential cases. Second, these potential cases were identified from the midwives records at the delivery station. Third, all pregnancies with proteinuria and hypertension registered in the computerized hospital database were identified.

Through these 3 procedures, approximately 1300 women were identified as having potential cases of preeclampsia. The hospital record for each case was subsequently reviewed, and mothers (and offspring) who fulfilled the strict diagnostic criteria of preeclampsia as described in the Collaborative Low-dose Aspirin Study in Pregnancy (CLASP)¹³ were selected as cases. Among a total of 12,804 consecutive singleton deliveries, 307 women who were diagnosed with preeclampsia and another 619 women without preeclampsia who were matched by maternal age or by delivery date participated as cases and controls in the birth study.^{10,14-18}

The preeclampsia cases were categorized as clinically mild, moderate, or severe according to the criteria in the CLASP.¹³ Thus, after 20 weeks of gestation, persistent diastolic blood pressure of at least 90 mm Hg had to develop in addition to an increase in diastolic blood pressure of at least 25 mm Hg. Cases with a baseline diastolic blood pressure of 90 mm Hg or higher were included if diastolic blood pressure had increased by at least 15 mm Hg. Proteinuria was defined as 0.3 mg/L (semiquantitative dipstick +1) in at least 1 urine sample after 20 weeks of gestation without simultaneous urinary infection.

Mild preeclampsia ($n = 73$) was defined as an increase in diastolic blood pressure of at least 25 mm Hg and proteinuria 1+ on semiquantitative dipstick after gestational week 20. Moderate preeclampsia ($n = 91$) was defined as a diastolic blood pressure increase of at least 25 mm Hg and proteinuria 2+ on semiquantitative dipstick. Severe preeclampsia ($n = 54$) was defined as diastolic blood pressure of at least 110 mm Hg and proteinuria 3+ on semiquantitative dipstick or at least 500 mg per 24 hours. HELLP (hemolysis, elevated liver enzymes, and low platelet count) syndrome and eclampsia were included in the severe preeclampsia category.¹⁶

At 10.8 years for mothers who delivered girls and 11.8 years for mothers who delivered boys, the mother-offspring dyads were invited to a follow-up, essentially coinciding with the time when the children were expected to enter puberty. From the original study, 601 dyads

participated in the follow-up study, among whom 218 mother-offspring pairs were part of the preeclampsia group and 383 pairs were part of the group without preeclampsia. The Figure shows a flow chart of the original study participants and those (65%) who attended the follow-up study and were included in the analyses of the present study. There was no differential loss to follow-up between the participating groups. Moreover, there were no differences between the participants in this study and the nonparticipants at follow-up with respect to the baseline values, such as maternal age, weight and height, frequency and severity of preeclampsia, or gestational age and birthweight of the children.¹¹ The majority of the participants were European whites ($n = 585$), whereas 16 were of non-European ethnicity.

Data collection

The follow-up study included questionnaires, a clinical examination, and collection of fasting blood samples from mothers and their offspring. The clinical examination was conducted by 3 trained nurses at the pediatric outpatient clinic at Stavanger University Hospital.

Body weight was measured to the nearest 0.1 kg using an electronic scale (Seca 770; Seca, Hamburg, Germany) and height was measured to the nearest 0.1 cm, using the Harpenden stadiometer (Holtain; Crosswell, Crymych, Wales, UK), and recorded as the mean of 2 successive measurements. BMI was calculated as weight divided by height squared, and the waist to hip ratio was calculated as the mean value of 2 measurements of waist and hip circumference, recorded to the nearest 0.1 cm.

After sitting for 5-10 minutes, systolic and diastolic blood pressures were measured electronically with Criticare 506N (Criticare Systems Inc., Waukesha, WI); the measurements were conducted 3 times, and the average of the second and third measurement was recorded to the nearest millimeter of mercury.¹¹

Fasting blood samples (ranging in time since dinner from 8 hours or more) were collected by trained biomedical technicians.¹⁰⁻¹² Total serum cholesterol, high-density lipoprotein

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