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Cardiovascular phenotype in women 1–3 years after hypertensive pregnancy disorders: Impact of sex-independent and pregnancy-specific risk factors

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ABSTRACT

Objectives: The underlying mechanisms for adversely altered cardiovascular phenotype after hypertensive disorders of pregnancy (HDP) remain poorly understood. We aimed to explore the impact of sex-independent cardiovascular disease (CVD) risk factors on associations between HDP and postpartum echocardiographic findings.

Study design: Echocardiography was conducted in 100 women 1–3 years after HDP (n = 65) and normotensive pregnancies according to a standard protocol (n = 35).

Main outcome measures: Associations between previous HDP and echocardiographic measurements were explored by uni- and multivariate regression analyses. We adjusted for age, body mass index, mean arterial blood pressure and family history of CVD. P-value < 0.05 was considered statistically significant.

Results: Women after HDP displayed more adverse cardiometabolic profiles, including more frequent Stage 2 hypertension and less frequent normal blood pressure compared to controls (18 % vs 3 % and 46 % vs 83 %, both p < 0.05). The HDP group had more adverse echocardiographic profiles compared to controls. In univariate regression analyses, HDP was associated with Total Vascular Resistance and Septal Wall diameter. After adjustments for sex-independent cardiovascular risk factors, HDP was significantly associated with Septal Wall diameter in diastole and Relative Wall thickness.

Conclusions: Associations between previous HDP and postpartum echocardiographic findings remained significant after adjustment, but were mostly explained by sex-independent CVD risk factors. Women with previous HDP also displayed more adverse cardiometabolic profiles, including higher hypertension rates. Our findings highlight the need for intensified postpartum CVD prevention in women after HDP. In this cohort,

Abbreviations: BMI, Body mass index; BP, Blood pressure; BSA, Body Surface Area; CO, Cardiac output; CVD, cardiovascular disease; DBP, diastolic blood pressure; GDM, Gestational diabetes mellitus; GH, Gestational hypertension; GW, Gestational week; HAPPY, Health after pregnancy complications study; HDP, Hypertensive disorders of pregnancy; HR, Heart rate; ISSHP, International Society for the Study of Hypertension in Pregnancy; LAV, Left atrial end systolic volume, biplane; LAVi, Left atrial volume indexed to body surface area; LV, Left ventricular; LVIDd, LV inner dimension in diastole; LVCODopp, Left Ventricular Cardiac Output imaged by Doppler Technique; LVEF, Left ventricular ejection fraction; LVIDd, Left ventricular internal diameter in diastole; LVM, Left ventricular mass; LVMi, Left ventricular mass indexed to body surface area; MAP, Mean arterial blood pressure; NCEP ATP III, National Cholesterol Education Program Adult Treatment Panel III; OGTT, oral glucose tolerance test; PE, Preeclampsia; PWd, Posterior wall diameter in diastole; RWT, Relative wall thickness; S', peak systolic annular velocity; SBP, systolic blood pressure; SV, Stroke volume; SWd, Septal wall diameter in diastole; TAPSE, tricuspid annular plane systolic excursion; TRVmax, Tricuspid regurgitation maximum; TVR, Total vascular resistance.

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echocardiography provided limited additional information beyond well-known risk factors in the evaluation of CVD risk in asymptomatic women with previous HDP.

1. Introduction

Cardiovascular disease (CVD) is the leading cause of death worldwide.[1] Female CVD is underrecognized for several reasons, including sex-specific differences in clinical presentation, developmental patterns, and risk factors.[2] CVD development is impacted by classical as well as female-specific risk factors.[1–3] Hypertensive disorders of pregnancy (HDP) are important female-specific CVD risk factors,[4–7] associated with a 2–8-fold increased CVD risk as compared to normotensive pregnancies.[6] Within 10 years after HDP, women have an excess risk of hypertension,[8] as well as 2-fold increased risk of heart failure, in particular heart failure with preserved ejection fraction (HFpEF).[9,10] Pregnancy represents a stress-test for womeńs future cardiovascular health. Hence, HDP is a marker of abnormal cardiovascular adaptation during pregnancy, which may be seen as a warning for adverse future maternal cardiovascular health.[6,11] Early cardiovascular risk factor detection and management is likely important for prevention and postponement of CVD and CVD-associated morbidity.[12] HDP and CVD share several important sex-independent risk factors, including age, elevated blood pressure, overweight, and dyslipidemia.[13–16] Women experience HDP early in life. Postpartum clinical risk assessment and timely intervention may reduce their increased risk of adverse cardiovascular outcomes later in life.[17]

Cardiovascular adaptation is well-documented in normotensive and HDP pregnancies.[18] Studies assessing cardiovascular function and structure following HDP report persistent cardiac dysfunction postpartum, including adverse cardiac remodeling and increased arterial stiffness.[18–22] A weakness of most of these follow-up studies is the scarcity of data on sex-independent CVD risk factors.[22] Consequently, the roles of risk factors in the increased morbidity associated with CVD following HDP remain unclear. We hypothesized that the associations between a history of recent HDP and abnormal echocardiographic findings could be partially attributed to classical sex-independent CVD risk factors. We therefore aimed to investigate whether associations between recent HDP and cardiovascular structure and function 1–3 years postpartum differed after adjustment for sex-independent CVD risk factors. For comparison, we included a group with a recent uncomplicated normotensive pregnancy.

2. Methods

2.1. Study population

In this prospective cohort study, we included a subgroup of women from the Health after pregnancy complications study (HAPPY).[23] This encompassed women ≥ 18 years who delivered a singleton baby one or three years previously at Oslo University Hospital, Ullevål, Norway. Women with an index pregnancy complicated by HDP or a normotensive and euglycemic index pregnancy were eligible for inclusion (Fig. 1). A randomly selected subset of study participants was offered echocardiography. The selection was impacted by availability of the echocardiography apparatus and a cardiologist with echocardiography expertise. Echocardiography data were available for 135 women. Echocardiographic data for both one and three years were available for one participant, in which 3-year data were used. Data from 100 women were analyzed after exclusion of study participants who were pregnant, breastfeeding, or had diabetes, rheumatic, renal, malignant, or established cardiovascular disease other than previous HDP. One-year follow-up data were available for 71 % of the cohort, and three-year follow-up data were available for the remaining 29 % (Fig. 1 and Table 1).

The HDP group encompassed index pregnancies complicated by gestational hypertension (GH), preeclampsia (PE) or chronic hypertension with superimposed PE. GH was defined as new-onset hypertension (systolic blood pressure (SBP) ≥ 140 mmHg and/or diastolic blood pressure (DBP) ≥ 90 mmHg) after gestational week 20, without proteinuria or any evidence of PE-associated hematological or biochemical complications.[24] PE was defined as GH and at least one new-onset PE-associated feature after gestational week 20 (e.g., new-onset proteinuria, elevated transaminases, fetal growth restriction or stillbirth), according to the 2018 International Society for the Study of Hypertension in Pregnancy guidelines.[24] Chronic hypertension with superimposed PE was defined as hypertension diagnosed before gestational week 20, or predating the pregnancy, and at least one new-onset PE-associated feature after gestational week 20.[24] Women with eclampsia and/or HELLP (hemolysis, elevated liver enzymes and low platelets) in their index pregnancy were defined as PE. Fetal growth restriction was defined as an offspring with birthweight below the national 3rd percentile (in the absence of malformations or chromosomal abnormalities), adjusted for fetal sex and gestational age at delivery.[25]

Controls included women with normotensive and euglycemic index pregnancies, who delivered newborns without fetal growth restriction at term (≥ gestational week 37^0), without HDP in any previous pregnancy.

2.2. Clinical cardiovascular assessments and blood sampling

In-patient hospital blood pressure (BP) was measured according to clinical routine with a validated device (Dinamap Pro, 100VE, GE Medical Systems Information Technology, Inc. Milwaukee, WI, USA). BP at follow-up was measured as described by us.[26] Blood pressure

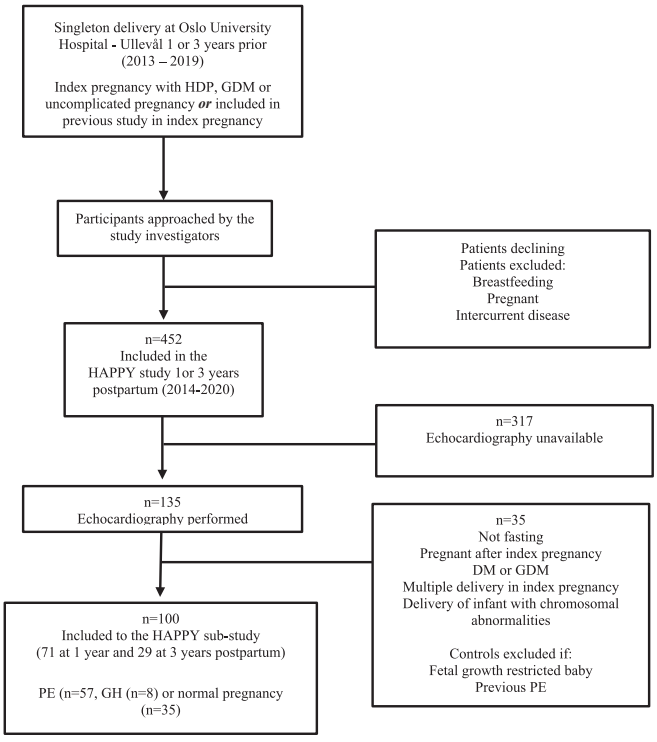

Fig. 1. Flowchart of the participant inclusion and exclusion to this HAPPY substudy (Health After Pregnancy Complications) one or three years postpartum. DM: diabetes mellitus; GDM: gestational diabetes mellitus; GH: gestational hypertension; HDP: hypertensive disorders of pregnancy (including both PE and GH patients); PE: preeclampsia.

Table 1
Clinical characteristics of the study population at 1–3-year follow-up (n = 100), by index pregnancy group. Data are presented as medians (interquartile range) for continuous variables. Categorical variables are presented as rates (%). p-values are compared with the control group. A p-value < 0.05 is marked with asterix (*).

Clinical data 1–3 years post-partum	Controls (n = 35)	HDP (n = 65)	PE (n = 57)	GH (n = 8)
Age (years)	34.9 (32.6–38.0)	35.3 (32.0–39.0)	35.0 (31.5–37.9)	38.1 (35.5–39.3)
Systolic BP (mmHg)	109 (101–112)	117 (108–126)*	117 (107–125)*	123 (113–132)*
Diastolic BP (mmHg)	63 (60–70)	76 (70–86)*	75 (70–85)*	90 (76–98)*
Mean Arterial BP (mmHg)	78 (74–84)	91 (83–99)*	90 (82–96)*	98 (91–110)*
Normal BP (SBP < 120 and DBP < 80)	82.9 % (29)	46.2 % (30)*	49.1 % (28)*	25.0 % (2)*
Elevated BP (SBP 120–129 and DBP < 80)	0.0 % (0)	10.8 % (7)	10.5 % (6)	12.5 % (1)
Stage 1 hypertension (SBP 130–139 and/or DBP 80–89)	14.3 % (5)	24.6 % (16)	24.6 % (14)	25.0 % (2)
Stage 2 hypertension (SBP ≥ 140 and/or DBP ≥ 90)	2.9 % (1)	18.5 % (12)*	15.8 % (9)	37.5 % (3)*
Antihypertensive medication	0.0 % (0)	9.2 % (6)	8.8 % (5)	12.5 % (1)
Recently diagnosed hypertension**	0.0 % (0)	6.2 % (4)	5.3 % (3)	12.5 % (1)
Metabolic syndrome	0.0 % (0)	7.7 % (5)	7.0 % (4)	12.5 % (1)
Anthropometric data				
Weight (kg)	65 (60–73)	70 (60–78)	70 (60–81)	68 (60–75)
Height (cm)	167 (164–172)	167 (164–172)	167 (164–172)	168 (160–172)
BMI (kg/m^2)	22.8 (21.8–26.0)	24.8 (22.1–27.2)	25.0 (22.1–27.2)	23.6 (21.9–25.7)
Overweight (BMI ≥ 25 kg/m^2 and BMI < 30 kg/m^2)	25.7 % (9)	36.9 % (24)	38.6 % (22)	25.0 % (2)
Obesity (BMI ≥ 30 kg/m^2)	8.6 % (3)	12.3 % (8)	12.3 % (7)	12.5 % (1)
Patient characteristics				
Premature CVD in 1st degree relative	17.1 % (6)	38.5 % (25)	36.8 % (21)	50.0 % (4)
Education > high school	51.4 % (18)	72.3 % (47)*	71.9 % (41)*	75.0 % (6)
White/not stated ethnicity	94.3 % (33)	95.4 % (62)	94.7 % (54)	100.0 % (8)
1-year visit data/3-year visit data	80.0 % (28)	66.2 % (43)	68.4 % (39)	50.0 % (4)
Time from pregnancy to follow-up (months)	13.5 (12.8–15.8)	13.9 (12.7–36.3)	13.9 (12.7–36.2)	25.8 (13.2–37.1)
Current smoking (Yes)	20.0 % (7)	0.0 % (0)*	0.0 % (0)*	0.0 % (0)

Blood pressure categories are defined according to The American College of Cardiology/American Heart Association (ACC/AHA) Task Force on Clinical Practice Guidelines.[27] The Mann-Whitney U test for continuous variables and Chi square or Fisheŕs mid-p corrected test for categorical variables comparing pregnancy complication groups (e.g., HDP) to controls. Values are presented as medians (ranges) or rates (n). BMI: body mass index; BP: blood pressure; CVD: cardiovascular disease; DBP: diastolic blood pressure; GH: gestational hypertension; HDP: hypertensive disorders of pregnancy (including both PE and GH patients); MAP: mean arterial blood pressure; PE: preeclampsia; SBP: systolic blood pressure.
**Recently diagnosed hypertension: includes women with new-onset hypertension after delivery (either diagnosed at this follow-up or started antihypertensive medical treatment after delivery).

categories (displayed in Table 1) were defined according to The American College of Cardiology/American Heart Association (ACC/AHA) Task Force on Clinical Practice Guidelines.[27]

At follow-up, participants completed questionnaires regarding family CVD history and own cardiovascular health, traditional cardiovascular risk factors, general health, socioeconomic factors and previous pregnancies before index pregnancy. A family history of CVD was defined as CVD or cardiovascular death in the participant's father before 55 years of age or in the participant's mother before 65 years of age. Metabolic syndrome was defined according to The National Cholesterol Education Program (NCEP) Adult Treatment Panel III (ATP III) definition.[28]

2.3. Image acquisition

Two experienced cardiologists (TGvL, KA) conducted and interpreted all echocardiographic examinations. Echocardiography was conducted after 10 min of rest, during resting breathing conditions, with women in the left lateral position, following local clinical protocol in a calm, temperature-controlled room with dimmed lights. Echocardiographic measurements are described in Supplementary Material 1. Computed echocardiographic measurements are described in Supplemental Table S1. Pathological cardiac remodeling was defined as the presence of concentric remodeling, concentric hypertrophy, or eccentric hypertrophy.[29] Definitions are displayed in Supplemental Table S2. Age- and sex-specific reference ranges for all echocardiographic variables are presented in Supplemental Table S3.

2.4. Statistical analyses

Statistical analyses were conducted with the Statistical Package for the Social Sciences (PASW Statistics 29) and statistical software for data science (Stata Statistical Software: Release 18. College Station, TX, STATACorp LLC). Most continuous variables were not normally distributed. Therefore, Mann-Whitney *U* test was used to test group differences for continuous variables. The Chi square test and Fisheŕs mid-*p* corrected test were used to test differences between groups for categorical variables. Continuous data are presented as median and ranges and categorical data as numbers and rates (%). Uni- and multivariate median regression analyses were conducted to assess associations between previous HDP and echocardiographic variables. Regression analyses were not conducted on the GH group due to the small sample size (n = 8). Confounding variables (maternal age, mean arterial blood pressure (MAP), Body Mass Index (BMI) and CVD family history) were identified by using a Directed Acyclic Graph (DAG) (Supplemental Fig. S1). Missing data were analyzed as missing for all variables, except from missing CVD family history information, for which we presumed no CVD family history (n = 7). A *p*-value < 0.05 was considered statistically significant. Correction for multiple testing was not conducted due to the exploratory nature of the study.

3. Results

Among the 100 included women (Fig. 1), 65 had HDP during their index pregnancy (PE: n = 57; GH: n = 8) and 35 had normotensive index pregnancies (controls). Postpartum clinical characteristics of the study cohort are displayed in Table 1 and 2. Index pregnancy characteristics are shown in Supplemental Table S4. Three women with superimposed PE were included, representing a more severe cardiovascular burden than PE alone, but sensitivity analyses without these patients did not change the main results.

Median age at postpartum follow-up was 35 years for the total cohort, with no significant differences between the study groups (Table 1). Median time from pregnancy to follow-up for the total cohort was 14 months. The previous HDP group displayed significantly higher blood pressure, as well as significantly higher rate of Stage 2

Table 2
Echocardiography measures of cardiovascular structure and function of the study population 1–3 years postpartum (n = 100), by index pregnancy group. Data are presented as medians (interquartile range) for continuous variables. p-values are compared with the control group. A p-value < 0.05 is marked with asterix (*).

Cardiac and vascular structure and function 1–3 years post-partum	Controls (n = 35)	HDP (n = 65)	PE (n = 57)	GH (n = 8)
CARDIAC MORPHOLOGY/STRUCTURE				
LVIDd (cm)	4.9 (4.7–5.2)	4.9 (4.5–5.1)	4.8 (4.5–5.1)	5.0 (4.8–5.2)
PWd (cm)	0.7 (0.6–0.7)	0.7 (0.6–0.8)	0.7 (0.6–0.8)	0.6 (0.6–0.8)
SWd (cm)	0.7 (0.6–0.7)	0.7 (0.6–0.8)	0.7 (0.6–0.8)*	0.7 (0.6–0.8)
LVM (g)	98.4 (88.5–110.8)	104.9 (91.6–119.3)	107.6 (91.8–119.3)	98.3 (87.0–121.8)
LVMi (g/m^2)	56.3 (51.8–60.7)	57.5 (49.5–66.8)	57.9 (49.1–66.8)	53.4 (50.0–66.4)
LAVi (mL/m^2)	19.4 (17.5–21.7)	20.5 (15.8–25.1)	20.8 (15.8–25.1)	18.5 (16.4–21.5)
RWT	0.27 (0.24–0.30)	0.28 (0.26–0.32)	0.29 (0.26–0.33)*	0.26 (0.24–0.30)
CARDIAC FUNCTION				
TVR ($dyne.s^{-1}\ cm^{-5}$)	1374.02 (1256.88–1657.85)	1560.56 (1402.09–1788.93)*	1551.66 (1409.79–1786.87)*	1897.54 (1315.23–2041.57)
HR (bpm)	67 (61–75)	68 (62–76)	69 (62–76)	66 (63–75)
SV (mL) (Doppler)	69 (62–76)	65 (60–71)	65 (60–71)	67 (59–74)
CO (L/minute)	4.49 (4.15–4.97)	4.46 (4.02–5.15)	4.46 (4.01–5.19)	4.54 (4.33–4.75)
LVEF (%) (biplane)	63 (62–65)	63 (61–65)	63 (61–65)	63 (61–65)
DIASTOLIC FUNCTION				
E (cm/s)	76 (65–87)	77 (66–87)	77 (66–87)	77 (69–84)
A (cm/s)	43 (37–48)	44 (39–53)	43 (39–54)	45 (40–47)
E/A ratio	1.90 (1.52–2.10)	1.66 (1.44–2.04)	1.65 (1.42–2.05)	1.74 (1.62–1.86)
E/é ratio	6.27 (5.62–6.68)	6.21 (5.58–7.27)	6.20 (5.54–7.32)	6.62 (5.78–7.15)
MITRAL VALVE				
S' velocity (cm/s)	8 (7–9)	8 (7–8)	8 (7–9)	8 (7–8)
RIGHT HEART FUNCTION				
TRVmax (m/s)	2.04 (1.86–2.16)	2.07 (1.90–2.23)	2.07 (1.90–2.23)	2.09 (1.94–2.23)
S' velocity (cm/s)	13(11–14)	12(11–14)	13(11–14)	12(11–13)
TAPSE (mm)	23(21–26)	25(23–28)*	25(23–28)*	25(22–27)

The Mann-Whitney U test for continuous variables and Chi square or Fisheŕs mid-p corrected test for categorical variables comparing pregnancy complication groups (e.g., HDP) to controls. Values are presented as medians (ranges) or rates (n). CO: cardiac output; HR: heart rate; LAVi: Left atrial volume indexed to body surface area; LV: left ventricular; LVEF left ventricular ejection fraction; LVIDd: LV internal diameter in diastole; LVM: left ventricular mass; LVMi: LVM indexed to body surface area; PWd: posterior wall diameter in diastole; RWT: relative wall thickness; Ś: peak systolic annular velocity; SV: stroke volume; SWd: septal wall diameter in diastole; TAPSE: Tricuspid annular plane systolic excursion; TRVmax: tricuspid regurgitation maximum; TVR: total vascular resistance.

hypertension, compared to controls (Table 1). All women with current antihypertensive medication had previous HDP (9.2 %). Moreover, there was a strong trend for higher rates of overweight, obesity and metabolic syndrome in the HDP group at follow-up, although not statistically significant (Table 1).

Women with previous HDP were more often primiparous, delivered newborns with lower birthweight and at a lower gestational age, compared to controls (Supplemental Table S4). No women in the previous HDP group were smoking at follow-up, in contrast to 20 % among controls (Table S1). Education and smoking were not adjusted for due to a high number of missing data and uneven distributions between study groups, respectively.

3.1. Echocardiography results postpartum

At follow-up, we found no evidence of heart failure (neither with reduced nor preserved ejection fraction) or pathological cardiac remodeling. Women with previous HDP displayed significantly higher Total Vascular Resistance (TVR) than controls (Table 2). Median TVR was slightly above normal range for all HDP subgroups (Table 2 and Supplemental Table S3 and S5). Remaining echocardiographic markers displayed no significant differences in median values between the previous HDP group and controls, except for a somewhat higher median tricuspid annular plane systolic excursion (TAPSE) in the HDP group. Median values for all echocardiographic markers, except for TVR, were within normal range in the total cohort (Table 2 and Supplemental Table S3 and S5).

When subdividing the previous HDP group into preeclampsia (PE) and gestational hypertension (GH), TVR was significantly higher in the PE group, compared to controls. Median TVR was non-significantly higher in the GH group compared to controls (Table 2). Further, median Relative Wall Thickness (RWT) was significantly higher in the previous PE group compared to controls, however within normal range for both study groups (Table 2 and Supplemental Table S4). The median Septal Wall diameter in diastole (SWd) was within the normal range for both the PE and control groups. SWd had same median value in the PE

Table 3
Unadjusted and adjusted associations between HDP (n = 65) and cardiovascular structure and function at follow-up. Adjustments were made for maternal age, BMI, MAP and CVD family history. Significant associations (p < 0.05) are marked with asterix (*).

Cardiovascular structure and function 1 or 3 years postpartum	HDP Unadjusted b (95 % CI)	p	HDP Adjusted b (95 % CI)	p
CARDIAC MORPHOLOGY/STRUCTURE				
LVIDd (cm)	−0.10 (−0.33 – 0.13)	0.40	0.02 (−0.22 – 0.27)	0.87
PWd (cm)	0.00 (−0.05 – 0.05)	1.00	−0.00 (−0.05 – 0.05)	1.00
SWd (cm)	0.10 (0.05 – 0.15)	<0.01*	0.10 (0.04 – 0.16)	<0.01*
LVM (g)	5.89 (−4.74 –16.53)	0.27	5.62 (−7.60 – 18.84)	0.40
LVMi (g/m^2) •••	1.09 (−4.48 – 6.66)	0.70	2.61 (−3.29 – 8.50)	0.38
LAVi (mL/m^2) •••	1.23 (−2.10 – 4.57)	0.47	−0.60 (−4.05 – 2.85)	0.73
RWT	0.02 (−0.01– 0.04)	0.12	0.03 (0.01– 0.06)	0.01*
FUNCTION				
TVR (dyne.s^{-1}cm^{-5}) ••	195.48 (17.11– 373.85)	0.03*	137.10 (−47.04 – 321.23)	0.14
HR (bpm)	1.00 (−5.28 –7.28)	0.75	−1.37 (−7.66– 4.92)	0.67
SV (mL) (Doppler)	−4.00 (−9.10 – 1.10)	0.12	−3.82 (−9.79 – 2.14)	0.21
CO (L/minute)	−0.03 (−0.40 – 0.34)	0.87	−0.05 (−0.48 – 0.39)	0.84
LVEF (%) (biplane)	0.00 (−1.83 – 1.83)	1.00	−0.89 (−2.40 – 0.61)	0.24
DIASTOLIC FUNCTION				
E (cm/s)	0.01 (−0.08 – 0.10)	0.83	−0.03 (−0.12 – 0.07)	0.58
A (cm/s)	0.01 (−0.05 – 0.07)	0.74	−0.00 (−0.07 – 0.07)	0.95
E/A ratio	−0.23 (−0.47 – 0.01)	0.06	−0.18 (−0.41 – 0.05)	0.13
E/é ratio	−0.06 (−0.65 – 0.53)	0.84	−0.02 (−0.75 – 0.71)	0.96
MITRAL ANNULAR MOTION				
S' velocity (cm/s)	0.00 (−0.50 – 0.50)	1.00	0.00 (−0.68 – 0.68)	1.00
RIGHT HEART FUNCTION				
TRVmax (m/s)	0.05 (−0.12 – 0.22)	0.56	0.00 (−0.17 – 0.17)	0.99
S' velocity (cm/s)	−1.00 (−2.32 – 0.32)	0.14	−0.13 (−1.24 – 0.98)	0.81
TAPSE (mm)	2.00 (0.49 – 3.51)	0.01*	1.90 (−0.21 – 4.01)	0.08

Beta (95 % confidence interval) and p-values for univariate regression analyses. BMI: body mass index; CI: confidence interval; CO: cardiac output; CVD: cardiovascular disease; HR: heart rate; LAVi: Left atrial volume indexed to body surface area; LV: left ventricular; LVEF left ventricular ejection fraction; LVIDd: LV internal diameter in diastole; LVM: left ventricular mass; LVMi: LVM indexed to body surface area; MAP: mean arterial blood pressure; PWd: posterior wall diameter in diastole; RWT: relative wall thickness; SWd: septal wall diameter in diastole; SV: stroke volume; TAPSE: Tricuspid annular plane systolic excursion; TRVmax: tricuspid regurgitation maximum; TVR: total vascular resistance. •• not adjusted for MAP ••• not adjusted for BMI.

Table 4
Unadjusted and adjusted associations between PE (n = 57) and cardiovascular structure and function at follow-up. Adjustments were made for maternal age, BMI, MAP and CVD family history. Significant associations (p < 0.05) are marked with asterix (*).

Cardiovascular structure and function 1 or 3 years postpartum	PE Unadjusted b (95 % CI)	p	PE Adjusted b (95 % CI)	p
CARDIAC MORPHOLOGY/ STRUCTURE				
LVIDd (cm)	−0.10 (−0.36 – 0.16)	0.45	−0.02 (−0.29 – 0.25)	0.87
PWd (cm)	0.00 (−0.05 – 0.05)	1.00	0.00 (−0.06 – 0.06)	1.00
SWd (cm)	0.10 (0.05 – 0.15)	<0.01*	0.10 (0.04 – 0.16)	<0.01*
LVM (g)	7.55 (−3.89 – 18.99)	0.19	5.79 (−7.61 – 19.19)	0.39
LVMi (g/m^2) •••	1.62 (−3.74 – 6.97)	0.55	4.02 (−2.74 – 10.78)	0.24
LAVi (mL/m^2) •••	1.55 (−1.97 – 5.08)	0.38	0.72 (−3.03 – 4.46)	0.70
RWT	0.02 (−0.01 – 0.04)	0.14	0.03 (0.01 – 0.06)	0.01*
FUNCTION				
TVR (dyne.s^{-1}cm^{-5}) ••	177.60 (6.26 – 348.95)	0.04*	144.98 (−35.79 – 325.76)	0.11
HR (bpm)	2.00 (−4.22 – 8.22)	0.52	0.03 (−6.51 – 6.56)	1.00
SV (mL) (Doppler)	−4.00 (−9.12 – 1.12)	0.12	−3.82 (−9.77 – 2.13)	0.21
CO (L/minute)	−0.03 (−0.51 – 0.45)	0.90	−0.20 (−0.65 – 0.25)	0.38
LVEF (%) (biplane)	0.00 (−1.81 – 1.81)	1.00	−0.78 (−2.34 – 0.78)	0.33
DIASTOLIC FUNCTION				
E (cm/s)	0.01 (−0.09 – 0.11)	0.84	−0.03 (−0.13 – 0.07)	0.54
A (cm/s)	0.00 (−0.06 – 0.06)	1.00	−0.01 (−0.08 – 0.07)	0.89
E/A ratio	−0.25 (−0.52 – 0.02)	0.07	−0.17 (−0.43 – 0.10)	0.21
E/É ratio	−0.07 (−0.59 – 0.45)	0.79	−0.00 (−0.71 – 0.71)	1.00
MITRAL ANNULAR MOTION				
S' velocity (cm/s)	0.00 (−0.50 – 0.50)	1.00	0.00 (−0.70 – 0.70)	1.00
RIGHT HEART FUNCTION				
TRVmax (m/s)	0.05 (−0.12 – 0.22)	0.56	−0.02 (−0.17 – 0.14)	0.83
S' velocity (cm/s)	0.00 (−1.00 – 1.00)	1.00	0.52 (−0.56 – 1.60)	0.34
TAPSE (mm)	2.00 (0.19 – 3.81)	0.03*	1.82 (−0.26 – 3.90)	0.09

Beta (95 % confidence interval) and p-values for univariate regression analyses. BMI: body mass index; CI: confidence interval; CO: cardiac output; CVD: cardiovascular disease; HR: heart rate; LAVi: Left atrial volume indexed to body surface area; LV: left ventricular; LVEF left ventricular ejection fraction; LVIDd: LV internal diameter in diastole; LVM: left ventricular mass; LVMi: LVM indexed to body surface area; MAP: mean arterial blood pressure; PWd: posterior wall diameter in diastole; RWT: relative wall thickness; SWd: septal wall diameter in diastole; SV: stroke volume; TAPSE: Tricuspid annular systolic excursion; TRVmax: tricuspid regurgitation maximum; TVR: total vascular resistance. •• not adjusted for MAP ••• not adjusted for BMI.

and control groups, but a different SWd distribution for these two groups led to a statistically significant group difference (Table 2 and Supplemental Fig. S2). All previous HDP sub-groups displayed non-significantly lower median E/A ratio and higher rates of E/é outside reference range, compared to controls (Table 2 and Supplemental Table S5).

In univariate regression analyses, a previous HDP or PE history significantly associated with elevated postpartum TVR, TAPSE and SWd (Table 3 and 4 and Fig. 2). After adjustments for sex-independent CVD risk factors, both pregnancy complication groups remained significantly associated with SWd. In contrast, no study groups remained significantly associated with TVR or TAPSE after adjustments for maternal age, BMI and family history of CVD (Table 3 and 4 and Fig. 2). While no study group were significantly associated with RWT in univariate regression analyses, both HDP and PE were significantly associated with RWT after adjustments for sex-independent CVD risk factors (Table 3 and 4).

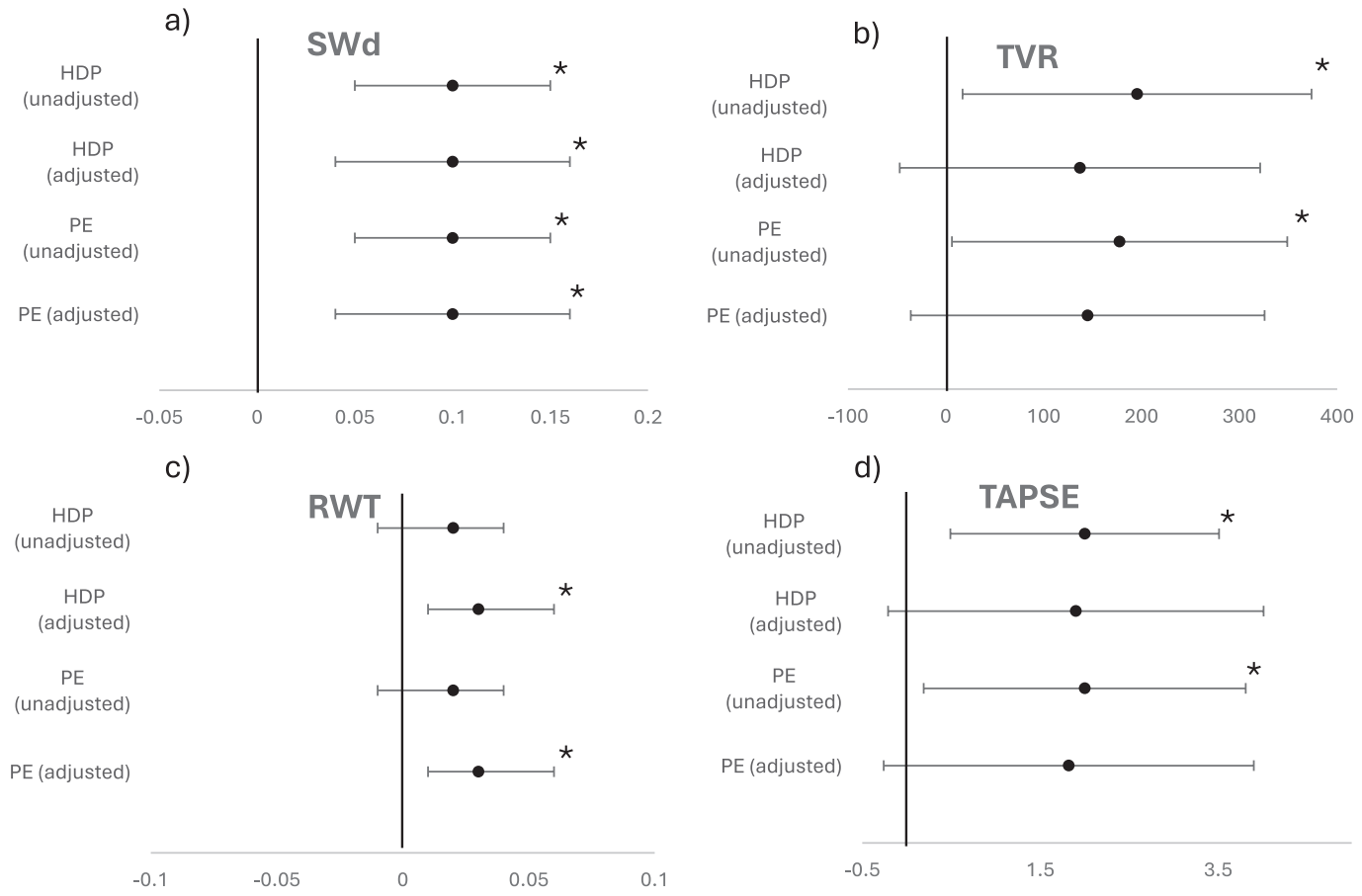

Fig. 2. Uni- and multivariate regression analyses of echocardiography levels in women with previous hypertensive disorders of pregnancy and preeclampsia. Multivariate regression analyses are adjusted for maternal age, BMI, MAP and CVD family history. TVR is only adjusted for maternal age, BMI and CVD family history. Median: filled circles. 95 % confidence interval: horizontal black lines. *$p < 0.05$. BMI: body mass index; CVD: cardiovascular disease; HDP: hypertensive disorder of pregnancy; MAP: mean arterial blood pressure; PE: preeclampsia; RWT: relative wall thickness; SWd: septal wall diameter in diastole; TAPSE: Tricuspid annular systolic excursion; TVR: total vascular resistance.

4. Discussion

Overall, echocardiographic examinations at 1–3 years postpartum demonstrated limited significant differences between the study groups, except from a significantly higher TVR and TAPSE in the previous HDP and PE groups, compared to controls. Following adjustments for sex-independent CVD risk factors, previous HDP or PE were not associated with TVR or TAPSE, but remained significantly associated with echocardiographic markers of hypertrophy, including SWd and RWT.

In our cohort, no woman exhibited evidence of pathological cardiac remodeling in the form of concentric remodeling or eccentric/concentric hypertrophy (Supplemental Table S2),[29] and few women exhibited echocardiographic findings outside reference ranges (Supplemental Table S5).

All previous HDP groups displayed notably higher median TVR, compared to controls, however only significant for the total HDP and the PE groups. TVR is blood pressure-dependent, thus higher TVR in women following HDP may be largely attributed to elevated blood pressure and more adverse cardiometabolic profiles, including more frequent overweight, obesity and metabolic syndrome. This supports our previous report from a similar cohort, showing that higher blood pressures largely explained adverse levels of Pulse Wave Velocity (PWV) one year after HDP, compared to controls.[26]

In our present study, no pregnancy complication group was significantly associated with TVR after adjustments for sex-independent confounders. The associations were mainly explained by a family history of CVD, suggesting a genetic or socioeconomic impact on the stress placed on the cardiovascular system. This aligns with findings suggesting that adverse pregnancy outcomes, including HDP, may reveal an underlying susceptibility to future coronary artery disease risk, driven by shared genetic liabilities.[30]

Despite no pathological cardiac remodeling after HDP in our cohort, we found some significant differences in cardiac structure between our study group. Similarly to previous findings in women up to 26 years following HDP,[22,31] women with previous PE showed significantly higher RWT, compared to controls; consistent with subclinical adverse cardiac remodeling. Both HDP and PE were significantly associated with RWT and SWd after adjustments for sex-independent CVD risk factors. This is well in line with other reports of greater left ventricular wall thickness and ventricular mass within a decade after HDP.[19,20,22] However, median values for both RWT and SWd were within normal range for all groups in our cohort. This contrasts with previous findings of cardiac remodeling in almost 80 % of women with previous HDP and persistent hypertension 8–10 years postpartum.[19] These discrepancies in findings are likely explained by shorter follow-up time and a much more favorable cardiometabolic profile in our cohort (with a 19 % hypertension rate compared to 63 %).[19] Interestingly, the associations between pregnancy complication groups and RWT, in our cohort, were only significant after adjustments for confounders, mostly driven by a CVD family history. This may indicate persistent physiological LV remodeling after PE, and an impact of traditional CVD risk factors on the postpartum normalization process after PE.[18,32,33]

In line with previous studies, we demonstrate increased hypertension rates 1–3 years after HDP.[34,35] Women with persistent hypertension

after HDP are suggested to be more prone to persistent cardiac abnormalities peripartum, including progression towards heart failure.[35] We did not however, reproduce previous findings of increased risk of diastolic dysfunction,[34] left ventricular hypertrophy [31] or increased risk of development of HFpEF after HDP.[10] Differences in sex-independent CVD risk factors likely explain much of these discrepant findings, as several aforementioned studies include patients with diabetes, higher age and higher rates of obesity and hypertension.[10,31]

All median systolic and diastolic markers were within reference ranges for all our study groups. Women with previous HDP and PE displayed significantly higher TAPSE, compared to controls, in contrast with previous findings of lower TAPSE following HDP.[36] Previous HDP and PE were significantly associated with increased TAPSE, but associations were no longer significant following adjustments for sex-independent CVD risk factors, mainly driven by blood pressure. In addition, all HDP sub-groups displayed (non-significant) lower E/A ratio and higher rates of E/é ratio outside reference range, indicating more adverse diastolic function, compared to controls, which is compatible with previous studies.[22,31] No pregnancy complication groups were significantly associated with altered E/A ratio or E/é ratio. This contrasts with previous findings of associations between HDP and left ventricular hypertrophy,[22] even after risk factor adjustments, likely due to prolonged hypertension.[31] The discrepancies in our findings compared to previous findings, may be explained by different cohort sizes and cardiometabolic risk profile, as well as our cohorts younger age and shorter follow-up time, hence a shorter time of high blood pressure exposure to a younger cardiovascular system.

Our study demonstrates that women with HDP display higher pre-pregnant and postpartum sex-independent modifiable risk factors for both HDP and CVD, compared to controls, except for the lower self-reported smoking rate (0 % vs 20 %). Our findings align with previous findings of increased rates of obesity, hypertension and CVD after HDP, suggesting that PE may indicate underlying cardiometabolic disease susceptibility.[37–40] Leading hypotheses explaining the association between HDP and CVD involve shared risk factors and accumulation of risk throughout gestation, which are likely synergistic.[6,41] Whatever the mechanisms, our study supports the notion of pregnancy as a stress-test for future CVD.[6,11]

Recent guidelines recommend screening and intervention following HDP.[5,42–47] However, the timing or optimal content of cardiovascular follow-up is not clear. Our findings of increased risk factors after HDP (e.g., overweight and Stage 2 hypertension) highlight the clinical potential in targeting modifiable CVD risk factors.[48] HDP leads to different and more long-lasting cardiovascular adaptations during pregnancy and postpartum, compared to normotensive pregnancies. The reported postpartum hypertension rates after HDP vary, from up to 50 % of women remaining hypertensive 2–3 months postpartum and 10 % developing chronic hypertension within a year,[18] to our previous [23] and current reports demonstrating 3–19 % hypertension one year following HDP. A cardiovascular risk profile assessment 1–3 years postpartum may represent a favorable timing, because it allows for cardiovascular normalization postpartum and likely identifies persistent or de novo cardiovascular risk factors before CVD becomes manifest. Our study demonstrates limited additional value of echocardiography 1–3 years after HDP in otherwise healthy women, due to generally few abnormal echocardiographic findings. Classical CVD risk factors, like elevated blood pressure and BMI, are more easily identified and more affordable and accessible than echocardiography, hence more feasible across different global settings. Regular screening for chronic hypertension, dyslipidemia, hyperglycemia and overweight/obesity after HDP likely represents a better population-based strategy, in line with national and international expert recommendations.[5,42–48] Systems for risk assessment, enabling optimal CVD prevention after HDP from a young age, are likely necessary to obtain significant long-term health effects across socioeconomic diverse populations.

4.1. Strengths and limitations

Our study is strengthened by a well-defined study cohort including a control group, detailed echocardiographic examinations, comprehensive clinical data and validated pregnancy complication definitions. Women with chronic diseases that may elevate CVD risk (e.g., diabetes) were excluded to more specifically ascertain the effects of HDP. Sensitivity analyses were conducted to ensure that the inclusion of superimposed PE in the HDP group and the assignment of CVD family history did not alter results. Furthermore, this study included a larger cohort, and a more comprehensive presentation of modifiable risk factors compared to many published studies investigating cardiovascular phenotype following HDP. Our echocardiography examinations are strengthened by few experienced echocardiographers and the use of the same apparatus for all participants, ensuring uniform conditions, image acquisition and image analyses. Echocardiographers were unaware of the study participants' pregnancy outcome during the examinations.

Nonetheless, our study has some limitations. We had limited self-reported data on breastfeeding practice of our cohort and did therefore not adjust for breastfeeding. We however assume that most women had breastfed for at least 6 months, in line with Norwegian population data.[49] We did not include any women who were still lactating at the 1 or 3 year follow-up, as lactating could impact cardiometabolic measures. Breastfeeding at this time is however uncommon in Norway. Several epidemiological studies have confirmed the benefits of breastfeeding on maternal cardiovascular health, with multiple potential mechanisms being discussed.[50]

A limited availability of the echocardiographic examinations may also have introduced selection bias. The use of one- and three-year data may introduce bias, but the main conclusion remained the same, following sensitivity analyses for one-year data. We did not perform regression analyses for the GH group, due to the limited sample size ($n = 8$), reducing the statistical power for this subgroup. Regression analyses addressed for confounding factors, although some confounding is likely still not accounted for. Ethnicity was not adjusted for due to large homogeneity in our cohort (95 % White ethnicity). Our study also included high rates of high education (representative of the general Norwegian population), living in a high-income country with free-of charge antenatal and obstetric care, limiting the external validity to more heterogeneous populations and different access to healthcare. Despite these limitations, we observed signs of echocardiographic changes short time after HDP in this presumed healthy population, but to a smaller degree when compared to reports from other populations.

5. Conclusion

Sex-independent, classical risk factors explained much of the subclinical echocardiographic cardiovascular changes after HDP. However, previous HDP and PE remained significantly associated with markers of cardiac hypertrophy and increased afterload 1–3 years postpartum, after adjustments for sex-independent cardiovascular risk factors. Women after HDP displayed more adverse cardiometabolic profiles, including more adverse blood pressure and cardiovascular phenotype. No woman in our cohort met formal criteria for pathological cardiac remodeling.

In our clinically healthy and asymptomatic population 1–3 years after HDP, echocardiography did not improve CVD risk stratification beyond classical risk factors. Our findings of adverse CVD risk profiles underscore the need for enhanced cardiovascular follow-up after HDP. Early identification of modifiable cardiovascular risk factors is important to provide disease prevention and treatment from a young age, when preventive measures likely are most effective, in clinically healthy women.

Data statement

Due to ongoing clinical follow-up, the sensitive nature of the study

data material and the patient informed consents (including ethical body approval) access to data material is restricted. Requests for access to portions of the anonymized dataset may be directed to the corresponding author.

Ethics statement

This study was conducted in line with the Helsinki Declaration and approved by the Regional Committees for Medical Research Ethics South East Norway (Reference: 2013/2092 REK Sørøst C, February 3rd, 2014). Participants were women ≥ 18 years who had delivered a singleton at Oslo University Hospital, Ullevål, Norway one or three years previously. All participants received written and oral information and provided written informed consent.

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Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.preghy.2025.101404.

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