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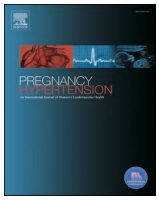

Review article

Hypertensive disorders of pregnancy and long-term maternal cardiovascular risk: Bridging epidemiological knowledge into personalized postpartum care and follow-up

Anne Cathrine Staff [a,b,*], Maria Laura Costa [c], Ralf Dechend [d,e], Daniel P. Jacobsen [a,b], Meryam Sugulle [a,b]

[a] Faculty of Medicine, University of Oslo, PB 1171, Blindern, 0381 Oslo, Norway
[b] Division of Obstetrics and Gynaecology, Oslo University Hospital, PB 4956 Nydalen, 0424 Oslo, Norway
[c] Department of Obstetrics and Gynecology, Universidade Estadual de Campinas (UNICAMP), Campinas/SP, Brazil
[d] HELIOS Clinic, Berlin, Germany
[e] Experimental and Clinical Research Center, Charité Medical Faculty and Max-Delbrueck Center for Molecular Medicine, and HELIOS Clinic Berlin, Germany

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ABSTRACT

Cardiovascular disease (CVD) is globally the leading cause of death and disability. Sex-specific causes of *female* CVD are under-investigated. Pregnancy remains an underinvestigated sex-specific stress test for future CVD and a hitherto missed opportunity to initiate prevention of CVD at a young age.

Population-based studies show a strong association between female CVD and hypertensive disorders of pregnancy. This association is also present after other pregnancy complications that are associated with placental dysfunction, including fetal growth restriction, preterm delivery and gestational diabetes mellitus. Few women are, however, offered systematic cardio-preventive follow-up after such pregnancy complications. These women typically seek help from the health system at first clinical symptom of CVD, which may be decades later. By this time, morbidity is established and years of preventive opportunities have been missed out. Early identification of modifiable risk factors starting postpartum followed by systematic preventive measures could improve maternal cardiovascular health trajectories, promoting healthier societies.

In this non-systematic review we briefly summarize the epidemiological associations and pathophysiological hypotheses for the associations. We summarize current clinical follow-up strategies, including some proposed by international and national guidelines as well as user support groups. We address modifiable factors that may be underexploited in the postpartum period, including breastfeeding and blood pressure management. We suggest a way forward and discuss the remaining knowledge gaps and barriers for securing the best evidence-based follow-up, relative to available resources after a hypertensive pregnancy complication in order to prevent or delay onset of premature CVD.

1. Pregnancy as a stress test for women's cardiovascular health

Cardiovascular disease (CVD) represents a leading cause of death and disability in women, and is also responsible for the most years of potential life lost [1]. Women with CVD remain "understudied, underrecognized, and undertreated", as recently summarized by the Lancet women and cardiovascular disease Commission [2]. A 2023 publication from The New England Journal of Medicine concluded from a large harmonized global cohort of individual data from >1.5 million participants, that 53 % of cardiovascular disease and 22 % of deaths in women were attributable to 5 well-known modifiable risk factors (body mass index, systolic blood pressure, non-high-density lipoprotein cholesterol, smoking and diabetes) [3]. The study found sex differences in how these risk factors affected cardiovascular disease and general death rates, indicating sex-specific causes of female CVD. These may include a range of reproductive factors from menarche to menopause. As suggested by the Lancet Commission [2], risk factors for female CVD can be divided into three general categories. The first category includes the well-established (modifiable) risk factors that affect both sexes, but that may affect women differently compared to men (e.g. hypertension,

* Corresponding author at: Division of Obstetrics and Gynaecology, Oslo University Hospital, Kirkeveien 166, Post Box 4956 Nydalen, 0424 Oslo, Norway.
E-mail addresses: uxnnaf@ous-hf.no, a.c.staff@medisin.uio.no (A.C. Staff).

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dyslipidemia, diabetes, obesity, unhealthy diet, sedentary lifestyle, smoking or tobacco use). The second category includes the female sex-specific risk factors (e.g. polycystic ovary syndrome, hypertensive disorders of pregnancy, preterm delivery, gestational diabetes mellitus, premature menopause) or risk factors that affect more women than men (e.g. systemic inflammatory and autoimmune disorders) [2]. The third category includes under-recognized risk factors that can be related to gender and a woman's environment (e.g. psychosocial risk factors, abuse and partner violence, socioeconomic deprivation, poor health literacy) [2]. Although all these factors may be important in the pathophysiology of female CVD, we would argue that pregnancy represents among these risk factors a "low hanging fruit" in assessing a premenopausal woman's risk for CVD. Most women will deliver at least one child during their life and most women also give birth in a health facility, making the result of the pregnancy as a "stress test" available for most women and their health providers across the world. We and other authors therefore argue that pregnancy can be seen as a clinically underexploited stress test and a premenopausal screening tool for cardiovascular risk in women [4–10].

As recently reviewed, women differ from men in many aspects of CVD [11]. Women often experience more diverse and unspecific symptoms than men, which may lead to silent or missed myocardial infarction [11]. Women with clinical coronary artery disease may present with more "atypical" symptoms, delaying diagnosis and likely resulting in poorer outcomes [12]. Women with acute coronary syndromes have fewer lipid-rich atheromas and a lower incidence of plaque rupture than men, but are more prone to microvascular dysfunction, and respond differently to cardiovascular stressors [13]. Sex hormones and sex-specific molecular mechanisms influence glucose and lipid metabolism, as well as cardiac energy metabolism and function [14].

Hypertension may represent a more important risk factor for CVD in women than in men, supporting that early detection and treatment of elevated blood pressure may be especially important in women. A population-based study from Norway showed that systolic blood pressure elevation is important for permanent atrial fibrillation complications in women, but not in men [15]. The main causal and modifiable atherosclerotic CVD risk factors in both sexes are hypertension, dyslipidemia, adiposity, cigarette smoking, and diabetes mellitus [16]. However, females exhibit a more accelerated rise in blood pressure than men already from their 30′s [17], two decades before menopause, at which point women catch up with men in hypertension rates. Furthermore, CVD is associated with elevations from lower systolic blood pressures in women than in men, possibly due to smaller arterial diameter relative to body size [18]. The rate of immediate postpartum hypertension after hypertensive disorders of pregnancy (HDP), a significant risk contributor, is unknown in most countries, including Norway, and implementation of guidelines for postpartum follow-up is scarce or lacking [19].

Observational studies from USA and UK reported already in 1976 increased long-term cardiovascular morbidity in women following eclampsia and preeclampsia [20,21]. These were followed by epidemiological population-based studies from 2001 and onwards, all from Northern Europe and all demonstrating a strong association between female CVD and HDP [22–25]. Most studies report a 2-fold increased risk for a broad range of CVD after gestational hypertension or term preeclampsia, as summarized in [8]. The risk is particularly high after more severe forms of disease, e.g. preeclampsia with preterm delivery and/or fetal growth restricted baby, recurrent preeclampsia [19,26–29]. Systematic reviews and meta-analyses conclude that not only women with previous gestational hypertension or preeclampsia are at increased risk for a broad range of CVDs, but that this increased risk also applies to females who have had pregnancies complicated by gestational diabetes mellitus, preterm birth, stillbirth and placental abruption [26,30–32]. Most data come from high-income settings, and therefore the impact of risk factors associated with adverse pregnancy outcomes on long-term maternal health is unknown in low and middle-income countries. These epidemiological findings support the view that women with a broader array of pregnancy complications, mostly linked to placental dysfunction [33], should be assessed and offered risk factor management beyond the postpartum period [34].

2. Pathophysiological mechanisms associating adverse pregnancy outcomes with future CVD

The pathophysiological mechanisms behind the associations between a previous adverse pregnancy outcome like preeclampsia and increased risk for maternal CVD are likely multiple, heterogeneous and synergistic [8,35]. Many risk factors for preeclampsia and CVD are shared (e.g. obesity, hypertension, dyslipidemia, increasing age) [36], which supports the shared risk factor hypothesis. Still, population-based studies have demonstrated that women developing hypertensive disorders have an increased risk for CVD even after adjusting for prepregnancy obesity, blood pressure and dyslipidemia [37–39], supporting that the risk may be mediated by more than shared prepregnancy risk factors.

Another main hypothesis, which does not exclude the relevance of the first, is that an adverse pregnancy outcome also induce or aggravate existing risks. Several population-based studies support a pregnancy-mediated risk in that there is a higher risk of long-term maternal cardiovascular diseases with more severe hypertensive disorders of pregnancy [22–27], such as complicated by fetal growth restriction [27], as well as with recurrent preeclampsia [29,40]. We have argued that long-term maternal CVD may partly be driven by proinflammatory products from a dysfunctional placenta during pregnancy. A dysfunctional placenta, as seen in preeclampsia and fetal growth restriction, mediates excessive release of inflammatory factors to the maternal circulation, including antiangiogenic signalling molecules [8]. We suggest that clinical features of preeclampsia, including a dysregulated angiotensin system [41], dysregulated circulating angiogenic proteins [8], hypertension, dyslipidemia and excessive inflammation and insulin resistance, all could contribute to accelerated vascular dysfunction towards more premature development of atherosclerosis [42], microvascular disease [43] and heart failure [44]. In our recent work on fetal microchimerism, we have found that these long-lasting fetal cells are present at increased rates in maternal circulation across pregnancies with increased placental dysfunction, including preeclampsia. Our concept is that these cells may have the potential to contribute to the association between preeclampsia and long-term maternal CVD, through immunological and inflammatory responses [45].

Animal studies support that pregnancy complications like preeclampsia have long-term impact of maternal cardiovasculature. This includes persistently enhanced postpartum vascular response to injury [46] and enhanced sensitivity of smooth muscle cell mineralocorticoid receptors to activation [47].

Following HDP, women are already from the first year postpartum at increased risk of chronic hypertension [48–50], which represents a major modifiable risk factor for CVD. However, mediation analyses suggest that chronic hypertension only explains 50–60 % of the increased heart failure and coronary artery disease risks following HDP [51], suggesting that other features like excessive inflammation and microvascular dysfunction could play a role [44], features that are typically involved in the preeclampsia pathophysiology [33,52]. Likewise, the increased risks after HDP for developing diabetes mellitus [25] and end-stage renal disease [53] are well established, both representing important mediators of maternal cardiovascular morbidity and mortality.

There is emerging evidence that also offspring exposed to preeclampsia in utero may be at increased risk of cardiovascular disease later life, as reviewed previously [35,54]. The mechanisms driving the relationship between preeclampsia and offspring CVD are likely very complex, due to potential synergy of in utero programming, preterm birth, shared alleles with the mother, as well as shared family lifestyle

Table 1
Examples of guideline for clinical follow-up for prevention of future cardiovascular disease after adverse pregnancy outcomes. ACOG: American College of Obstetricians and Gynecologists; AHA: American Heart Association; BMI: body mass index; BP: blood pressure; FEBRASGO: Brazilian Federation of Gynecology and Obstetrics Associations; FGR: fetal growth restriction; FIGO:;GDM: gestational diabetes mellitus; HDP: Hypertensive disorders of pregnancy; ISSHP: The international Society for the Study of Hypertension in Pregnancy; NGF: The Norwegian Society of Gynecology and Obstetrics; NICE: The National Institute for Health and Care Excellence; OGT: Oral glucose tolerance test; PE: preeclampsia; SGA: Small for gestational age.

Publishing body	Last updated guideline	After Adverse Pregnancy Outcomes	After Hypertensive Disorder of Pregnancy
AHA[1]	2021	**Adverse pregnancy outcomes** (HDP, preterm delivery, GDM, SGA, abruption, pregnancy loss): Healthy lifestyle and breastfeeding recommended. CVD risk assesment and follow-up recomenned (not specified the timing postpartum). Refers to AHA 2011 guideline [2] (assess BP, lipids, blood glucose, BMI)	
ACOG (Practice Bulletin)[3]	2020		**GH and PE:** Preventive strategies and vigilance to be considered by patients and heakth care providers (healthful weight, exercise, diet, smoking cessation)
Brazilian (FEBRASGO)[4]	2019		**After PE:** Avoid early discharge (hospital monitoring recommended at least 3 days postpartum). Reassessment 7 days postpartum. Recommends multidisciplinary follow-up, including control of BP, renal function lipid and glycemic profiles, as well as lifestyle change (physical activity, balanced diet).
Canadian Cardiovascular Society[5]	2021	**Adverse pregnancy outocomes** (HDP, GDM, preterm birth, stillbirth, low birth weight infant, or placental abruption); **Screening** for elevated blood lipids in the late postpartum period. Counsel women about increased risk of lifetime CVD, receommendation of healthy lifestyle.	
CoLab[6] **(**Flow-chart Figure in Appendix A**)**	2016	**Adverse pregnancy outcomes** (PE, GH, FGR, GDM); Triage at 6–12 weeks postpartum, including a simple CVD risk assessment) at community health follow-up (general practitioner) or specialist according to clinical outcome severity. Earlier follow-up of not normalised BP and glucose homeostasis at discharge). Further follow-up and patient education depending on assessed CV risk.	
Dutch multidisciplinary guideline[7]	2016	**Adverse pregnancy outcomes** (PE, GH, preterm delivery, SGA); at 6 weeks postpartum to 49 years: recommended optimization of lifestyle. At 50 years (follow-up only recommended after PE): full CV risk profiling.	
FIGO[8]	2023		**Following HDP: Optimally:** Multidisciplinary approach. Lifestyle recommendations, including *breastfeeding* and lifestyle modifications (incl. exercise recommendations)**.** **First year: Optimally:** follow-up first week, at 6–12 weeks, 6 and 12 months after birth**.** Undergo BP assessment every 6–12 months and CV screening every 4–6 years. **In limited resource settings**: annual BP assessment, starting within the first year postpartum. **Follow-up visits**: history taking and physical examination (incl. BP; BMI, waist circumference), OGT, lipid profile, and urine protein.
ISSHP[9]	2021		**All hypertensive pregnancies:** BP monitored at least once day 3–7 postpartum. *Breastfeeding* recommended. Clinical assessment 3 months postpartum, and optimally at 6 months postpartum (recommend lifestyle changes if BP $\geq$ 120/80). Recommended annual medical review the first 5–10 years postpartum, as well as a healthy lifestyle, aiming for BP $<$ 120/80.
NGF[10]	2020	**After GDM, FGR or Preterm delivery**: similar recommendations as for HDP.	**After PE, GH:** Patients with unresolved proteinuria or hypertension at discharge to attend intensified follow-up. If preexisting renal or CV disease: continue check-ups as before. CV triage at 6–12 weeks postpartum with simple CV risk evaluation (BP, lifestyle evaluation) and recommended healthy lifestyle (incl.physical activity of at least 150 min weekly, healthy diet, normal weight, avoid smoking, limit alcohol intake). OGT at 3–6 months postpartum (increased risk for DM after HDP). Renewed CV triage at 1 years postpartum, adding lipid assessment and HbA1c (glucose intolerance screening). Further follow-up (BP, lifestyle assessment) depending on risk factors, but at least every 5 years until 50 years of age.
NICE[11]	2019 (added updates in 2023)		**HDP**: Offer medical review 6–8 weeks postpartum (GP or specialist). Agree a care plan (for follow-up care and medical review, BP monitoring frequency). **Information:** Increased risk of hypertension and its complications later in life. **Advice:** Normal maternal weight (BMI 18.5–24.9 kg/m^2) and healthy diet.

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factors after birth.

3. Current clinical follow-up and CVD prevention strategies after HDP

Current CVD risk calculators are not useful for young women after HDP in risk stratification for primary cardiovascular follow-up and prevention, as these calculators neglect the impact of pregnancy complications and often are not applicable below 40 years of age [50]. In older women, tests provide more adequate CVD risk scoring [55], but at this age preventive effects are likely less effective [56]. Population-based studies from Denmark [48] and Norway [57] have shown that one third of women develop hypertension within 10 years following an HDP. Middle age may therefore be too late to start intensified follow-up and prevention of CVD risk. Pregnancy represents a life-changing event where women are particularly interested in their own and their offspring's health. This setting may therefore represent an optimal time point to empower women to improve their own cardiovascular health for life.

The American Heart Association's guideline from 2011 [58] was important for raising awareness in both cardiology and obstetric communities [59] about the importance of adverse pregnancy complications in predicting increased risk for CVD in women. Most guidelines prior to 2016 were lacking specific follow-up suggestions after HDP, as revised by us in [8] (in the Paper's Supplemental Material).

Table 1 presents examples of suggestions for cardiovascular follow-up strategies after HDP.

The World Health Organization (WHO) "Prevention and treatment of pre-eclampsia and eclampsia" guideline from 2011 [60] is not listed in this Table. This is because this WHO guideline does not address long-term cardiovascular health, except for mentioning there is a need for further studies of treatment schedules for women with postpartum hypertension, "including timing of stopping treatment". Unfortunately, none of the ensuing six updates of this WHO guideline have addressed the long-term cardiovascular risk for these women or proposed a follow-up strategy.

An international (CoLab) guideline from 2016 went further than previous guidelines, in being more specific about triaging for follow-up postpartum [8]. A Dutch multidisciplinary guideline from the same year [61] suggested similar follow-up principles after preeclampsia in recommending optimization of modifiable risk factors after pregnancy, but suggested 50 years of age (around menopause) before any cardiovascular risk profiling after a preeclamptic pregnancy. This approach would represent a missed opportunity for early prevention for the many women developing hypertension within the first ten years after pregnancy [48]. The 2016 CoLab flow-chart for follow-up after pregnancy complications acknowledges other placenta dysfunction-related pregnancy outcomes than preeclampsia, including also a recommendation for a screening/follow-up strategy the first year postpartum after pregnancies complicated by gestational hypertension, fetal growth restriction, or gestational diabetes mellitus (Supplemental Fig. 1) [8]. A more simplified postpartum screening program was thereafter developed for Norwegian obstetric guidelines and preeclampsia patient brochures [62], as well as presented in a US-based review paper [19].

The ISSHP (International Society for the Study of Hypertension in Pregnancy) guidelines prior to 2018 [63] did not mention prevention of long-term maternal disease following HDP. The guidelines were updated from 2018 on this topic [64] and recommendations were further specified in the last 2021 revision [65].

FIGO (International Federation of Gynecology and Obstetrics) launched in 2019 a Postpregnancy Initiative: Long-term Maternal Implications of Pregnancy Complications-Follow-up [66]. FIGO recommended a follow-up 6–12 weeks after a pregnancy complicated by HDP or any other placenta-associated pregnancy complication, including blood pressure measurements and to consider screening for other risk factors. The screening for other risk factors, including blood lipid screening, is further elaborated in an updated 2023 paper [67]. FIGO also suggests a collaboration between obstetricians and general practitioners following delivery and during the first year postpartum [67], as suggested in the CoLab flow chart [8].

Several suggestions exist for postpartum health records, freely available for patients as well as their health carers, such as the 2024 ISSHP supported follow-up record from the US Preeclampsia Foundation [68], the Canadian "The MotHERS Program" (Supplemental Fig. 1, [69]) as well as the recent "Pregnancy Passport" from FIGO [70], with a QR code to access more information for patients after delivery and before discharge from the hospital.

In general, the recent guidelines are more detailed and comprehensive than previous ones in their follow-up suggestions for CVD prevention after HDP and other pregnancy complications. We do however not know what the optimal follow-up system is, and local health systems may differ largely in their abilities and capacities for any follow-up after HDP. Access to health care of good quality remains a problem not only in low-income countries, but also in high-income countries without a health system that secures equity and universal access to health services. This highlights the need for available patient health information after such adverse pregnancy outcomes, so that females in any societies are empowered to act on modifiable risk factors from an early time in life, when early and preclinical cardiovascular dysfunction may still be reversible.

4. Pregnancy and modifiable risk factors that may be underexploited in in the long-term prevention of CVD in females

4.1. Prepregnancy care: as healthy as possible prior to pregnancy

As it is likely that a pregnancy complicated by HDP and/or gestational diabetes also contributes to the long-term increased

cardiovascular risk in the woman, an important prevention strategy would be to reduce the risk for these outcomes or their severity. As recently highlighted by the American Heart Association, all fertile women should ideally prior to any pregnancy reduce their risk for developing HDP and placental dysfunction [71]. A healthy lifestyle, including a normal weight and diet, abstinence from smoking, alcohol, narcotic substances, as well as being physically active are important measures for preventing CVD, as well as for preventing HDP and gestational diabetes.

Conversely, smoking is associated with reduced risk of preeclampsia [72]. This well-known "smoking paradox" has been linked to carbon monoxide effects on placental production of proinflammatory angiogenic factors [72], but should not obscure the fact that smoking cessation prior to pregnancy reduces the risk for multiple adverse pregnancy outcomes, including fetal growth restriction and intrauterine death, as well as the most severe preeclampsia cases [72]. All epidemiological studies therefore support the importance of recommending smoking cessation prior to pregnancy. Also, women with any form of chronic disease, including renal disease/renal transplant, prepregnancy diabetes, chronic hypertension, autoimmune diseases, overweight or obesity should be recommended to optimize their health before getting pregnant. Whether such prepregnancy counselling is performed is not known, neither if women adhere to any received recommendations or plan their pregnancies according to whether they have achieved such risk reduction or not. In such an antepartum care context, it is especially worrying that a large rate of pregnancies are reported to be unplanned, especially in populations of low socio-economic status [73].

Some studies indicate that periconception lifestyle interventions, combining e-health and personalized face-to-face counselling, may be efficient in promoting a healthier lifestyle, especially if partners also participated in the program [74]. Mobile app lifestyle interventions have also been shown to improve nutrition in women before and during early pregnancy [75].

4.2. Screening and prevention of early-onset preeclampsia

Another important strategy for reducing HDP risk is first trimester screening for preeclampsia, as recommended by obstetric guidelines, including from FIGO [67] and ISSHP [65]. In several high-income countries this now includes assessment of placenta-associated biomarkers at gestational week 11–14, including level of maternal placenta growth factor (PlGF) and umbilical artery pulsatility index, mean arterial pressure (MAP) and classical medical and obstetric risk factors for preeclampsia [76]. Several guidelines recommend this first-trimester screening to be based on classical maternal factors and MAP, if the resources are limited [65,67]. If this screening evaluates the woman being at high risk, she is recommended to take 100–162 mg aspirin daily to reduce the risk for preeclampsia. It is likely, but not evidence-based epidemiologically, that since fewer women develop early-onset preeclampsia with this aspirin prophylaxis strategy, these same women will also reduce their risk for long-term CVD. Whether the risk of future CVD in women developing preeclampsia despite using aspirin differs from the future CVD risk in women developing preeclampsia without aspirin prophylaxis, is not known.

After a pregnancy complication like HDP, it would be beneficial for any woman to reduce her risk factors before embarking on a new pregnancy. Thus, access to safe contraception is an important priority for all populations, and Long-acting reversible contraceptives (LARC) are generally recommended and are well tolerated. Recent data from Latin America has demonstrated that low socioeconomic status and young age are risk factors for inadequate use of safe contraception, including LARC. Overall, among sexually active women, LARC use was less than 5 % in 13 of the 23 countries in the study [77].

4.3. Stopping preeclampsia earlier?

Some observational studies have indicated that preeclampsia duration could impact maternal long-term CVD [78], but a recent UK randomized trial testing prompt or delayed delivery did not show any difference in cardiovascular outcomes 6 months after delivery [79]. In general, the risk of severe maternal obstetric complications increases by prolonging a preeclamptic pregnancy, but a very preterm delivery also has the implicit risk of short- and long-term complications for the offspring. The ISSHP guideline recommends starting the delivery process in any woman with preeclampsia at gestational week 37 [65], to reduce the risk of severe complications during pregnancy.

4.4. Blood pressure control and follow-up plans after birth

Although it is recommended to return to normotension following pregnancy for any woman, this is not always achieved in the postpartum period after new-onset hypertension during pregnancy. Antihypertensive therapy that is started antepartum is recommended continued after birth [65], and gradual discontinuation is done according to whether target blood pressure is achieved or not (e.g. 85 mmHg diastolic) [65]. As the ISSHP guidelines recommend, blood pressure should be measured at least once during day 3–7 in the postpartum period of any woman, where it is likely to be highest after birth [65]. As many women have been discharged at this time from hospital, postpartum preeclampsia may thus be missed in some previously normotensive women, as may aggravation of hypertension in those who have undergone preeclampsia and gestational hypertension. A planned transfer of follow-up is therefore important after discharge after birth in a woman with HDP, which may include hospital/specialist outpatient and/or community health system follow-ups according to medical needs and resources available [8].

Home monitoring of blood pressure (with validated devices) may also be a way to go for some populations, in helping to achieve normotension after birth. A recent randomized UK trial demonstrated better blood pressure control at 9 months postpartum (24-hour mean diastolic blood pressure) after preeclampsia or gestational hypertension, in a group that used self-monitoring and physician-guided titration of antihypertensive medication compared to usual outpatient care [80]. The same study also showed that the intervention group had more favorable changes in cardiovascular structure and function [81], which are associated in general with more favorable cardiovascular outcomes.

4.5. Breastfeeding as a cardiovascular modulator

There are many good reasons for strongly supporting women to breastfeed, whenever possible, but it may be of additional importance as a cardio-preventive measure after HDP (or any other pregnancy disorders that are linked to increased CVD risk) [65]. A recent systematic review and meta-analysis of 8 studies, including >1.1 million parous women, concluded that breastfeeding was associated with reduced maternal risk of CVD outcomes [82]. Pooled multivariable-adjusted hazard ratios comparing parous women who ever breastfed to those who never breastfed were 0.89 for CVD (95 % CI, 0.83–0.95), 0.86 for coronary heart disease (95 % CI, 0.78–0.95), 0.88 for stroke (95 % CI, 0.79–0.99) and 0.83 for fatal CVD (95 % CI, 0.76–0.92).

The mechanisms for the association between breastfeeding and a reduced risk of maternal CVD have however been debated. For instance, socioeconomic status influences both breastfeeding behavior and CVD risk [83]. Some authors suggest a beneficial "resetting" of the maternal metabolism after delivery by breastfeeding, as the metabolic changes induced by pregnancy (e.g. accumulation of visceral fat, insulin resistance and dyslipidemia) appear to reverse more quickly, and more completely, with lactation [84].

4.6. Weight normalization

Guidelines recommend women to aim for an ideal body weight after HDP [65], which is challenging for many, especially for those who were overweight or obese before pregnancy [85]. Also, overweight and obese women are more likely to exceed the recommended weight gain recommendations during pregnancy [85], adding to the postpartum weight reduction challenges in this group. High BMI is however a very strong and modifiable risk factor for both CVD and development of hypertensive disorders in the next pregnancy. Postpartum weight retention (defined as the weight difference between preconception and weight at 12 months postpartum [85]), remains a problem for many women. Efforts should be made to support women in a realistic weight reduction in order to reach a normal weight. A recent randomized control trial from Norway supports that postpartum weight retention can be avoided in intensified follow-up after pregnancy. The small trial included obese women at 8 weeks postpartum (n = 29). The dietary treatment, compared to ordinary postpartum care, resulted in higher weight loss and reduction of waist circumference as well as lower fasting blood glucose at 12 months postpartum [86].

4.7. Postpartum care plan established before discharge from hospital

As supported by the CoLab flowchart [8] and the more recent guidelines from FIGO [67,70] and ISSHP [65], the postpartum follow-up should be started up shortly after delivery in any woman with HDP. An individual plan for follow-up and at which level (specialist or general community health care system) should ideally be made, to ensure efficient follow-up without a large unnecessary burden on the health system and the individual. In some settings, the maternal follow-up for CVD prevention could ideally be linked with the appointments that are made for the baby's follow-up and vaccination program, which is not done today in most countries. Another way of integrating health follow-up visits would be to integrate cardiovascular follow-up at a general practitioner for women after HDP to general screening programs, such as cervical or breast cancer screening programs, as suggested by us previously [8].

5. The way forward

5.1. Better precision medicine by improved prediction strategies

There are several unresolved aspects that represent potential barriers to optimal prevention strategies for CVD after HDP. One is that we do not know how to best identify women at highest risk for CVD after HDP, except for the associations between severity of pregnancy outcomes and risk for CVD. Further research into surrogate markers of cardiovascular health in women with a history of preeclampsia is thus highly warranted.

Our recent study adds support to the concept of crosstalk between cardiovascular and placental tissues, by demonstrating a correlation between dysregulated CVD biomarkers and clinical proxies of placental dysfunction [49]. These findings are also in line with the concept of a stressed placenta as the culprit in the development of preeclampsia [33,52]. Our finding of circulating higher postpartum GDF-15 levels in women who had undergone early-onset preeclampsia compared to healthy controls is an example of a promising biomarker that could be tested for CVD risk stratification in premenopausal women after HDP [49]. In the future, further stratification on maternal risk for CVD, for example by adding such CV-related biomarkers to today's inadequate CV risk assessments [50,59], may potentially better guide the level of follow-up (specialist vs. community health services), its content and timing, for optimal primary prevention of CV events. This could include creating an easily available risk calculator for young women that takes their pregnancy history into account, and that summarizes all potential modifiable risk factors they can address to lower that risk.

The pathophysiology and risk factors for developing HDP and CVD are multiple and heterogeneous [8,33]. There is likely no "one size fits all" that applies to a cardio-preventive long-term follow-up of all women after HDP. As an example, a Norwegian epidemiological study showed that women who go on to have a normotensive, uncomplicated pregnancy after a first pregnancy with term preeclampsia, only have very modestly elevated cardiovascular death risk [87] compared to women without any history of preeclampsia. In contrast, women delivering preterm preeclampsia, have repeatedly been shown to have the highest long-term cardiovascular morbidity and mortality [8]. It is likely that the first partum follow-up year would reveal clinical risk factors (e.g. hypertension, dyslipidemia, diabetes, metabolic syndrome, obesity), that would help to individualize the frequency and content of the cardio-preventive follow-up.

5.2. Inequities in opportunities for a healthy lifestyle and access to healthcare of high quality

Prevention of CVD should be a global priority, as incidence and costs are rising [88]. The access to good quality antenatal, obstetric and postpartum care is unequally distributed across the world, and even within high-income regions and countries. Moreover, the opportunity to lead a healthy lifestyle may be restricted for many persons. There is a striking lack of long-term cardiovascular follow-up data after HDP in low-income countries, where also the maternal mortality and morbidity rates are the highest. Human rights include opportunities for a safe reproductive health, which will benefit the mother as well as offspring, both short- and long-term.

An interdisciplinary approach, especially during the first year peripartum, is likely needed to offer every woman with adverse pregnancy outcomes the best opportunity to take care of her long-term cardiovascular health. Some guidelines have suggestion for such shared responsibility for follow-up [67], including personalizing level of care according to severity of individual risk factors [8]. All health carers should join their efforts in facilitating a personalized and sustainable program for follow-up after pregnancy complications such as preeclampsia.

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Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.preghy.2024.101127.

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