Case Reports in Women's Health 40 (2023) e00569

Contents lists available at ScienceDirect

Case Reports in Women's Health

journal homepage: www.elsevier.com/locate/crwh

Postpartum subarachnoid hemorrhage – questions on gestational hypertension diagnosis and treatment threshold: A case report

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ARTICLE INFO

Keywords:
Postpartum subarachnoid hemorrhage
Gestational hypertension
Hypertensive disorders of pregnancy

ABSTRACT

Hypertensive disorders of pregnancy (HDP) are a leading cause of morbidity and mortality for pregnant patients, but how aggressively to address non-severe hypertension in pregnancy remains controversial. The American College of Obstetrics and Gynecology (ACOG) currently recommends a blood pressure treatment threshold of 140/90 mmHg during pregnancy. However, 2017 American College of Cardiology/American Heart Association (ACC-AHA) guidelines define stage 1 hypertension by blood pressures of >130/80 mmHg within the general population. There is now an understudied population of pregnant patients considered to have stage 1 hypertension by ACC-AHA guidelines but who do not meet the treatment threshold by ACOG's standards. This article presents a patient who met ACC-AHA-defined stage 1 hypertension throughout her pregnancy and went on to develop severe hypertension and a postpartum subarachnoid hemorrhage (SAH) secondary to venous hemorrhage. She presented to the emergency department 17 days postpartum complaining of an extreme headache and with a blood pressure of 230/125 mmHg. Magnetic resonance imaging showed SAH in the parietal region adjacent to the superior sagittal sinus. Magnesium and labetalol were administered followed by a clevidipine drip. The patient was continued on antihypertensives and made a full recovery. This article's objective is to draw attention to the urgent need for increased clarity of practice guidelines, consensus between societies, and further study of peripartum health outcomes for pregnant patients defined as having stage 1 hypertension by 2017 ACC-AHA criteria.

1. Introduction

Postpartum subarachnoid hemorrhage (SAH) is a rare yet devastating pregnancy complication. Maternal stroke accounts for 5%–12% of all maternal deaths [1]. Up to 66% of maternal strokes are hemorrhagic, and these are often associated with hypertensive disorders of pregnancy (HDP) [2].

HDP have become increasingly common in the United States and their management presents a challenge for clinicians. Data suggest that many deaths attributable to maternal stroke may be prevented with aggressive blood pressure treatment [2]; however, unlike in non-pregnant hypertensive patients, there is no consensus on ideal blood pressure targets for patients with HDP [2]. While it is widely accepted that severe hypertension (>160/110 mmHg) in pregnancy must be urgently controlled, how aggressively to address non-severe hypertension remains controversial.

Based on a 2022 practice advisory, the American College of Obstetrics and Gynecology (ACOG) recommends a blood pressure treatment threshold of 140/90 mmHg for pregnant patients [3]. However, as of 2017, the American College of Cardiology/American Heart Association (ACC-AHA) defines stage 1 hypertension by blood pressures of >130/80 mmHg within the general population [4]. To date, there has been no large trial examining prenatal or peripartum health outcomes for pregnant patients defined as having stage 1 hypertension by the 2017 ACC-AHA criteria. This case report presents a patient who met ACC-AHA-defined stage 1 hypertension and went on to develop a SAH. Its goal is to stimulate further discussion surrounding gestational blood pressure guidelines and call for further study, increased clarity of practice guidelines, and consensus between societies to facilitate improved HDP prevention, recognition, and intervention.

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https://doi.org/10.1016/j.crwh.2023.e00569
Received 15 November 2023; Received in revised form 23 November 2023; Accepted 24 November 2023
Available online 27 November 2023

2. Case Presentation

A 37-year-old woman, G5P2, presented to the emergency department (ED) 18 days postpartum with the "worst headache of her life." The patient was induced and delivered vaginally at 38.3 due to gestational hypertension noted at her 37-week clinic visit. Prior to severe-range blood pressures at this visit, the patient's blood pressures ranged from 128 to 138 systolic and 60 to 88 diastolic (Table 1). Urinalysis was positive for proteinuria at two prenatal visits (Table 1).

The patient stated her headache was occipital in location and began the day before presenting to the ED. She also reported intermittent headaches, nausea, and vomiting for several days. Sitting up lessened the pain and lying supine exacerbated it. Pertinent positives included nausea and photophobia. The patient's medical history was significant for a pre-pregnancy BMI of 36, migraines, and gestational hypertension in a previous pregnancy which was continued to term. Medications at the time of ED presentation included aripiprazole 5 mg, sertraline 100 mg, omeprazole 20 mg, and a prenatal multivitamin. She had a 3-pack-year smoking history. Her family history was positive for hypertension and hypercholesteremia.

On exam, blood pressure was 230/125, pulse 69, respirations 20, BMI 39.7, and temperature 96.7° F. Physical exam was otherwise normal, including a nonfocal neurologic exam. Laboratory studies showed proteinuria (30 mg/dL). Other relevant laboratory results included platelet count of 367 K/mcL, AST 26 U/L, ALT 24 U/L, and INR 1.0.

Differential diagnosis included subarachnoid hemorrhage, reversible cerebral vasoconstriction syndrome, cerebral sinus venous thrombosis, migraine, meningitis, and postpartum preeclampsia.

Computed tomography (CT) angiography, CT venography, and non-contrast CT scan of the brain revealed a possible small subarachnoid hemorrhage (SAH). This was confirmed on magnetic resonance imaging (MRI), which showed SAH in the parietal region adjacent to the superior sagittal sinus without significant mass effect or shift. The patient underwent a cerebral angiogram, which showed no aneurysm. She was diagnosed with a subarachnoid hemorrhage secondary to a peri-mesencephalic bleed (i.e. venous hemorrhage).

Magnesium and two doses of labetalol were administered followed by a clevidipine drip. These improved the headache dramatically. The patient was admitted to the hospitalist service and continued on antihypertensives. She was discharged two days later with instructions to follow up with obstetrics and primary care as planned and interventional radiology in 4–6 weeks. She made a full recovery.

Table 1
Blood pressure and proteinuria data collected at routine OB visits throughout gestation and up to six week postpartum.

Estimated Gestational Age (Weeks)	Blood Pressure (mm Hg)	Urine Protein (mg/dL)	Notes
8W4D	138/82	Neg	Initial OB appt
12	126/78	Neg	
16	138/82	Neg	
20	130/80	Neg	
22	122/80	Not Collected	COVID-19 Dx
24	136/88	30	
28	112/68	Neg	
31	132/80	Neg	
34	130/80	Neg	
36	128/60	Neg	
37	182/120	Neg	BP Recheck: 160/92 then 140/84. Pt reports increased headaches.
37W6D	142/76	30	BP Recheck: 148/85 Induction for delivery. Only one blood pressure was available in records. At time of discharge blood pressure was noted to be downtrending.
38W3D	160/93	Not Collected	
6 W PP	138/100	Not Collected	Blood pressure noted to be managed by primary care.

3. Discussion

Guidelines for hypertension in pregnancy are rapidly evolving. As recently as 2020, ACOG guidelines indicated that intervention with antihypertensive therapy should be initiated only when a patient's blood pressure rises above 160/110 mmHg [5]. This threshold was addended in 2022 with the aforementioned practice advisory recommending a treatment threshold of 140/90 mmHg [3]. The Society for Maternal Fetal Medicine similarly recommends treatment with antihypertensive therapy for mild chronic hypertension in pregnancy to a goal blood pressure of <140/90 mmHg [6].

These updated recommendations are based on the Chronic Hypertension and Pregnancy (CHAP) trial, which found that the treatment of hypertension in pregnancy to a target blood pressure of <140/90 mmHg improved maternal and perinatal outcomes without harming fetal growth [7]. Patients with previously normal blood pressures meet the threshold after having blood pressures of systolic 140 mmHg or greater, or diastolic 90 mmHg or greater, on two occasions at least 4 h apart after 20 weeks of gestation [3].

Based on these critera, it can largely be agreed that the patient in this case should have been closely monitored and potentially started on treatment for blood pressures in the severe range at her 37-week clinic appointment (Table 1). However, during that same visit, the patient's blood pressure was documented as 140/84 upon recheck after two high readings. It is reasonable to think that this measurement, which was taken within the same appointment time, may have reassured treating clinicians, who at the time of this case were acting on 2020 practice guidelines to decide on treatment. It also highlights some of the challenge in defining hypertension and deciding on treatment in a population hovering close to or at threshold.

Indeed, the more interesting question revolves around this patient's blood pressure management earlier on in her pregnancy. While this patient did not meet ACOG treatment criteria for gestational hypertension until 37 weeks, she had multiple instances of blood pressures elevated >130 mmHg, approaching 140 mmHg even as she was establishing prenatal care. She also had risk factors of obesity and smoking in addition to a family history of cardiovascular disease.

3.1. Clinical Question

In 2017, ACC-AHA introduced updated guidelines that lowered the threshold for diagnosis of stage 1 hypertension from 140/90 mmHg to 130/80 mmHg in the general population [4]. This change was based on evidence that treating hypertension at lower levels decreases overall cardiovascular risk.

As this case demonstrates, there is now a population of pregnant patients considered to have stage 1 hypertension by ACC-AHA guidelines, but who do not meet the threshold for treatment by the ACOG practice advisory. Does this population have meaningfully different outcomes in maternal and fetal health during pregnancy and postpartum? To apply this question to the patient in this case specifically: could the adoption of ACC-AHA definitions of stage 1 hypertension have facilitated intervention much earlier in this patient's pregnancy, and would such an intervention have prevented not only her SAH, but also her development of severe hypertension in the first place?

The answers remain unclear. Concerns remain that lowering maternal blood pressure may compromise maternal-placental circulation or that antihypertensive therapies may confer adverse effects to fetus [8]. There is also a lack of historical data documenting measurable health benefits of stricter blood pressure control during the relatively short duration of pregnancy [8].

Nevertheless, emerging data in obstetrical populations does suggest a link between ACC-AHA-defined stage 1 hypertension and adverse pregnancy outcomes: in retrospective studies, patients with blood pressures ranging from 130 to 140 mmHg systolic and from 80 to 90 mmHg diastolic have been found to have a significantly increased risk of gestational diabetes [9], preeclampsia, and preterm birth compared to normotensive patients [9,10]. Furthermore, a recent meta-analysis found that patients with blood pressures below the 140/90 mmHg treatment threshold but above 120/80 mmHg had overall worse pregnancy outcomes than patients with blood pressures lower than 120/80 mmHg [11].

3.2. Call to Action

Close to one-third of mothers who died during hospital delivery between 2017 and 2019 had a documented HDP [12]. When it comes to maternal stroke and SAH, no prediction tools currently exist for identifying patients with increased risk. For pregnant patients, there is an urgent need for improved HDP prevention, early recognition, and prompt intervention. Critical steps towards addressing this include further study, increased clarity of practice guidelines and consensus between societies, and robust education and dissemination of information to community practices.

Contributors

Elana A. King-Nakaoka contributed to the conception of the case report, acquiring and interpreting data, undertaking the literature review, and writing and editing the manuscript.

Heather M. Beasley contributed to acquiring and interpreting data and writing and editing the manuscript.

Stephen R. Kessinger served as the attending emergency medicine physician for the patient and contributed to acquiring and interpreting data and editing the manuscript.

David C. Pfeiffer contributed to writing and editing the manuscript.

All authors approved the final submitted manuscript.

Funding

No funding from an external source supported the publication of this case report.

PATIENT CONSENT

The patient gave consent for this case report to be published.

Provenance and peer review

This article was not commissioned and was peer reviewed.

CONFLICT OF INTEREST STATEMENT

The authors declare that they have no conflict of interest regarding the publication of this case report.

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