An Atypical Case of Hypertension During Pregnancy

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INTRODUCTION

Hypertension during pregnancy is typically categorized into one of four categories:

- Chronic hypertension - diagnosis before 20 weeks
- Gestational hypertension - diagnosis after 20 weeks; no signs of preeclampsia
- Preeclampsia - involving proteinuria or significant end-organ dysfunction
- Chronic hypertension with superimposed preeclampsia - diagnosis before 20 weeks with preeclampic symptoms

The overall prevalence of hypertension during pregnancy is reported to be 8.6%[1].

INITIAL PRESENTATION

- 38F G1P0 with history of asthma, hypothyroidism and polycystic ovarian syndrome on levothyroxine and budesonide at home presenting to the ED at 14 weeks gestation.
- On presentation to the ED, her vitals were T 98.4 F, HR 64 bpm, BP 173/91, RR 18 and SpO2 100% on room air.
- Admission labs were notable for Hgb 10.0, MCV 74.7 and platelet count 254. Na 134, K 3.4, BUN 8, Cr 0.45, Alk-phos 55 and AST 18, ALT 12. Initial urinalysis was negative for protein, blood, nitrite and leukocyte esterase, with few bacteria appreciated.
- After four days the patient was discharged with nifedipine 30-60 mg, with instructions to return to medical attention if her pressure remained greater than 140/90 or she developed a headache, changes to vision or epigastric pain. Outpatient management dictated an increase to 60 mg BID.

HOSPITAL COURSE

- At 21 weeks gestation, the patient was asymptomatic, but was readmitted for uncontrolled hypertension (170/93), despite outpatient management.
- She subjectively denied headaches, visual disturbances and abdominal pain. Laboratory values were notable for platelets, creatinine, and transaminases within normal limits. She was noted to be hypokalemic to 2.8 mEq/L. Urinalysis demonstrated proteinuria with a random urine protein total of 77 mg/dL and an elevated UP/Cr ratio of 1.4; however, this finding was confounded by proteinuria secondary to nephrolithiasis at 18 weeks. Coagulation panel: aPTT 25.3, PT 12.7, INR 1.06
- Progression of hypertension mandated escalating doses of anti-hypertensive agents, ultimately requiring labetalol 800 mg TID, hydralazine 25 mg QID and nifedipine 60 mg BID to maintain a goal systolic between 120-150 and diastolic 80-100.
- Repletion of potassium required 40 meq orally daily (goal K > 3.3).

Third Trimester Plasma Renin and Aldosterone Values in Hypertensive Parturients

	Reference	Normal Pregnancy[3]	Chronic HTN w/o SPE[2]	Chronic HTN w/ SPE[2]	Our Patient
Plasma Renin Activity (ng/mL/hr)	0.2 - 5.4	10.1 ± 2.58	7.96 ± 1.01	5.97 ± 1.45	144
Urine Aldosterone (ng/dL)	< 23	81.1 ± 22.7	81.5 ± 14.41	59.6 ± 16.77	141

[2]Malha - values during pregnancy in women with chronic hypertension who do and do not develop (SPE) superimposed preeclampsia

WORKUP

A series of multidisciplinary discussions including nephrology, cardiology, obstetrics, maternal-fetal medicine and anesthesiology was held, yielding the following workup:

- Urine microscopy revealed no dysmorphic red blood cells or casts
- Serologic workup for glomerulonephritis was unremarkable
- Echocardiogram revealed no evidence of long-standing hypertension
- Renin/aldosterone panels revealed markedly increased values (Renin: 144 ng/mL/hr vs normal 0.2-5.4 ng/mL/hr, Aldosterone 141 ng/dL vs normal <23 ng/dL)
- Urine metanephrines ruled out pheochromocytoma
- Renal doppler and CT angiogram were negative for renal artery stenosis
- MRI of the abdomen revealed no adrenal masses
- Renal biopsy was negative for IgA nephropathy
- Genetic panel for hereditary kidney disease was inconclusive

LABOR & DELIVERY

- At 33 weeks and 5 days, the patient's blood pressure became resistant to ongoing therapy and remained elevated.
- Escalation of antihypertensive needs continued. At 33 weeks and 5 days, despite rescue doses of nifedipine 10 mg IR x3 and hydralazine 10/20 mg IV, the patient remained asymptomatically hypertensive to 194/91.
- Multidisciplinary discussion was held regarding course of action, and the decision was made to induce labor.
- An epidural catheter was placed for analgesia.
- Following 10 hours of labor, the patient opted for cesarean delivery. At this time, fetal tracing was category 1.
- Analgesia was provided via lidocaine and fentanyl through epidural. The patient's blood pressure was 165/90 following the final dose of analgesic.
- A right radial arterial line was placed for hemodynamic monitoring alongside standard monitors given risk factors

LABOR & DELIVERY (cont.)

- Cesarean section was performed, with an incision to delivery time of 4 minutes. The placenta was sent for pathologic investigation. At time of uterine incision, the patient's blood pressure was 145/85.
- Upon delivery, the patient's blood pressure fell to 95/60. Due to the rapid rate of decrease, a phenylephrine infusion was initiated.
- The patient was transferred to the surgical ICU for higher level of hemodynamic monitoring given pressor requirement in the setting of extended hospitalization.
- EBL 1164 mL, Urine 200 mL, IVF 3000 mL

POSTPARTUM PERIOD

- The patient remained in the SICU for 1 day. During this time, the patient was weaned from pressors, restarted on antihypertensives and stabilized to blood pressure 130/60.
- The patient was discharged on postoperative day 4 with a blood pressure of 155/97 on nifedipine 60 mg BID, labetalol 600 mg TID, hydralazine 25 mg QID, enalapril 2.5 mg, and 10 meq oral potassium daily.
- After discharge, the patient's antihypertensive regimen was steadily reduced (BP 116/80 at 4 week nephrology follow-up, 129/82 at 3 month nephrology follow-up). At three months postpartum, the patient was on enalapril 2.5mg and nifedipine 30 mg only.
- At 3 month follow-up, aldosterone was 14.1 ng/dL and renin was 1.673 ng/mL/hr.
- Placental studies revealed localized AVM malformations, but did not reveal a gross renin secreting tumor.

DISCUSSION

- Categorization of maternal hypertension has significant importance in terms of management. This case of severe hypertension in a previously normotensive patient developing before 20 weeks, resolving within 1 month of delivery and without clear proteinuria or other symptoms of preeclampsia does not neatly fit within any category.
- Although it is tempting to label this case as chronic hypertension, her acute presentation, abnormal RAAS labs, unexplained hypokalemia, lack of cardiac remodeling and abrupt resolution upon delivery suggest that this was not merely an unmasking of a chronic condition.
- Additionally, although our patient eventually developed urine protein levels suggestive of proteinuria, our ability to interpret her urine protein was confounded by her gross hematuria from nephrolithiasis. As she was otherwise asymptomatic, this made it difficult to immediately justify a diagnosis of preeclampsia.

[1]Kuklina EV. Hypertension in Pregnancy in the US-One Step Closer to Better Ascertainment and Management. JAMA Netw Open. 2020;3(10):e2019364. Published 2020 Oct 1. doi:10.1001/jamanetworkopen.2020.19364
[2]Malha L, Sison CP, Helseth G, Sealey JE, August P. Renin-Angiotensin-Aldosterone Profiles in Pregnant Women With Chronic Hypertension. Hypertension. 2018;72(2):417-424. doi:10.1161/HYPERTENSIONAHA.118.10854
[3]Elsheikh A, Creatsas G, Mastorakos G, Milingos S, Loutradis D, Michalas S. The renin-aldosterone system during normal and hypertensive pregnancy. Arch Gynecol Obstet. 2001;264(4):182-185. doi:10.1007/s004040000104