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ADRENAL HYPERTENSION
Breaking Boundaries in Adrenal Disorders
ANAH - AFES Joint Symposium 2025
14 – 16 Nov 2025 | Ariyana Convention Center, Da Nang city, Vietnam

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Meet-the-Professor Session

Adrenal Disorders in Pregnancy: Challenges in Diagnosis and Treatment

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Objectives

At the end of the session, you will be able to:

- **Recognize adrenal disorders in pregnancy**, specifically:
  - Adrenal Insufficiency (AI) and Adrenal Crisis
  - Cushing Syndrome (CS)
  - Primary Aldosteronism (PA)
  - Pheochromocytoma
- **Understand the pathophysiology complicating pregnancy** and **address the challenges** on
  - Diagnosis
  - Treatment

Meet-the-Professor Session

Case-Based Discussion

Case 1

- 29 years old
- G1P0
- Age of Gestation, 10 weeks
- Diagnosed and operated for Pituitary Cushing Disease at age 23 years
- On hydrocortisone replacement therapy
- **(+) nausea and vomiting**
- Physical examination:
- BP: 120/80, PR: 78
- Negative for orthostatic hypotension
- No cushingoid features

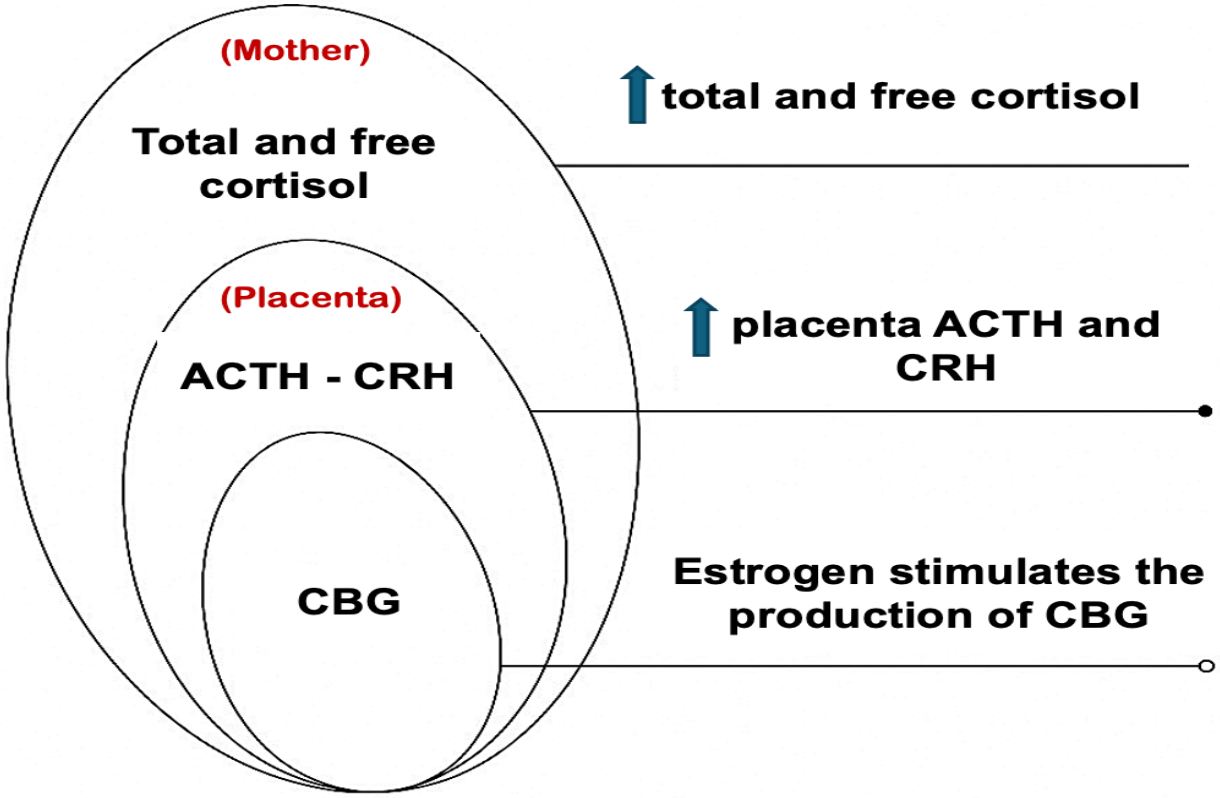
Choose the Best Answer: What to do?

A. The nausea and vomiting are signs of adrenal insufficiency (AI), so **outright increase the dose of hydrocortisone.**

B. There is negative orthostatic hypotension, **so observe further.**

C. **Do hormonal test** to fully assess the situation.

AI in Pregnancy: Changes in HPA Axis
Challenges



↑ total and free cortisol

The peak is at 26 weeks
Diurnal variation is maintained

↑ placenta ACTH and CRH

ACTH directly acts on the adrenal gland
CRH stimulates the adrenal gland directly and indirectly

Estrogen stimulates the production of CBG

↑ pituitary ACTH levels

↑ adrenal cortisol secretion

Ahmed, The Obstetrician & Gynaecologist 2021; https://doi.org/10.1111/tog.12761, Suri et al, JCEM, 2006:91:3866-72

AI in Pregnancy: *Diagnosis*

- Signs and Symptomatology
  - Weight loss
  - Prolonged vomiting
  - Hyperpigmentation in skin folds
  - Hypoglycemia, hyponatremia and hyperkalemia
- **First trimester**
  - **Hyperemesis**
  - **Infection**
- Third Trimester
  - Delivery, vaginal or CS
- Hormonal test
  - **Salivary free cortisol**
- Other
  - Adrenal antibodies, for autoimmune disease
  - ACTH stimulation test, caution on increased adrenal response in pregnancy

Calina et al. Int J Environ Res Public Health, 2019;16:781, Zelissen et al, Addison's disease. 1995;8:121-30, Suri et al, JCEM, 2006:91:3866-72

AI in Pregnancy: *Management*

- In current case, **if suspicious for AI, increase HC dose**
- **For replacement:**
  - HC is short-acting, does not cross placenta, 15-25 mg, BID-TID
  - Prednisolone OD, good alternative
  - Fludrocortisone, 0.05-0.1 mg/day
- **For AI**
  - HC at 40 mg has MR effect (0.1 of fludrocortisone)
  - Prednisolone, no MR effect, increase by 20-30%.

Bornstein et al, JCEM. 2016;101:364-89.

Adrenal Crisis in Pregnancy: *Diagnosis*

- Abdominal pain, vomiting, shock
- High index of clinical suspicion.
  - Severe vomiting, disrupted HC tabs absorption
- Adrenal hemorrhage and thrombosis, sepsis, labor, surgery

Manoharanet al.. J Obstet Gynaecol 2019;40:749–58, Keizer AL, et al. BMJ Case Rep2013;2013:bcr2012008062

Adrenal Crisis in Pregnancy: *Management*

- **PREVENTIVE:** From Replacement HC oral 5-20 mg/day to intrapartum, 50-100 mg IV q 8 hrs x 24 hrs

- **If in shock**
  - 2- 3 litres of 0.9% saline or 5% dextrose in 0.9%
  - HC IV at 100 mg every 6–8 hours, or as a continuous infusion of 200–300 mg in 24 hours
  - Monitor VS and electrolytes

- **Once stable**, parenteral tapered over 1–3 days; then switch to oral HC and fludrocortisone. Find and resolve cause.

Calina et al. Int J Environ Res Public Health, 2019;16:781, Langlois F, et all. Curr Opin Endocrinol Diabetes Obes. 2017;24:184–92.

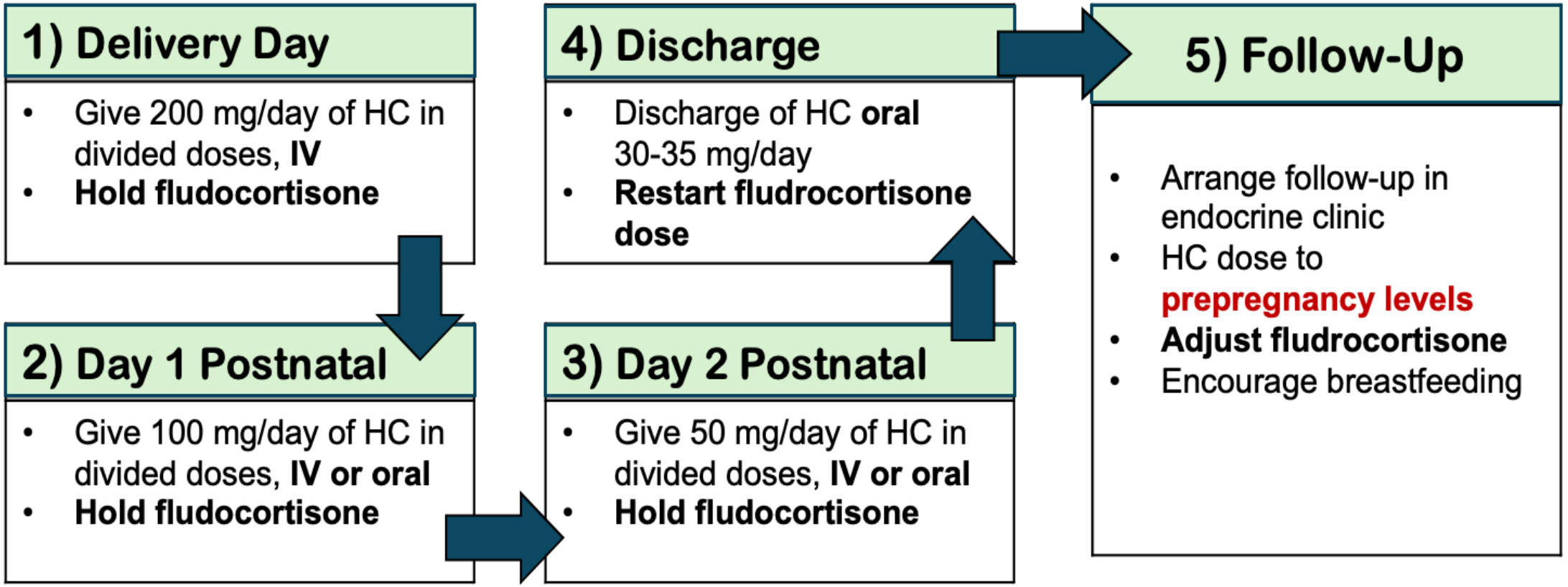
QUESTION: When will you shift to prepregnancy maintenance/replacement HC and MC doses?

A. Day 1 postpartum, if on a diet

B. Upon immediate discharge

C. Upon follow-up

Management of Glucocorticoids and Mineralocorticoids During Delivery and in the Puerperium



Ahmed, The Obstetrician & Gynaecologist 2021;https://doi.org/10.1111/tog.12761

Choose the Best Answer: What to do?

A. The nausea and vomiting are signs of adrenal insufficiency (AI), so **outright increase the dose of hydrocortisone.- Check for source of infection, quantify magnitude of vomiting**

B. There is negative orthostatic hypotension, **so observe further. Be vigilant, do not wait!---Fetal FGR, increased maternal morbidity and mortality**

C. **Do hormonal test** to fully assess the situation. **Cautious on interpretation**

Calina et al. Int J Environ Res Public Health, 2019;16:781, Schneiderman et al. BJOG 2017;124:1772-9

Question: When will you shift to prepregnancy maintenance/replacement HC and MC doses?

A. Day 1 postpartum, if on a diet.

B. Upon immediate discharge.

C. Upon follow-up .

Case 2

- 35 years old
- Obese since age 25, irregular menstruation
- (+) Infertility history
- Prediabetes, on metformin
- Now, G1P0, 12 weeks AOG
- BP 137/84, PR 82
- BMI, >95th centile for AOG
- Upper limb weakness
- Purplish striae
- Easy bruisability

QUESTION: What is/are the significant feature/s to suspect Cushing syndrome (CS)?

A. Gynecologic history

B. State of prediabetes

C. Weight history and current BMI

D. Physical examination findings

CS in Pregnancy: *Challenges*

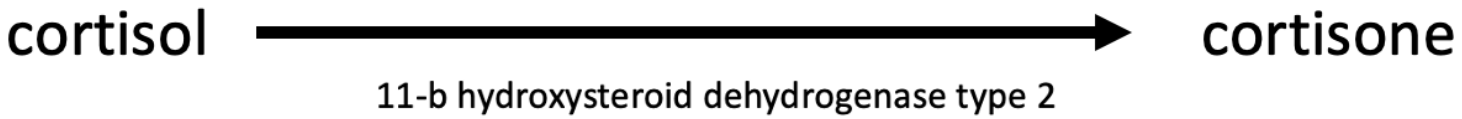
- Adrenal adenoma, 60% in pregnancy, **44% with CS, 263 cured CS**

	Active CS (%)	Cured CS (%)
GDM	36.9	2.3
GHTN	40.5	2.3
Preeclampsia	26.3	2.3
Fetal loss	23.6	8.5
Global fetal morbidity	33.3	4.9

Caimari et al. *Endocrine* **55**, 555–563 (2017). https://doi.org/10.1007/s12020-016-1117-0

Fetal Status and Hypercortisolemia

- The fetus is relatively shielded from maternal hypercortisolism:



- Active CS leads to fetal risks:
  - Miscarriage
  - Fetal growth retardation
  - Preterm delivery
  - Stillbirth
  - Neonatal AI

Anand et al, Eur J Endocrinol 2018;178:R45–53, Caimari et al. Endocrine 55, 555–563 (2017). https://doi.org/10.1007/s12020-016-1117-0, Brue et al, Eur J Endocrinol 2018;178:R259–66.

Predictors of Fetal Loss

- Etiology of hypercortisolism, **pregnancy-induced CS**, OR 4.7
  - Publication period, 1975-1994
- Treatment during gestation, OR medical 0.25, surgical, 0.34
- Period of diagnosis of CS, predictor of overall fetal morbidity/mortality.
- Both **medical treatment and surgery** during pregnancy appeared to be **protective i**n avoiding fetal loss.
- **Timely diagnosis is a big challenge.**
- **Early treatment of CS increases live birth rate.**

Caimari et al. Endocrine 55, 555–563 (2017). https://doi.org/10.1007/s12020-016-1117-0, Eschler et al, Endocrinol Metab Clin North Am 2015;44:381–97.

Pregnancy-Associated CS

- Definition
  - CD onset during gestation or within 12 months after delivery or abortion

- Pathophysiology
  - **Increased estrogen (100-fold)-**---stimulates pituitary angiogenesis, mitogenesis, and adenohypophyseal hormone secretion.

  - **Placental CRH-**---activates HPA axis, desensitizes negative feedback----surge of plasma ACTH and cortisol----**pituitary tumorigeneses.**

Tang et al, Front Endocrinol, 11:305. doi: 10.3389/fendo.2020.00305 Brue et al, Eur J Endocrinol. (2018) 178:R259–R66. doi: 10.1530/EJE-17-1058, Lloyd et al, Pathol Res Pract. (1991) 187:584–6. doi: 10.1016/S03 44-0338(11)80150-0, Manoranjan et al, Anticancer Res. (2010) 30:2897–904. Available online at: http://ar.iiarjournals.org/content/30/7/2897.long, Shimon and Melmed, JCEM. (1997) 82:1675–81. doi: 10.1210/jcem.82.6.3987

Features of Pregnancy-Associated CD

- Incidence (N=70): 27.1%
- Timing of diagnosis: 2.7 ± 3.4 yrs postpartum
- **Remission rate: 89.5%**
- Common: HTN, severe DM, LBW babies
- Rate of abortion and pre-term birth: 26.3, 28.6%, respectively.

Tang et al, Front Endocrinol, 11:305. doi: 10.3389/fendo.2020.00305

Clues to Pregnancy-Associated CS

- High degree of clinical suspicion for CD in the peripartum period.

- Patients with symptoms suspicious for CD throughout pregnancy and after childbirth:
  - Early-onset hypertension
  - Severe hyperglycemia
  - Persistent weight gain

- Other reported maternal complications
  - Wound infection
  - Heart failure
  - Psychiatric disorders
  - Maternal death

- Should be carefully diagnosed and closely monitored.

Tang et al, Front Endocrinol, 11:305. doi: 10.3389/fendo.2020.00305, Ahmed, The Obstetrician & Gynaecologist 2021; https://doi.org/10.1111/tog.12761, BMJ Case Rep 2013;2013:bcr2012008062, Caimari et al. Endocrine 55, 555–563 (2017).

QUESTION: How will you **screen/diagnose** CS in pregnancy?

A. Clinical features, 1 mg dex/8 mg dex suppression

B. Diurnal plasma cortisol, UFC/8 mg dex suppression

C. Late night salivary cortisol, UFC/8 mg dex suppression

CS in Pregnancy: *Clinical Diagnosis*

- Overlapping pregnancy-associated features with CS vs. non-CS associated:
  - Weight gain, hypertension, striae, muscle, fatigue, and glucose intolerance.

- CS-specific features:
  - Proximal myopathy
  - Easy bruising
  - Early onset hypertension in pregnancy
  - Presence of red or purple striae (instead of pale striae of normal pregnancy)
  - Hirsutism, osteopenia/osteoporosis-induced fractures

Frise & Williamson, Clin Med (Lond), 2013;13:176–81, Lindsay et al. J Clin Endocrinol Metab 2005;90:3077–83.

CS in Pregnancy: *Hormonal Diagnosis*

- **Screening**: Midnight plasma cortisol level, diurnal variation is maintained though higher than nonpregnant.

- **Diagnosis:** Late-night salivary cortisol and UFC, >3x upper limit of normal values.
  - **Thresholds,** 1st, 2nd, 3rd trimester: <6.9, <7.2, and <9.1 nmol/L, respectively, depends on assay.

- One (1 mg) dex suppression- false positive owing to pregnancy-induced hypercortisolism, placental ACTH is not suppressed by dexamethasone.

- **Eight (8) mg dex suppression- Cushing disease if >4.4 pmol/L.**

Frise & Williamson, Clin Med (Lond), 2013;13:176–81, Lindsay et al. J Clin Endocrinol Metab 2005;90:3077–83, Eschler et al, Endocrinol Metab Clin North Am 2015;44:381–97

QUESTION: How will you **manage** CS in pregnancy?

A. None, until postpartum

B. Medical

C. Surgical

CS in Pregnancy: *Surgical Management*

- Second trimester
  - Laparoscopic unilateral adrenalectomy and trans-sphenoidal surgery
  - Bilateral adrenalectomy for refractory cases

- Manage AI with adequate HC replacement therapy
  - Important in the intrapartum and immediate postpartum period

Lindsay et al. J Clin Endocrinol Metab 2005;90:3077–83, Caimari et al. Endocrine 55, 555–563 (2017), Eschler et al, Endocrinol Metab Clin North Am 2015;44:381–97, Brue et al, Eur J Endocrinol 2018;178: R259–66.

CS in Pregnancy: *Medical Management*

- **Medical treatment**
  - Second-line option
  - If not fit or suitable for surgery.
- **Metyrapone**, steroidogenesis inhibitor
  - Increases the risk of hypertension through the accumulation of MC precursors
- **Ketoconazole and Mitotane**
  - Teratogenic
- **Cabergoline**
  - In pituitary cushing

Calina et al, Int J Environ Res Public Health 2019;16:781, Manoharan et al, J Obstet Gynaecol 2019;40:749–58, Lindsay et al. J Clin Endocrinol Metab 2005;90:3077–83, J Appl Toxicol 2012;32:98–107.

Optimal Pregnancy Outcome in CS

- **Good control** of gestational hypertension, DM
- Prompt assessment and management of **preterm labour**.
- **Vaginal delivery** is encouraged, because of poor wound healing.

QUESTION: What is/are the significant feature/s to suspect Cushing syndrome (CS)?

A. Gynecologic history

B. State of prediabetes

C. Weight history and current BMI

D. Physical examination findings

QUESTION: How will you **screen/diagnose** CS in pregnancy?

A. Clinical features, 1 mg dex/8 mg dex suppression

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C. Late night salivary cortisol, UFC/8 mg dex suppression

QUESTION: How will you **manage** CS in pregnancy?

A. None, until postpartum

B. Medical

C. Surgical

Case 3 (a 22-yr old Case Report)

- 38 years old
- One (1) year history of HTN
- G2P0, miscarriage on first
- HTN on 2nd pregnancy, metoprolol 50 mg/day
- 12-13 wks AOG, endo referral
- Severe headache, muscle weakness
- BP: 160/100
- Serum K, 1.9 mmol/L
- **Admitted**:
  - **Plasma aldo, 5,420 pmol/L** (NR 145-445)
  - **PRA: 13.7 ng/mL/hr** (NR 0.15-2.33)
- Normal creatinine
- **Diagnosis: Primary Aldosteronism (PA)**

Amoranto-Santos, Mercado-Asis, JAFES, 2003;21:62-62

Case 3: *Clinical Course (1)*

- **Treatment**
  - IV Hydralazine, oral nifedipine, metoprolol
  - K supplement
  - **Spironolactone tab, 300 mg/day**
- **4th Hospital Day**
  - Serum K - 4.1 mmol/L
  - BP: 110-130/60-80
- **AT 38 wks AOG**, cesarean section
  - Live, normal baby girl
  - Apgar 8,9,9
- **6 wks postpartum**
  - Plasma aldo postpartum remained high
  - PRA, low normal
  - CT scan, negative

Amoranto-Santos, Mercado-Asis, JAFES, 2003;21:62-62

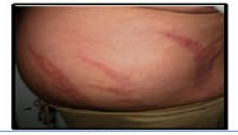
Case 3: *Clinical Course (2)*

- **18 mos postpartum**
  - **BAVS**- localized lesion on the **right**
  - Unilateral adrenalectomy, right
  - 12 hrs postop, normal BP w/ no anti-HTN meds
  - Patho confirmed **adrenal adenoma**
- **19 mos postpartum**
  - Normal BP and serum potassium with **no meds**

Amoranto-Santos, Mercado-Asis, JAFES, 2003;21:62-62

Pathophysiology of Hypertension

Cushing



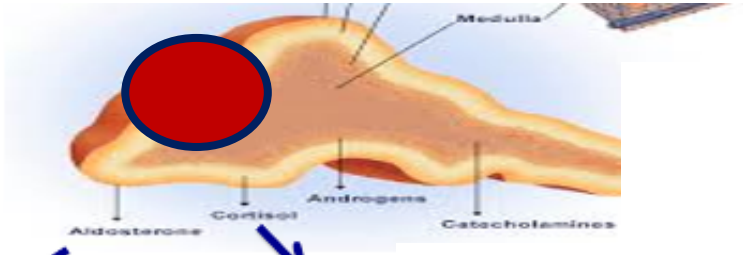
Striae

Easy bruisability

Aldosteronism

Increased volume

Approach to BP Control



Primary Aldosteronism

Cushing syndrome

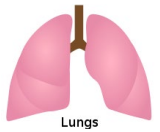
Hypercortisolemia

Decreased Renin

Angiotensinogen

Angiotensin I

ACE

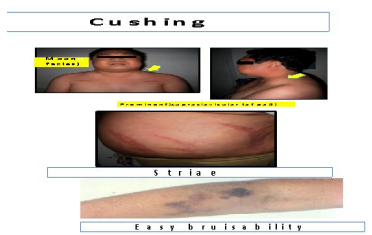


Angiotensin II

Lenders et al. JCEM. 2014;99:1915, Nieman et al. JCEM. 2015;100:2807, Mulatero et al. JCEM. 2004;89:1045-50, Mercado-Asis & Castillo. Hypertension Journal. 2019; 5:87, Brown et al. Ann Intern Med. 2020;173:10

Approach to BP Control

Anti-adrenal
Ketoconazole



Tube

Blood

Increased volume

suppressed Renin-Angiotensin System (NO SARTANS, NO ACEs)

Give calcium channel blocker

Spironolactone:

- **To block effect of aldo/cortisol to mineralocorticoid receptor**
- **Diuretic**

Approach to Hypokalemia

K= Potassium

- Spironolactone
- Potassium supplement

PA in Pregnancy

- PA prevalence in pregnancy, 0.6-0.8%, 50 cases in literature.

- Increase in renin activity
  - 4-fold in the first 8 weeks of gestation
  - 7-fold in the third trimester

- Increase in plasma aldosterone
  - 3 to 8-fold in 1st and 2nd trimester
  - Plateau, in 3rd trimester

- **Progesterone antagonizes** the **RAAS** and reduces urinary potassium excretion
  - Pregnant women are usually **normotensive and normokalemic.**

Corsello et al, Endocrinol Metab Clin North Am 2019;48:829–42, Malha and August, Curr Hypertens. Rep 2015;17:53, Eschler, Endocrinol Metab Clin North Am 2015;44:381–97., Landau and Amar. Ann Endocrinol, (Paris) 2016;77:148–60., irani and Xia, Placenta 2008;29:763–71.

Question: When will you suspect PA in pregnancy?

A. All hypertensive pregnant patients

B. Hypertensive patients with hypokalemia and resistant hypertension

PA in Pregnancy: *Report of 40 cases*

- **Clinical features**:
  - 81%, HTN for the first time in pregnancy
  - 19%, previous HTN, not screened for PA
  - **68%, hypoK**
  - 19% HTN controlled, 32% resistant HTN

- **Clinical course:**
  - 23% developed preeclampsia,
  - **61% induced vaginall delivery**
  - **44% CS**
  - 9.4% stillbirth

- **Treatment**:
  - Calcium channel blockers, beta-blockers, and alpha methyldopa.
  - **19.4%, spironolactone, highest 500 mg/day**
  - 16.1%, adrenalectomy

Landau and Amar. Ann Endocrinol, (Paris) 2016;77:148–60., irani and Xia, Placenta 2008;29:763–71.

PA in Pregnancy: *Diagnosis*

- Before 20 weeks AOG, **resistant HTN**, **hypokalemia**
- High ARR
- Saline infusion, not recommended
- **MRI, BAVS, after delivery**

Funder et al, J Clin Endocrinol Metab. 2016;101(5):1889–1916, Kamoun et al, Am J Med Sci 2014;347:64–73,, Rossi et al, Hypertension 2014;63:151–60, Riester and Reincke. Eur J Endocrinol 2015;172:R23–30.

Question: How will you treat PA in pregnancy?

A. Medical

B. Surgical

C. Both

PA in Pregnancy: *Treatment*

- Control of BP, alpha-methyldopa, labetolol, CCB
- **Spironolactone, feminization (?), FGR**
- Eplerenone, non teratogenic
- Thiazides, amiloride, 2nd to 3rd trimester
- Unilateral adrenalectomy, second trimester, high fetal morbidity and mortality

Hecker et al, Acta Endocrinol (Copenh)1980;95:540–5, Liszewski and Boull, J Am Acad Dermatol 2019;80:1147–8, U.S. Department of Health and Human Services website. https://www.fda.gov/downloads/ drugs/guidances/ucm078932.pdf., Cabassi et al, Hypertension 2012;59:e18–9, Morton and Laurie, Hypertens 2017;7:54–5,

Spironolactone: *Issue on fetal feminization*

Cases of human males exposed to spironolactone in utero

Case no.	Maximum daily dose, mg	Results
1	400	Normal genitals at birth and puberty
2	400	Normal genitals at birth
3	200	Normal genitals at birth
4	50	Normal genitals at birth
5	25	Normal genitals at birth

US FDA approved, 25-200mg/day

Liszewski and Boull, J Am Acad Dermatol 2019;80:1147–8, U.S. Department of Health and Human Services website. https://www.fda.gov/downloads/drugs/guidances/ucm078932.pdf.

Guide to Diagnose and Manage PA in Pregnancy

HTN + Hypokalemia

High Aldo, low PRA, high ARR

CBB, betablocker,
K supplement, spironolactone

Early delivery, CS

Postpartum, localize PA and remove

LBMA, 2025

Case 4

- 27 years old
- HTN at age 23, nonobese, nonsmoker, non-cushingoid
- 14 weeks AOG
- Presenting as “preeclampsia”
- BP: 180/110, no edema
- FH, kidney stones

- Work-up:
  - 2dECHO, septal dyskinesia
  - Serum aldo, PRA, normal
  - Urine, (-) protein
  - 24-hr urine metanephrines, 3x normal high

Red Flags

- 27 years old
- HTN at age **23, nonobese, nonsmoker,** non-cushingoid
- **14 weeks AOG**
- Presenting as **"preeclampsia"**
- **BP: 180/110, no edema**
- **FH, kidney stones**

- Work-up:
  - 2dECHO, septal **dyskinesia**
  - Serum aldo, PRA, normal
  - **Urine, (-) protein**

- **Diagnosis**
  - **24-hr urine metanephrines, 3x normal high**

Pheochromocytoma/Paraganglioma (PPGL) in Pregnancy

- Prevalence in pregnancy of PPGL 1 in 15 000 to 1 in 54 000 pregnancies
- The clinical presentation of PPGL during pregnancy is similar to that in nonpregnant women- HTN, headache, palpitations
- Most patients become **symptomatic with increasing gestation**, most likely because **of mechanical factors from the growing uterus and fetal movements**.

Lenders et al, Endocrinol Metab Clin North Am 2019;48:605–17, Bravo, Kidney Int 1991;40:544–56, Archer et al, Int J Obstet Anesth 2008;17:247–54.

Dramatic clinical presentations, laboratory and imaging findings and clinical outcome of patients with unsuspecting pheochromocytoma

Signs and symptoms	Headache, agitations, diaphoresis, nausea, vomiting Acute coronary syndrome Severe congestive heart failure Arrythmia
Laboratory	Elevated creatine kinase Normal to elevated troponin
Clinical outcome	Resolution of signs and symptoms after adrenalectomy Normalized LV function and ejection fraction Persistent systolic and diastolic impairment Death

Mercado-Asis, Siao, Amba, 2017, J Med UST, DOI 10.35460/2546-1621.2017-0050

Paraganglioma in Pregnancy: A Review

- Estimated prevalence, 0.0007%
- N=8 pregnancies

Mortality (%)	Paraganglioma	Pheochromocytoma
Maternal	3.6	9.8
Fetal	12	16

Wing et al. J Clin Endocrinol Metab 2015;100:3202–9.

PPGL on Pregnancy and Fetal Outcomes

- **High maternal catecholamines-**-- uteroplacental vasoconstriction---FGR, fetal hypoxia, fetal death

- **Paroxysmal maternal BP elevation/hypotension-**-- placental abruption, intrauterine hypoxia---adverse fetal outcomes

- **Decline in fetal mortality**
  - 1960s, 50%
  - 1990s, 25%
  - Last decade, 9.5%
  - If PPGL diagnosed **antenatally**, **7%**, **missed diagnosis, 17%**

Archer et al, Int J Obstet Anesth 2008;17:247–54, Wing et al. J Clin Endocrinol Metab 2015;100:3202–9

Question: How will you treat PPGL in pregnancy?

A. Medical

B. Surgical

C. Both

Catecholamines on the Various Receptors (adrenergic, dopaminergic) and Their Common Manifestations in Pheochromocytoma (1)

Target organ system	Receptor types	Sympathetic action	Common manifestations in pheochromocytoma
Skin and mucosa	α1, α2	Vasoconstriction, localized secretion of sweat glands	Pallor, diaphoresis
Peripheral vascular	α1, α2, β2	Vasoconstriction α1, α2 Vasodilation β2	Hypertension
Orthostatic hypotension			
Brain	α1, D1	Vasoconstriction	Headache
Heart	β1, β2, D1	Increase in heart rate, contractility, automaticity, conduction velocity	Palpitations, tachycardia, angina

Mercado-Asis, Siao, Amba, 2017, J Med UST, DOI 10.35460/2546-1621.2017-0050

Catecholamines on the Various Receptors (adrenergic, dopaminergic) and Their Common Manifestations in Pheochromocytoma (2)

Target organ system	Receptor types	Sympathetic action	Common manifestations in pheochromocytoma
Lungs	α1, β2	Pulmonary arteriole vasoconstriction, tracheal and bronchial muscle relaxation	Dyspnea
Gastrointestinal	α1, α2, β2	Decrease gastrointestinal motility and secretion, constricts sphincters, increases liver glycogenolysis and gluconeogenesis, increases pancreatic release of insulin and glucagon	Nausea, abdominal pain, constipatic hyperglycemia
Kidneys	β1	Increase renin secretion	Hypertension
Adipocytes	β1, β3	Increase lipolysis	Weight loss

Mercado-Asis, Siao, Amba, 2017, J Med UST, DOI 10.35460/2546-1621.2017-0050

PPGL in Pregnancy: *Diagnosis and Management (1)*

- Similar in nonpregnant patients.

- **For high BP**, phenoxybenzamine, doxazocin, betablockers, CCB
  - Check neonatal hypotension, respiratory depression

- **Biochemical tests**, plasma and 24 hr urine metanephrines, 100% sensitivity, 98% specificity
    - Stop phenoxybenzamine, clonidine, reserpine, levodopa, antidepressants

Lenders et al, J Clin Endocrinol Metab, 2014;99:1915–42. Lenders et al, Endocrinol Metab Clin North Am 2019;48:605–17, Iijima, Gynecol Endocrinol 2019;35:280–6

PPGL in Pregnancy: *Diagnosis and Management (2)*

- **MRI**, imaging during pregnancy, MIBG/PET in postpartum
- **Adrenalectomy**, before 24 weeks AOG or postpartum
- **Mode of delivery,** well-timed/planned CS is preferred

Lenders et al, J Clin Endocrinol Metab, 2014;99:1915–42. Lenders et al, Endocrinol Metab Clin North Am 2019;48:605–17, Iijima, Gynecol Endocrinol 2019;35:280–6

Overall Summary and Insight (1)

- Reports of **adrenal disorders (AI, CS, PA, PPGL) in pregnancy** is limited, therefore, their rarity could be relative.

- We have to **increase awareness** of these entities, for favorable outcomes for both the mother and the baby.

- **Early diagnosis** during the antepartum period with **optimal medical management (**well-adjusted HC replacement in AI, control of BP and other comorbidities in CS, PA and PPGL) **decrease maternal and fetal morbidity and mortality.**

Overall Summary and Insight (2)

- **Close fetal monitoring** must be done to alleviate fetal adverse effects of drugs.

- **Well-timed and planned cesarean section mode** of delivery for PA and PPGL, and vaginal for CS lead to favorable maternal and fetal outcomes.

- For more **definitive approaches for diagnosis and treatment** (CS, PA, and PPGL), these could be done in the **postpartum period.**

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