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Diabetes and Hypertension Project ECHO* Clinic

*ECHO: Extension of Community Healthcare Outcomes

February 25, 2021

If you haven't, visit vcuhealth.org/echodmhtn for instructions on creating your account.

Be thinking of something you're looking forward to this spring season to share during introductions!



Helpful Reminders





*Rename your Zoom screen with your name and organization

Helpful Reminders





- You are all on mute.
 Please unmute to talk
 - If joining by telephone audio only, press *6 to mute and unmute



MVCU



- Please type your full name and organization in the chat box
- Use the chat function to speak with our team or ask questions

VCU Health Diabetes & Hypertension ECHO Clinics



- Bimonthly, 1.5-hour tele-ECHO clinics on 2nd and 4th Thursdays
- Every tele-ECHO clinic includes a 30-minute didactic presentation followed by case discussions
- Didactic presentations are developed and delivered by interprofessional experts
- Website: <u>www.vcuhealth.org/echodmhtn</u>
 - Directions for creating an account and claiming CE can be found here also
 - You have up to six days after our session to claim CE by texting **19149-18817** to **804-625-4041**

Hub and Participant Introductions



VCU Team	
Principal Investigator	Dave Dixon, PharmD
Administrative Medical Director ECHO Hub	Vimal Mishra, MD, MMCi
Clinical Experts	Niraj Kothari, MD Trang Le, MD
Project Coordinator/IT Support	Madeleine Wagner, BA
Program Manager	Bhakti Dave, MPH

- Use chat function for introduction
 - Name
 - Organization

Reminder: Mute and unmute screen to talk or press *6 for phone audio

Share your name, organization, and something you're looking forward to this spring season!

ECHO is all teach, all learn



Interactive



Co-management of cases



Peer-to-peer learning



Collaborative problem solving



Housekeeping items

- Please feel free to eat your lunch or step away briefly if needed
- We are recording and can share sessions upon request
 - Each session's slides are available on www.vcuhealth.org/echodmhtn
 - We encourage you to keep your camera on, but if you are uncomfortable being recorded, feel free to turn it off
- Please **do not share any protected health information** in your discussion or the chat
- Project ECHO operates on the "All Teach, All Learn" model
 - Feel free to ask questions in the chat or unmute to ask questions at designated times
 - We're all here to learn from each other and value each person's input and expertise!

What to Expect







Let's get started! Didactic Presentation





Disclosures

Trang Le, MD has no financial conflicts of interest to disclose. Niraj Kothari, MD has no financial conflicts of interest to disclose. There is no commercial or in-kind support for this activity.



Secondary Hypertension





Learning Objectives

- Understand when screening for secondary etiologies of HTN is appropriate
- Recall the most common etiologies of secondary HTN
- Describe typical approaches towards evaluation and management of secondary HTN

Most patients do not require screening for secondary HTN



- 90% of HTN is essential (no identifiable secondary cause)
- Evaluation for secondary HTN carries the risk of incidental findings (i.e. adrenal incidentalomas) requiring potential interventions which may increase overall morbidity/mortality



Secondary HTN

• Whom to screen:

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- Age of onset <30 without obesity or family hx HTN
- Severe (>180/110mmHg) or resistant HTN
 - Resistant HTN: BP above goal despite 3 different classes of antihypertensive agent, <u>including a diuretic</u>, or at goal on four classes <u>including a diuretic</u>
 - Rule out pseudohypertension
- Abruptly worsened BP in previously controlled pt
- Worrisome clinical features



"Your blood pressure is up. Quit taking campaign promises with a grain of salt."



Etiologies of secondary HTN

- Renal parenchymal disease
- Hyperaldosteronism (not all have hypokalemia)
- Renovascular HTN
 - >90% atherosclerosis
 - Fibromuscular dysplasia (especially young women)
- Pheochromocytoma
- Thyroid dysfunction
- Obesity
- OSA
- Medications

Top 3 causes

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Kidney disease and HTN



- Acute glomerular disease tends to cause sodium retention
 - RAAS suppression
- HTN is very common, present in ~85% of CKD patients
 - Worsens as GFR falls
 - Sodium retention (even in nonedematous patients)
 - Increased RAAS activity
 - Possible increased sympathetic activity
 - Secondary hyperparathyroidism
- Management: standard CKD/nephrology care



Primary hyperaldosteronism

- Underdiagnosed
- Classic presentation includes hypertension and hypokalemia, however many patients are normokalemic
- Check aldosterone-renin ratio (ARR)
 - Suspicious if renin is suppressed and aldosterone is over 10ng/dL with ARR > 20
 - Confounding meds:
 - Mineralocorticoid receptor antagonists: cause renin elevation, falsely lowering ARR
 - RAAS blockade: can also cause renin elevation
 - Suppressed renin in a patient on RAAS blockade is suggestive of primary hyperaldosteronism
- Confirmation: oral sodium loading, saline infusion testing
 - Oral test: urine aldosterone excretion >12mcg/24 hrs c/w hyperaldosteronism
 - Saline infusion: plasma aldo levels > 10ng/dL c/w hyperaldosteronism
- Treatment:

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- Surgery for unilateral adenoma or hyperplasia
- Mineralocorticoid receptor antagonists (spironolactone, eplerenone)



Hypertension, hypokalemia, and metabolic alkalosis: A triad driven by aldosterone, cortisol, or other



Evaluation of mineralocorticoid excess in patients with the triad of hypertension, hypokalemia, and metabolic alkalosis.HTN, Hypertension; GRA, Glucocorticoid-remediable aldosteronism; ACTH, Adrenocorticotropin hormone.¹Glycyrrhizic acid and its hydrolytic product glycyrrhetinic acid from licorice are potent competitive inhibitors of 11 β-hydroxysteroid dehydrogenase, the enzyme required to metabolize cortisol to its inactive form, cortisone.²Apparent mineralocorticoid excess: mutation of 11 β-hydroxysteroid dehydrogenase enzyme

A 46-year-old woman with a history of poorly controlled hypertension is sent for evaluation of severe hypertension resistant to losartan 100 mg daily, chlorthalidone 25 mg daily, amlodipine 10 twice daily, and metoprolol 100 mg twice daily. She also takes oral potassium chloride 40 mEq twice daily. She denies use of nonsteroidal anti-inflammatory drugs (NSAIDs), decongestants, or herbal products. She states that she follows a low-salt diet. BP in both arms ranges from 185 to 200/95 to 105 mm Hg, with a pulse of 55 beats/min. Examination is unremarkable without abdominal bruit. Labs reveal K+, 3.6mEq/L; HCO3, 27 mEq/L; BUN, 16 mg/dL; serum Cr, 1.1 mg/dL. Complete blood count is normal. Urinalysis reveals trace protein, and the urine sediment is bland. Plasma renin activity is 0.9, and aldosterone is 28 ng/mL (aldosterone renin ratio ~ 30). A salt-loaded 24-hour urine contains 19 μ g/day of aldosterone. Both tests were obtained while on losartan and chlorthalidone.

Virginia Commonwealth University

Which is the BEST imaging test to make the diagnosis in this patient?

- A. MRI of the adrenal glands
- B. PET/CT of the kidneys
- C. CT of the adrenal glands
- D. CT angiogram of the renal arteries
- E. MIBG scan

ASN BRCU 2017

Annals of Internal Medicine

Virginia Commonwealth University

REVIEW

Comparative Effectiveness of Management Strategies for Renal Artery Stenosis An Updated Systematic Review

Gowri Raman, MD, MS; Gaelen P. Adam, MLIS; Christopher W. Halladay, BA, ScM; Valerie N. Langberg, ScM; Ijeoma A. Azodo, MD, ChM; and Ethan M. Balk, MD, MPH

- Meta-analysis of studies comparing angioplasty + stent placement vs. medical therapy alone in atherosclerotic RAS
- No significant benefit for mortality, ESKD, major CV events, or BP control
- Limitations: the RCTs included usually had asymptomatic pts with moderate stenosis, moderately controlled HTN, stable renal function. Non-randomized studies had pts with uncontrolled BP



Renovascular HTN

- Not everyone needs imaging
- Indications:
 - No evidence of CKD, hyperaldosteronism, pheochromocytoma
 - Intervention planned if stenosis found
 - Short duration of HTN (weeks to months)
 - Failure of BP control or intolerance of medical therapy (including Cr rise after RAAS blockade)
 - Progressive renal insufficiency from suspected renovascular disease
 - Suspected fibromuscular dysplasia
 - Recurrent flash pulmonary edema or refractory HF
 - Don't screen patients who respond well to medical therapy
- Methods
 - Renal artery dopplers
 - MR/CT angio
 - Gold standard: renal arteriography (only if intervention planned)
 - Renin/aldosterone generally unhelpful



Fibromuscular dysplasia

- Lesions often more distal, more than 2cm from aorta
- Dopplers not great for diagnosis as they most effectively detect proximal renal artery lesions
- Commonly seen in 15-50yo females with early onset HTN
- Percutaneous renal angioplasty (can be repeated)
- Surgical revascularization if aneurysmal dilations > 1.5cm



Healthjade.com

A 70-year-old woman is admitted with pulmonary edema and elevated serum creatinine (1.9 mg/dL). BP on admission is 210/95 mm Hg. Her symptoms of dyspnea improve after administration of furosemide, nitrates, and carvedilol. BP the next morning is 150/90 mm Hg. Previous medications include lisinopril, felodipine, hydrochlorothiazide, atorvastatin, aspirin, and ezetimibe.

On the day after admission, serum creatinine rises to 2.4 mg/dL. Transthoracic echocardiogram demonstrates an ejection fraction of 55% and moderate aortic stenosis. Renal artery duplex ultrasound demonstrates an atrophic left kidney (length 6 cm with no identifiable blood flow) and a normal sized right kidney (10.8 cm) and elevated renal artery velocities.

Which is the MOST accurate regarding long-term outcomes for this patient?

- A. Prospective, randomized trials indicate no benefit of renal vascularization
- B. Hospitalization for recurrent symptoms is unlikely if the ACEi is withheld
- C. Mortality is increased in patients managed medically compared with renal artery stenting
- D. Placement of an AVF will decrease GFR further
- E. Laparoscopic nephrectomy of the left kidney will reduce the risk of recurrence







Pheochromocytoma

- Catecholamine-secreting tumor -> excess norepi/epi
- Paroxysmal HTN
- Classic triad: flushing/pounding headache/sweating
- Serum metanephrines are sensitive but not as specific—confirm with urinary metanephrines, followed by CT/MRI of the abdomen and pelvis
 - If CT/MRI negative, can perform iodine-123 MIBG scan
 - PET for identification of metastatic disease
- Surgical removal
 - α -blockade with phenoxybenzamine (goal BP 130/80 seated and SBP > 90 while standing).
 - DO NOT USE β -BLOCKERS BEFORE ADEQUATE α -BLOCKADE!
 - Metastases can be found well after diagnosis
 - Follow with annual plasma/urine metanephrines
- Association with MEN 2A and 2B, neurofibromatosis type 1, von Hippel-Lindau
 - Careful history essential



Medications causing secondary HTN

Black licorice (European)	Antiretrovirals
EtOH	VEGF antagonists i.e. bevacizumab
NSAIDs	Glucocorticoids
Sympathomimetics, decongestants, amphetamines	OCPs
SSRIs/SNRIs	Caffeine
ESAs i.e. erythropoietin	Cocaine
CNIs (cyclosporine > tacrolimus)	



A 57-year-old woman is evaluated during a preoperative physical examination for a total left knee replacement. Medical history is significant for osteoarthritis; her only medication is over-the-counter ibuprofen, which she takes multiple times daily for pain relief.

On physical examination, blood pressure is 152/90 mm Hg, and pulse rate is 64/min. BMI is 34. Severe osteoarthritic changes are noted in the left knee. Trace pitting edema in the ankles is noted. The remainder of the examination is unremarkable.

Laboratory studies show a serum creatinine level of 1.2 mg/dL (106.1 µmol/L) and a serum potassium level of 5.1 mEq/L (5.1 mmol/L); urine dipstick demonstrates no blood or protein.

Which of the following is the most appropriate next step in the management of the patient's blood pressure?

- A. Begin a low-dose ACEi
- B. Begin low-dose HCTZ
- C. Discontinue ibuprofen
- D. Obtain a plasma aldosterone/renin ratio



OK, so what do I actually order in the clinic

- Based on history and physical exam, can order:
 - TSH
 - Serum/urine metanephrines
 - Serum aldosterone/renin (even if normokalemic)
 - Renal dopplers or MR/CT angio (if intervention would be performed for renovascular HTN)
 - CT angiography (if a ortic coarctation suspected based on exam)
 - 24-hr urine free cortisol or late evening salivary cortisol (if Cushing syndrome suspected)
 - Overnight dexamethasone suppression test can also be used, unnecessary if pt on chronic glucocorticoids
 - Polysomnography (if suspicion for OSA)



Case Studies

- Anyone can submit cases: <u>www.vcuhealth.org/echodmhtn</u>
- Receive feedback from participants and content experts
- Earn **\$150** for submitting and presenting

Provide Feedback



www.vcuhealth.org/echodmhtn

- Feedback
 - Overall feedback related to session content and flow?
 - Ideas for guest speakers?

Access Your Evaluation

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Education

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Virginia Opioid Addiction ECHO

Virginia Sickle Cell Disease ECHO

Diabetes and Hypertension Project ECHO

Welcome to the Diabetes and Hypertension Extension for Community Health Outcomes or ECHO, a virtual network of multidisciplinary diabetes and hypertension experts. An ECHO model connects professionals with each other in real-time collaborative virtual sessions on Zoom. Participants present de-identified cases to one another, share resources, connect to each other, and grow in their expertise. This ECHO will address practice level issues and solutions related to managing complex patients with difficult to control diabetes and hypertension. Register now for an ECHO Session!

Network, Participate and Present

- Engage in a collaborative community with your peers.
- Listen, learn and discuss informational and case presentations in real-time.
- Take the opportunity to submit your de-identified case study for feedback from a team of specialists for diabetes and hypertension.
- Provide valuable feedback.
- Claim CE credit by texting in attendance.

Benefits

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VCU Diabetes & Hypertension Project ECHO Clinics

2nd and 4th Thursdays — 12-1:30 p.m.

Mark Your Calendars — Upcoming Sessions

March 11: Concentrated Insulins

March 25: HTN in pregnancy

Please register at www.vcuhealth.org/echodmhtn





Thank you, and see you in two weeks!



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