What’s New in Hypertension? A Contemporary Primer

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A recent commentary of mine regarding hypertension was entitled, “Are Prescribing Practices for Antihypertensives Primitive? The Truth Hurts.” The criticism surfaced in the Wall Street Journal.

What’s the problem? The Journal quoted Dr. Michael Alderman, who appropriately insinuated that many of us—myself included—are in a stale time warp when it comes to antihypertensive therapy. So if this is the case, how about some new facts to bring us up to snuff? What are “hot topics” in the contemporary world of hypertension that will rescue us from being labeled as primitive?

Defining, Diagnosing, and Treating Resistant Hypertension

The old paradigm: If a patient’s blood pressure isn’t controlled with 3 antihypertensive drugs, add more (the “damn the torpedoes, full speed ahead” older approach).

The new paradigm: If a patient’s blood pressure isn’t controlled with a 3-drug regimen (and if he or she is adherent to the regimen), stop and identify the patient as having resistant hypertension. Consider associated disease states (eg, Obstructive Sleep Apnea [OSA] or chronic kidney disease [CKD]) and add spironolactone (25 mg/d to start) to the treatment regimen. Spironolactone’s potency in this particular situation may allow discontinuation of other antihypertensive medications.

Correctly Identify Populations That Frequently Have Resistant Hypertension

The old paradigm: Primary hypertension is all the same.

The new paradigm: Although secondary causes of hypertension may be active in individuals who have resistant hypertension (eg, primary aldosterone syndromes, pheochromocytoma, or renal artery stenosis), certain groups make up a significant portion of those with resistant, primary hypertension. OSA is one such cause. The rest of the news is good. Patients with OSA respond well to the addition of spironolactone. This drug not only helps with blood pressure control, but may benefit those with hypopneic and apneic episodes as well.

The epidemic of CKD has also prominently propelled this group into the resistant cohort. Data are preliminary and come from small sample sizes; however, it may be that we will one day prescribe spironolactone for this hyperkalemia-prone group as well. Wait for further news in this regard.

Thiazide Diuretics Are Good Drugs For High Blood Pressure, But They’re Not Perfect

The old paradigm: The first definition of resistant hypertension observed that of the 3 drugs used for control, one must be a thiazide. But what if a thiazide isn’t helping?

The new paradigm: The new classification for Chronic Renal Disease (the CKD stages 1-5) defines CKD stage 3 (the level at which patients should be referred to a nephrologist) as a glomerular filtration rate (GFR) of < 60 mL/min but ≥ 30 mL/min. Patients in this category represent another sizeable portion of the resistant group. Thiazide diuretics are not effective blood pressure medications when GFR is approximately 40 mL/min or lower. This is a treatment opportunity for furosemide, dosed twice a day because of its shorter half-life.

Not All Hypertensive Patients Are Created Equally

The old paradigm: Every hypertensive patient should receive the same battery of blood pressure medications (thiazide diuretics + beta blockers + an ACEI or ARB + a calcium channel blocker). This is the “one size fits all philosophy.”

The new paradigm: The old paradigm is wrong (it speaks exactly to Dr. Alderman’s point about primitive prescribing practices). Did you know that studies have demonstrated that beta-blockers either do not lower—or can actually raise—blood blood pressure in African Americans? In the ASCOT-BPLA trial (n= approximately 20,000), when atenolol was compared with amlodipine for blood pressure management, the trial was halted early because of a 11% lower all-cause mortality in the amlodipine limb.

If a black person with hypertension has an alternative indication for beta-blockade (decreased ejection fraction), use carvedilol instead.
Small Doses of Two Drugs are Better than Big Doses of Only One

The old paradigm: If 10 to 20 mg of an ACEI is good, then 30 mg is better and 40 mg is best.
The new paradigm: It is not only true that 2 heads are better than one. . . the same may be said for 2 antihypertensives.
A 2003 meta-analysis demonstrated that in 42 trials of hypertension therapy involving 10,968 participants, doubling the dose of monotherapy with 1 drug had only 1/5 the blood pressure lowering efficacy of adding another drug without increasing the initial drug’s dose.9

ACEIs with ARBs Don’t Add Bang To Your Buck as an Antihypertensive Combination

The old paradigm: If ACEIs are good for blood pressure and kidney disease, adding another similar renin-angiotensin-aldosterone medication (such as an ARB) will be even better.
The new paradigm: Adding an ARB to an ACEI regimen for blood pressure control provides only modest reductions in pressure (3 mm/Hg).10,11 The modest reduction may come at the price of hyperkalemia and blocking another medicine from doing a better job.
As in the first bullet point, the medicine to add in this setting may be spironolactone. This agent lowers BP in the same “add on” situation on average 25/12 mm/hg systolic and diastolic pressures, respectively.
These new paradigms are a starting point for antihypertensive therapy’s “Brave New World.” I don’t like being called “primitive” any longer.

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