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Potassium Management Made Easy

Announcer:
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Dr. Bakris:
Dyskalemia is common. Both hypo and hyperkalemia are associated with increased mortality and morbidity. It’s very important to know what the risk factors are for hyperkalemia, and we identified those a number of years ago in a large analysis, and they’re very simply two things. First of all, the kidney handles 90 percent of the body’s potassium, so reduced kidney function – GFR of less than 45 – definitely puts you at higher risk for developing hyperkalemia if you’re using agents that affect potassium in the kidney or are giving potassium. Secondly, it’s the serum potassium level itself. If it’s above four-and-a-half, you’re gonna be at higher risk for developing hyperkalemia, especially if you have reduced kidney function. Now, it’s important to understand this factor because the renin-angiotensin system inhibitors are critical in the management of kidney disease and heart failure, and diabetes, of course, is common to both genesis of those diseases, so these are very important drugs, which, in people with reduced kidney function, have limitations because of hyperkalemia. So, there really needs to be a way to manage hyperkalemia to enable or facilitate the use of these agents in this population of very high-risk patients. This is CME on ReachMD. I’m Dr. George Bakris.

So, let’s start with a patient case. This is a 54-year-old black man with a diagnosis of class III heart failure, diabetes, and chronic kidney disease, or CKD. Estimated GFR 43. Attempted to maximize heart failure therapy and CKD therapy, they were unable to tolerate angiotensin-converting enzyme inhibitors, or ACEI, except at very low doses, unable to tolerate spironolactone, intolerance to ACE inhibitor and spironolactone due to hyperkalemia – potassium greater than 5.5 – and note that his baseline potassium is 4.8 milliequivalents per liter. The patient requires optimization of RAASi therapy. Dr. Zannad, what is the level of risk for dyskalemia in this patient?

Dr. Zannad:
Well, this patient had the diabetes, heart failure, CKD, and a history of an episode of hyperkalemia, so this patient is really at highest risk for recurrent hyperkalemia because of these multiple risk factors, and this is very important because hyperkalemia is predictable, and we know about the risk factors, so we had better be really careful with these sort of combination of risk factors because hyperkalemia is a risk factor and risk marker, and we know that there is this U-shaped curve with low potassium is associated with worse outcome, but high potassium is associated with worse outcome, and this patient is at very high risk of a recurrent hyperkalemia, and in this sort of patient, because we know that the likelihood of a recurrent hyperkalemia is very high, we need to have a very careful monitoring of potassium, so repeatedly so, and actually the guidelines very clearly indicate that initiation or up-titration of any one of these RAASi and MRA need to be concomitant to potassium measure and then ideally three days later, one week later, one month later, and every four months. So, in a chronic state, we need to have potassium checked as per guidelines in normal condition every four months; in this condition because of the high likelihood of recurrent hyperkalemia, we may need even to have more frequent monitoring.

Dr. Bakris:
So, Dr. Weir, based on the totality of the literature, what conclusions can you reach about dosing of RAASi therapy and the risk of hyperkalemia?

Dr. Weir:
So, if you look at the totality of the available literature, we are learning a lot more about what are evidence-based doses of both ACE inhibitors, angiotensin receptor blockers, and mineralocorticoid receptor antagonists when but used to prevent progression of kidney disease or congestive heart failure. In general, the totality of evidence does support fully titrated doses. So, for example, in people with diabetic kidney disease, using a full dose of lisinopril or losartan or irbesartan has been shown to be effective in delaying progression of kidney disease, whereas lower doses we don't have any evidence at all really. Likewise in heart failure, evidence from clinical trials have looked at what is the right dose of the ACE inhibitor or the angiotensin receptor blocker or the spironolactone or eplerenone in terms of slowing progression of heart failure, and, again, the evidence is clear – lesser doses don't provide the same degree of benefit at all, and that is the major concern about why in clinical practice we need to optimize doses based on what's been shown to be effective.

Dr. Bakris:
You've got a patient like this where the potassium is greater than 5.5, and understanding that dietary management is going to be very difficult by itself, how do you approach this patient to achieve a safe potassium level?

Dr. Zannad:
Well, potassium diet is really challenging but should not be forgotten. I know that you guys in nephrology do it all the time that you sit down with your patient and explain the evidence of potassium diet. We don't do it much in cardiology. We should do it. We should not forget it because it may help at least in the chronic stage because, of course, there are alternatives, and the alternative in this specific case where we have a plain potassium diet is potassium binders. Well, luckily we have a new kids to the block here with at least two potassium binders, patiromer and ZS-9, which are very much better tolerated than the former potassium binder, and with the, a very large record of evidence of efficacy, tolerance, and usability, and, therefore, it is really very recommended that in this sort of patient we start these patients on any one of these potassium binders, and actually even if patient is already with high potassium, we can get potassium down to normal value, and also chronically keep the patient on potassium diet if the patient is a recurrent hyperkalemia, and this will allow up-titration. We have done trials of up-titration, forced up-titration, which was possible in patient taking potassium binders, and, therefore we need to reach guideline-recommended doses, and there is no limit. The limit is the guideline-created dose, and we need to get to the patient back each time to get any chance to get to this maximum dose because condition may vary, diuretics dose may vary, diet may vary, patient may, condition may vary, and then we should never give up until we are at the upper tolerated dose or to the guideline-recommended dose.

Dr. Bakris:
Thank you very much. Now, I'm gonna ask you both another question with a little bit of a different spin. In patients with heart failure that have normal kidney function, it's pretty easy to manage in terms of RAASi therapy, but obviously as we've already stated, it's difficult in people that have compromised kidney function, especially with GFRs of 45 or less. So, Dr. Zannad, how do you approach the heart failure patient with compromised kidney function in order to get them on RAASi therapy?

Dr. Zannad:
So, this is a really very important question because these patients with some CKD or kidney dysfunction are at highest risk of developing adverse events, including hyperkalemia, but they are also at high risk of developing cardiovascular and renal outcomes, and they are at the highest need of RAAS inhibitors therefore, and they need not be deprived from this therapy just because of concern about hyperkalemia. There are ways to get them on a protective therapy and RAASi inhibitors, and these ways include and, the use of potassium binders and the key approach in managing RAAS inhibitor in those patients is indeed to keep trying to initiate this therapy and up-titrating with the help of potassium binders whether it is used temporarily if patients run into hyperkalemia or on a continuous basis if patient is at risk, such as in this specific case, of recurrent hyperkalemia.

Dr. Bakris:
So, Dr. Weir, same question from a nephrology perspective.

Dr. Weir:
Well, George, this is a very interesting question that you posed. You know, when people have chronic kidney disease and reduced ejection fraction, it makes things that much more complicated because, on one hand, you don't want to boost the diuretic support, which may cause prerenal azotemia and hypotension and loss of kidney function so you really have to come up with another strategy. Dietary measures don't always work, so in my mind, I'd rather use a potassium binder that's well tolerated and effective so that I don't need to use diuretics as much, so I don't need to count on the diet as a consideration, and that way I can optimize my medical therapy for these

patients. Additionally, there are other considerations as well, and we've recently written a review which was published in the Journal of the American College of Cardiology, which I think outlines a lot of the different strategies one can use based on kidney function, serum potassium, and the degree of reduced ejection fraction in your patients.

Dr. Bakris:
Well, these are excellent points that you've both made, and unfortunately that's all the time we have today. I'd like to thank Drs. Zannad and Weir for helping us with the understanding of this complex area of managing potassium and at the same enabling agents to reduce mortality risk. Drs. Zannad and Weir, it was great speaking with you today.

Dr. Zannad:
Thank you so much. Very pleased to be part of this discussion.

Dr. Weir:
Thanks for having me today. I enjoyed it very much.

Dr. Bakris:
And thank you.

Announcer:
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