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ReachMD
www.reachmd.com
info@reachmd.com
(866) 423-7849

CKD, Diabetes, and Albuminuria: Keeping Kidney-Protective Therapy on Board Despite Hyperkalemia

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Dr. Kelepouris:
This is Continuing Education with Global Learning Collaborative, and I'm Dr. Ellie Kelepouris. Joining me today is Dr. Pauline Swift. In this episode, we're focusing on the dilemma of maximizing treatment for CKD, diabetes, and albuminuria while avoiding hyperkalemia.

Pauline, I understand you have a patient case for us to discuss. What do we need to know?

Dr. Swift:
So let's just consider a 68-year-old man with type 2 diabetes. He's got CKD G3a-A3, and his eGFR is now 38 mL per minute, and he has high-grade albuminuria with a urine ACR of around 110. So he's receiving an angiotensin receptor blocker, although he's not yet on the maximum tolerated dose, and he's started an SGLT2 inhibitor, all good.

But despite this, he remains at high risk of kidney disease progression and, of course, cardiovascular events because of his persistent high-grade albuminuria, and consideration, therefore, has been given to starting him with further optimization for his cardio-kidney protective therapy with finerenone, or a licensed nonsteroidal mineralocorticoid receptor antagonist. But here is the difficulty: he's had recurrent mild to moderate hyperkalemia with potassium values ranging between 5.4 and 5.9 mmol/L, and on a previous occasion, he was asked to reduce the dose of his angiotensin receptor blocker, which is already not at the sort of maximum tolerated dose.

So for me at this point, I think we need to step back a little bit and ask whether we're treating the lab results, the high K, and at the expense of really optimizing his therapies that are modifying this patient's long-term risks, because he's at long-term risk of kidney disease progression and, of course, cardiovascular events.

So for me, the first step is to confirm that the hyperkalemia is genuine and persistent and to ask if there is anything contributing. I personally would review the trajectory of their kidney function, intercurrent illnesses, whether they're constipated or not, whether they have metabolic acidosis, and look at their glycemic control. And I'd also, of course, review their medications carefully, looking for concomitant meds that can increase potassium, such as nonsteroidal anti-inflammatory agents, maybe salt substitutes with potassium chloride substituting for sodium chloride, and then I'd consider whether the diuretic therapy had been altered, perhaps reduced. And then I'd of course think about individualizing their dietary advice with just sort of blanket advice, really, but looking more specifically if there's any really high potassium things that they're having.

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Then we really have to think about a planned, long-term potassium management strategy. We do not want them coming in and out of the emergency room with high potassium, having their drugs stopped or reduced, and really inconvenient for everybody. So we have to think about the binders, the potassium binders.

And again, we should be individualizing them. So this patient has difficult blood pressure to control. He may or may not have edema and a tendency towards volume overload. And I may favor, if that was the case, a patiromer-based binder because it doesn't involve sodium exchange. But in contrast, if he presented with hyperkalemia requiring an ED admission, then I may want to use something that has a very rapid onset of action, such as SZC.

So our overarching goal really is not simply just to normalize the potassium, but to manage it safely and in the long term, and prevent this bouncing up and down of them coming in and out of hospital and their therapies being increased and reduced.

Dr. Kelepouris:
I think the important caveat here is how do we really manage patients with medications and actually how we can improve their quality of life. One of the first conversations that I have with patients is their diet. What is the potassium load in your diet? What is the potassium burden? And our knee-jerk reaction is to say, oh, you have to go on a potassium-restricted diet when you have CKD stage 3.

In fact, if you read the literature, there are benefits of nonrestrictive potassium diets in these patients if they don't have advanced chronic kidney disease and a GFR less than 15 or 20. And those benefits include really it may lower blood pressure, they're associated with lower risk of stroke, and also it supports overall health, but also quality of life so that the patients are not burdened with a specific diet.

But when you have advanced CKD and your GFR is dropping and you have albuminuria and you have all these wonderful medications to reduce albuminuria and arrest the progression of kidney disease—because this is what we're supposed to do, right, as physicians, do a holistic care approach—then using a binder in order to increase their GDMT therapy, not only for cardiovascular progression but for kidney progression, I think, is really critically important.

And all the guidelines, particularly on the kidney space as well as the heart failure space, really point to the importance of continuing GDMT in our 4 pillars, as you so eloquently pointed out, and actually using potassium binders in order to facilitate the use and up-titrate these agents, which have been really lifesaving

Dr. Swift:
And so historically, and I hate to say it, we have seen hyperkalemia as a reason to tell them to eat a less healthy diet and then to reduce or stop their RAASi therapy and their MRA therapy and everything else. But that, of course, leaves our patients really exposed to greater residual kidney risk and residual cardiovascular risk. So we are not doing them any favors by taking that approach.

So I think we should be asking: Can we manage the potassium differently with a binder rather than sacrificing their protective therapies?

And of course there'll be circumstances where potassium is sufficiently elevated, kidney functions really become unstable, or they're acutely unwell, where we have to stop those therapies, and safety always comes first. But really, what we're talking about here is the long, chronic management of mild to moderate hyperkalemia, and in those circumstances, we definitely shouldn't be reducing their GDMT, and we should be thinking long term, saying this is not an acute event, this is predictable. We need to manage a long-term strategy using potassium binders according to the phenotype of the patient that we've discussed before, with resistant salt-sensitive hypertension, edema from their heart failure and their diabetes, etc.

So the important shift for me is not which cardiorenal drug should I stop or discontinue or reduce the dose of, but rather how can I really help this patient and manage their potassium safely so that I can preserve therapies that are going to keep them out of hospital and improve their long-term outcomes?

Dr. Kelepouris:
I agree 100%, and really, Pauline, I think we nailed this. And I hope this case will be useful to you, our audience, in your practice. And thanks for listening.

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