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Cracking the Case: A Clinical Consult on Potassium Binders in Patients with Chronic Kidney Disease and Heart Failure

Announcer:
Welcome to CME on ReachMD. This activity, titled “Cracking the Case: A Clinical Consult on Potassium Binders in Patients with Chronic Kidney Disease and Heart Failure” is provided by Medtelligence. Prior to beginning the activity, please be sure to review the faculty and commercial support disclosure statements as well as the learning objectives.

Dr. Taub:
When treating patients with chronic kidney disease and heart failure, healthcare providers may not be optimizing therapy due to concerns regarding hyperkalemia. Today, we'll review treatment strategies including RAS inhibition and MRA therapy in combination with potassium binders that healthcare providers can use to overcome therapeutic inertia in clinical practice. This is CME on ReachMD, and I'm Pam Taub.

Dr. Kielstein:
Hi and I'm Dr. Jan Kielstein.

Dr. Taub:
Jan, let's begin by reviewing a patient case.

Dr. Kielstein:
You know, Pam, I always love to discuss patients with cardiologists, and the reason is that there's a huge overlap of chronic kidney disease and cardiovascular disease, especially heart failure. And the worse the kidney function gets, the higher is the percentage of heart failure patients. And I'm really happy that the American Heart Association recently acknowledged that and put parameters of renal health, eGFR and uACR into the risk score that we are talking about.

Yeah, Pam, I brought us a very challenging patient, a nice elderly gentleman, 81 years of age. And he lived a full life so far, and didn't really take care of his health as much as he should. He has rather poorly controlled arterial hypertension for more than 30 years, and you can see that on the echo with an increase in the interventricular septum. He has diabetes, which is insulin dependent for the last years. And we think, even though we haven't biopsied him, that the combination of hypertension and diabetes really caused his renal impairment. On top of that, he has chronic heart failure with a reduced ejection fraction, and his peak NT-proBNP was 27,930, which is a whopper. He has coronary artery disease and got a drug-eluting stent in his RIVA in August of last year. And unfortunately, he was a smoker with 45 pack years.

And in this situation, it's really very complex to adhere to guideline therapy. And of course, we gave him the four pillars of heart failure therapy with an ARNI with a beta blocker with an MRA and with an SGLT2 inhibitor. And even though we were on the forefront of modern pharmacotherapy, giving him finerenone as an MRA, as he has type 2 diabetes and chronic kidney disease, we steered in a situation where his potassium level went up on a regular basis to about 5.5 mmol/L, and this in the light of a creatinine of 6.5 mg/dL, and

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a mild metabolic acidosis with a pH of 7.3 and a bicarb of 17.1, was basically the challenging situation where at least from the nephrological viewpoint, there is some need for action. And I'm very curious about your take on that.

Dr. Taub:
Well, this is a very complex patient, and he really illustrates the interplay between the multiple organ systems, the kidney, the heart, the endocrine system, and he really needs to be on guideline medical therapy to reduce his future cardiovascular events, including heart failure hospitalizations. And for this, he needs to be maximized on his RAS inhibition and also his MRA. And the hyperkalemia, for many clinicians, is challenging, and this will limit the tendency to increase the medication to guideline-directed doses. And so sometimes these patients are either on no RAS inhibition and MRA, or on very, very low doses where they don't derive as much benefit. So this is a great case in which we can utilize a potassium binder to reduce the potassium levels, and then this will allow for the patient to be on really important lifesaving guideline-directed medical therapy.

Dr. Taub:
For those of you tuning in, you're listening to CME on ReachMD. I'm Dr. Pam Taub and. Here with Dr. Jan Kielstein. We are discussing best practices for using potassium binders in patients with chronic kidney disease and heart failure.

So Jan, what is the evidence behind the use of potassium binders in clinical practice?

Dr. Kielstein:
The interesting thing is there is a lot of data. When you think about it, potassium binders had their early big days in 1961 with really studies that shouldn't be the basis to use them. And I think the advent of the modern potassium binders really made a change, because the licensing process and the study program for the new potassium binders brought us a lot of insight when and how to use those drugs and how to optimize and how to enable heart failure medication, like in the patient we discussed.

So due to the side effects of the old-school potassium binders, I think we have the two choices, patiromer or SZC, I always say it's zirconium because it's easier. So you have these two choices of modern potassium binders, either patiromer or zirconium-based binders. And the important thing is that those potassium binders are swiftly acting. So this is a change as compared to the old drugs we had at hand.

The other important thing is that they have a very favorable side effect profile, and we have a plethora of studies illustrating how those drugs can be used. And when we turn to patiromer, we have, for instance, a study exactly in the patient population we're discussing, a patient with kidney disease and hyperkalemia due to RAS inhibition. And what we learned from the study by Weir that celebrates the 10-year anniversary this year, because it was published in 2015, is that we can see that patiromer was effectively lowering the potassium level in CKD patients on RAS inhibition, and thereby enabling the continuation of those lifesaving drugs.

And this is basically also what I discussed with the patient. Because the first question, and frankly, this is not only a question of patients, but sometimes of colleagues of ours as well, that they say, 'Well, if we have hyperkalemia or the risk of hyperkalemia, why don't we just stop RAS inhibition?' And I think we should not rest to explain that stopping RAS inhibition is depriving patients of an outcome-improving drug class. I mean, it's not less than that. It's depriving them of a drug class that prolongs life and reduces hospitalization. So when you made that clear to patients, and sometimes to referring colleagues as well, it's easy to understand why binding potassium is such a big advantage, especially when we look at the side effect profile of patiromer, as we have discussed right now.

So in this particular patient, I was very happy that he very swiftly agreed to continue the outcome-improving medication, and we indeed maximized that. And at the same time, we started patiromer in that patient.

Dr. Taub:
Well, this complex patient is really important for us to appreciate, because many clinicians might look at this patient and just say, 'I really don't want to increase any agent that could impact the potassium levels.' But this is the absolute wrong thing to do in this patient, because the drugs that are going to give this patient the most mortality benefit are the RAS inhibitors, as well as the MRA. And so we can't use hyperkalemia as an excuse not to maximize doses of these important medications. And so we really need to overcome this therapeutic inertia that exists and start utilizing potassium binders. They're very well tolerated. And when we use them with these guideline-directed medical therapy agents, what we're going to see is an overall improvement in cardiovascular outcomes.

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And in the heart failure with preserved ejection fraction space, we've seen some great data from the nonsteroidal MRA, finerenone, how that is very useful in also decreasing heart failure hospitalizations. And so for patients who have heart failure with preserved ejection fraction, we're going to be using MRAs even more.

Before we wrap up, let's each offer a final take-home message. Jan, what do you hope our listeners will leave with today?

Dr. Kielstein:
We live in exciting times, and I refer to the medical field. We have an opportunity to treat heart failure that we never had before with outcome-improving medication. And we also have the opportunity to overcome obstacles, hyperkalemia being probably one of the most important ones. So the formerly deemed prohibitive hyperkalemia, to then continue lifesaving medication, is not an excuse anymore. So let's use potassium binders, and if you experience an episode of hyperkalemia, even in the out or inpatient setting, don't stop the outcome-improving medication, but think of how to tackle the potassium. Potassium level is not a problem for the majority of patients, and the patients should not stop eating healthy fruit and vegetables in fear of hyperkalemia. They should take their medication. They should eat right. And if potassium is a problem, we have a very good remedy for that.

Dr. Taub:
So well said. And as you mentioned earlier, we're in a new era with the next-generation potassium binders that are very well tolerated, and hyperkalemia should no longer be an issue that prevents clinicians from maximizing important guideline-directed medical therapy that is lifesaving for our patients. So we need to be maximizing the doses of RAS inhibitors and the MRAs and utilizing potassium binders if needed, so that we can improve patient outcomes.

Well, that's all the time we have today. So I want to thank our audience for listening and thank you, Jan, for joining me and for sharing all of your valuable insights and expertise. It was great speaking with you today.

Dr. Kielstein:
Thank you very much. And thanks to the audience, and tackle the potassium, because you can.

Announcer:
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