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Differentiating Oral Potassium Binders: Mechanism, Onset, and Sodium Load

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Dr. Kelepouris:

I'm Dr. Ellie Kelepouris. I'm here today with Jim Burton, my colleague and friend. And our task today, Jim, is to differentiate oral potassium binders by mechanism of action, efficacy, tolerability, and practical use considerations within the space of CKD and heart failure.

So we know that potassium binders may help prevent hyperkalemia; what do our listeners need to think about when it comes to mechanism of action, tolerability, and other concerns when using them in the real world?

Dr. Burton:

Yeah, that is a great question, and I guess when we have different agents to choose from in any class, I guess it's important to remember that these aren't necessarily interchangeable, and they do have different mechanisms of action, and the choice should reflect, ideally, the individual patient that you've got in front of you.

So patiomer is a non-absorbed polymer that primarily exchanges calcium for potassium in the colon, and it increases fecal potassium excretion.

Sodium zirconium cyclosilicate, or SZC—I'm going to say that from now on because that's easier—that selectively captures potassium in exchange for sodium and hydrogen. It's generally got a faster onset, whereas patiomer has a more gradual effect.

But and what I'm going to say is neither should replace the standard emergency treatment for life-threatening hyperkalemia, and we're going to be talking a little bit more about the sort of chronic management of hyperkalemia because both of them can support the longer-term potassium control and can help keep patients, as we've talked about in some of the other episodes, on their goal-directed medical therapies, including ACEs and ARBs and MRAs, ARNI, and so on.

And heart failure guidelines, kidney guidelines recognize that the potassium binders in these pathways, because ultimately keeping people on goal-directed medical therapy is going to improve outcomes.

So practical issues matter, patiomer combined with some other oral medications. So we like to take it around 3 hours after other medications. SZC can change the GI pH. So again, a couple of hours between other medications because it can temporarily affect

gastric pH. And tolerability is also slightly different. Patiromer, constipation, which is a factor involved with hyperkalemia, of course, is it can be a bit more of a problem. We need to think about that.

SZC sodium exposure and fluid status probably deserve a special mention as a 5-gram dose has got approximately 400 mg of sodium, although we don't really know how much of that is absorbed. But edema has been reported, and heart failure occurrences in some of the studies. And though we don't want to think about causality in that, because they weren't powered for those clinical outcomes, it's just relevant for those with heart failure or particularly tenuous volume status.

Hypokalemia is just worth mentioning because although it has been reported in the studies, actually that's with the higher doses and severe episodes are actually quite uncommon.

So to come back round to where we started at the very beginning with what I said, ultimately, they're not interchangeable; they have different modes of action, and it's very much worth individually choosing the one that's right for the patient that is in front of you, based on those kind of descriptions. And I guess you would probably say the same when you choose between them, Ellie. I'm sure you would.

Dr. Kelepouris:

I totally agree with you. I think what we're learning about potassium binders and other medications, actually, is that there are different phenotypes of patients. There are patients, as you said, with heart failure, edema, volume overload, perhaps those patients should not be exposed to a binder that has a sodium signal. There are ways around that, however. Really being a physician and prescribing a diuretic is something that would help in that situation. I think that's really very important.

And again, constipation and GI distress is important in the 2 binders. And there have been reports of low magnesium levels with patiromer, and I monitor magnesium levels in my patients as well as potassium levels because, of course, as you said, episodes of hypokalemia are seen as well, and some patients do need magnesium supplementation, and they need a break from the potassium binder if their potassium goes too low. I think that patients are different and they respond differently. I do think that the phenotypes will determine use. I, as well, follow the sodium signal literature with SZC, and I try to mitigate the effect with diuretics or use patiromer as an alternative for those patients.

Again, what's really important here is what you mentioned, and that is that guideline-directed medical therapy should not be discontinued or stopped because of any issues with hyperkalemia or a rise in the serum creatinine. The latter is really a hemodynamic effect, completely reversible. And the use of binders to partner with the guideline-directed medical therapy has been supported, as you said, by the kidney guidelines.

KDIGO succinctly said that, really, we should not decrease or down-escalate or stop guideline-directed medical therapy, particularly RAASi blockers, for fear of hyperkalemia.

Using alternative therapies such as potassium binders, moderating the potassium diet, I think, is also important. However, I must say that, in the renal literature, hyperkalemia is recognized as a side effect, let's say, of RAASi blockers, but however, we are increasingly recognizing that dietary burden of low-potassium diets in the patients really lead to nutritional deficiencies, and we're actually being asked to liberalize potassium in the diet of patients with CKD so that they can have a more well-balanced diet. And it really is a gold of care issue and a quality of life point. My patients always say, "I may not have 20 years to live, but the few years that I have to live, I want to be able to enjoy my meals with my family and friends."

Dr. Burton:

What I would say, from the trials around the efficacy of these in maintaining normokalemia, is that they work and they're generally well tolerated, and they continue to work while patients take them.

In the past, sodium bicarbonate is not very palatable. Other older potassium binders are not palatable and have side effects. Dietary restrictions are miserable for patients and have limited efficacy. And now we have something that works and is well tolerated and recommended by guidelines, as you've just said, Ellie. And I think they should be absolutely up there in our armamentarium for managing people like this.

Dr. Kelepouris:

Well, thank you for your insights, Jim, and thanks to our learners for joining us.

Announcer:

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