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Oral Potassium Binders: A Novel Approach to Curb Hyperkalemia in CKD and HF

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Dr. Rossignol:
This is CME on ReachMD, and I'm Dr. Patrick Rossignol. Here with me today is my colleague, Dr. Ellie Kelepouris. Welcome, Ellie.

Dr. Kelepouris:
Thank you.

Dr. Rossignol:
Ellie, how do potassium binders work?

Dr. Kelepouris:
Thank you, Patrick. There are two novel potassium binders which we use in clinical practice. One is patiromer and the other one is sodium zirconium cyclosilicate. These two novel potassium binders work by blocking and removing potassium in exchange for calcium with patiromer and hydrogen ion and sodium with SZC. So to further define the mechanism of action, let's talk about each one of these individually.

Patiromer is a non-reabsorbed polymer designed to bind and remove potassium in exchange for calcium, primarily in the colon, thereby decreasing serum potassium in patients with hyperkalemia. So in the colon, it blocks potassium absorption and does not allow it back into the systemic circulation. And as I said, it basically exchanges potassium for calcium.

Sodium zirconium cyclosilicate is similar. It's an oral medication, similarly binds potassium in the small and large intestine, and removes potassium in the blood in exchange for hydrogen and sodium, which are then reabsorbed.

Dr. Kelepouris:
Although both of these agents, patiromer and SZC, bind potassium, they do it with a similar mechanism in the colon. However, the counterion that's delivered is different. In patiromer, it's calcium, and in SZC, it's sodium. So there's potentially less risk of edema when using patiromer than there is with SZC used chronically.

It may be a dose-dependent effect with the latter agent, but it's certainly something that one needs to consider in this clinical space where patients really have sodium retention, edema formation, and may need diuretics to overcome the edema, which is noted possibly

with use of the other agent.

So the two are really very important tools we have in the treatment of patients who receive a class of drugs that either elevate potassium or, in diabetes, when we know that with low renin and low aldosterone states, patients with diabetes and chronic kidney disease have hyperkalemia as a cause very early on in their disease pattern.

Although they have been available for use and are safe medications with efficacy in this clinical space, they are not used as frequently as one would like to see, given the fact that patients really need potassium control in order to receive guideline-directed medical therapy. So to assuage this fear of hyperkalemia, these agents are very, very valuable additions to our clinical armamentarium.

Dr. Rossignol:
Thank you very much, Ellie. This was a great insight. I wish I may mention as a nephrologist that it's of paramount importance to maintain RAASi at highest tolerated doses, and these two potassium binders may get this enabled. Also, there is more evidence for patiromer based on dedicated trials to demonstrate such RAASi-enabling effect.

So this was really insightful, but now it's our time. Thanks for listening.

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