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Hyperkalemia: Is It Still a Challenge in 2023?

Announcer:
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Dr. Piña:
While it's common to see hyperkalemia in patients with heart failure with reduced ejection fraction who also have progressive chronic kidney disease, is it still a challenge to manage medically? It's clear from multiple studies that optimizing guideline-directed medical therapy, or as we call it GDMT, is a key component to better outcomes in these patients.

So where are we with hyperkalemia in 2023? Is it still a challenge?

This is CME on ReachMD, and I'm Dr. Ileana Piña.

Dr. Butler:
And I'm Dr. Javed Butler.

Dr. Rossignol:
And I'm Dr. Patrick Rossignol.

Dr. Piña:
So, Javed, tell me about your thoughts on this and let's address these issues. Give me a patient case.

Dr. Butler:
Yeah, so this patient case that I would like to discuss is really an incredibly straightforward case that we see in our clinical practice all the time. A middle-aged person, history of heart failure, low ejection fraction, comes into the hospital with decompensated [heart] failure, gets diuretics, feels better, diuresis, goes home. After 5, 6 months of weight loss, comes to the clinic in outpatient setting. The person was in the hospital, came in on suboptimal medical therapy, left suboptimal medical therapy, and has now come into the clinic but is feeling a whole lot better. And the question is: what do you do? And this is a person that predominantly, if you look at the clinical practice, this is what happens is that, other than diuretics, we do nothing in a hospital setting. Now the problem here is that there's a lot of things borderline and, you know, this is very common in patients with heart failure. So blood pressure is about 100, GFR [glomerular filtration rate] is about 35-ish, potassium level is 5.0. Yeah, right in that sort of borderline zone, not on SGLT2 [sodium-glucose cotransporter-2] inhibitors, not on MRA [mineralocorticoid receptor antagonist], half-dose of beta-blockers and low doses of ACE [angiotensin-converting enzyme] inhibitors, not on ARNI [angiotensin receptor neprilysin inhibitor]. And the question is, just because this person is doing okay, do you leave this person alone because they have diuresed and their shortness of breath has gotten better, or do we need to attack this patient's medical condition and treat them optimally? And if you were to do it, which combination and how do you do that?

So those are sort of the common conundrums that we face in the clinic all the time.

Dr. Piña:
And that's where heart failure becomes an art, because we know the guidelines, we know what works, we need to do it, and we just need to get with it. The STRONG-HF trial taught us a lot.

Patrick, what do you think?

Dr. Rossignol:
Yes, from the nephrologist perspective, we must be aware that CKD [chronic kidney disease] patients with heart failure are the patients most prone to routinely develop hyperkalemia with getting up-titrated with RAASi [renin-angiotensin-aldosterone system inhibitors] and initiating potassium binders are certainly a key feature to get this addressed and fixed.

Dr. Piña:
And it's a patient that we see all the time. Very rarely do I see anyone with kidneys that are okay; they're not okay.

Dr. Butler:
So this is what the irony is, right? So that our heart failure patients during the journey will sooner or later develop chronic kidney disease. Chronic kidney disease patients, chronic kidney disease puts them at a higher risk for recurrent decompensation and fluid overload, so our highest-risk patients are in the highest need for therapy, but guess what? They are the least likely to receive therapy. So there is this mismatch that the highest-risk patients receive the least therapy, MRAs are contraindicated, a GFR less than 30, RAAS inhibitors are substantially underused – not less than 30, 25, 20. Even less than 45, the clinicians have this discomfort because of the risk of hyperkalemia. So this is one challenge that we have. And then, we give them suboptimal therapy – guess what? Heart failure gets worse, chronic kidney disease gets worse, and it's a spiraling course.

Dr. Piña:
It is, and you're lucky that they sent the patient home on something. Very often in my institution they stop it.

Patrick, do you have a case?

Dr. Rossignol:
Yes, indeed I have in mind a patient presenting with hyperkalemia 30 days after having been discharged from his first episode of acute decompensated heart failure. This is a patient with a history of chronic kidney disease, actually moderate chronic kidney disease, with an eGFR let's say around 58 mL/min. It happens at 1 month after discharge; the patient is almost optimally treated, stable eGFR, but still presenting with moderate hyperkalemia, 5.6 mmol/L. The point is that do we consider maintaining RAASi, or should we consider discontinuing the RAASi, ignoring the fact that discontinuing RAASi may deprive this patient from lifesaving drugs?

We should consider, based on the latest guideline, initiating a potassium binder. And actually this is what was implemented in this patient. And this helped, first of all, neutralizing, normalizing potassium, and second, this enabled further up-titration of RAASi. Interestingly, we know from the DIAMOND prespecified analysis in the CKD subgroups that patients with more advanced CKD were even more likely to benefit from patiromer with a safety profile which was consistent across the CKD strata.

Dr. Piña:
And that's such a wise statement, and what normally happens is that everything gets removed because everybody is so afraid of the potassium getting worse that they stop everything. And now we know that withdrawal of the drugs is actually bad. This actually leads to worse outcomes, both hospitalization and mortality, including both.

Dr. Rossignol:
Yes, indeed, it was consistently demonstrated across observational data that patients who get discontinued RAASi because of hyperkalemia routinely experience worse outcomes.

Dr. Piña:
So what comments do you have on Patrick's case?

Dr. Butler:
Yeah, so I'm sort of really sympathetic to our fellow clinicians because you're really kind of stuck, right? So on one hand, you have this acute risk that you can say, "Well, I'm going to power through it. I'm going to give all the medications despite low GFR, despite borderline potassium." But boy, I mean, you know, if you do run into a potassium of 6.5 or have ventricular arrhythmias or something like that, I mean, you're taking that sort of risk. You can say, "Well, you know, first, do no harm." Okay? So I am not going to treat and take that risk, and I'm going to withdraw the medication. The problem is that it's not that you're not doing any harm, you're just exchanging the short-term harm for a long-term harm by taking away the medication and the chronic disease gets worse. So we need to

really find ways by which we can enable the therapy without increasing the risk so that we are not put in a place where you are exchanging short-term and long-term risks.

Dr. Piña:
For those just tuning in, you're listening to CME on ReachMD. I'm Dr. Ileana Piña, and here with me today are Dr. Javed Butler and Dr. Patrick Rossignol. We're discussing how to best address hyperkalemia in our challenging patients who have both CKD and heart failure.

So where does it fall in your treatment of a patient with HFrEF? Do you start talking to them about potassium early on? I do. I start asking what's their diet like? What are they eating normally? And I try to get a sense of how much potassium they're actually eating. Maybe they are even on potassium salts, so we need to ask those questions as well.

Dr. Butler:
And then from my perspective, dietary potassium control and dietary counseling is really, really important. And, by the way, most clinicians, including myself, I mean, I'm not trained to give a really good comprehensive counseling on diet, and especially with different people with different backgrounds have different diets, so referring them to a nutritionist at least one time may be a really good idea. But that is really good for preventing sort of the, you know – if somebody has borderline potassium and you want to maintain it, that's fine. You cannot trust dietary potassium control to up-titrate medications in high-risk patients. You've got to do something more than that.

Dr. Piña:
So now that we've looked at 2 different patients which have similar problems – they're different patients – I think very importantly, let's go back to why are our colleagues not using guideline-directed medical therapy? Every time we look at registries we see that we have fallen so short of giving the right therapies at the right doses for the right reason, which is really the definition of quality. So address that, Javed, for me?

Dr. Butler:
Yes, so I mean, this is really complex, right? So right off the bat people would say that cost is a big issue. That is true, but actually if you look at the registry data, even drugs which are generic, like ACE inhibitors and MRA, they're significantly underused. If you look at settings where 100% of the coverage is available, like the VA setting in the United States, again the use is very low. And even in the other private setting, if you have 20% of the people getting guideline-directed medical therapy, you don't have these issues in 80% of the patients. So its much more than that. So that's where inertia comes in. Now the question is, what is the inertia driven by? So part of the inertia may be driven by that, you know, you may not have the knowledge-base of self-efficacy. But part of it is tolerability and either real contraindications, relative contraindications, or perceived.

That's the problem. So I think between tolerability and perceived contraindication, that takes care of a bunch of reasons because of which that perpetuates that inertia cycle. So, you know, we can at least as clinicians attack what is in our control, and some of these issues related to perceived contraindications, perceived intolerance as an enablement. That is something that we can own and take care of.

Dr. Piña:
And unfortunately, some of that happens in the primary care office as these are patients that we do not see.

What do you think about the education that we need still to do?

Dr. Rossignol:
Actually, education is really key because observational data clearly demonstrated that worsening renal function, hypertension, and hyperkalemia are associated with down-titration, discontinuation. Therefore, we should teach our colleagues that there are some actionable items such as hyperkalemia, which by itself may be addressed with new potassium binders to enable RAASi routinely and hopefully enable better outcomes.

Dr. Piña:
So what level of potassium are you uncomfortable with patiromer to be given?

Dr. Rossignol:
Yeah, it's a difficult question because I have knowledge that a different medical doctor from different medical specialties may observe differently. But, personally, I acknowledge that the new potassium binders were developed based on a definition above 5 mmol/L. This was the threshold which we –

Dr. Piña:
Do you get nervous at 5?

Dr. Rossignol:
I don't get nervous at 5. I personally, I get nervous, I would say, at 5.5, but observing that the risk, based on empirical data, is increased even above 4.5. So the compromise, to my understanding, is to pay attention to the level of RAASi drug. There's a target dose which I may want to achieve and get the patient normalized in the setting of optimized GDMT.

Dr. Piña:
So, Patrick, I think you just make a great point about the need to sort of think ahead of what could happen, and now we have the STRONG[-HF trial] heart failure data that we know that being aggressive and doing it early and rapidly is really safe.

So this has been a fascinating conversation with friends, but before we wrap up, Javed and Patrick, do you have any take-home messages that you would like to share with our audience?

Dr. Butler:
So I would say that I think it's critically important for people to realize that symptoms is a target of therapy, not a guide of therapy. We obviously need to make, you know, do whatever we need to do to make our patients feel better, but things like diabetes, high blood pressure, dyslipidemia – these diseases are asymptomatic to begin with, but nobody is going to withhold therapy. Many cancers are diagnosed with screening when the patients are totally asymptomatic.

So we need to treat the biology of the disease. So if somebody has less symptoms or more symptoms, that doesn't matter. They still have the disease, and they need to be treated as aggressively and appropriately as possible.

Dr. Piña:
Any final messages from you?

Dr. Rossignol:
Sure, I would say that new potassium binders along with a proper creatinine and serum potassium administering is a major asset to our optimization of GDMT for our CKD and our heart failure patients.

Dr. Piña:
Unfortunately, that's all the time we have today, so I want to thank our audience for listening in and thank you, Dr. Javed Butler and Dr. Patrick Rossignol, for being with me today me and for sharing all those valuable insights and your expertise. It was great speaking with you today.

Dr. Butler:
Absolutely a pleasure.

Dr. Rossignol:
My pleasure, Ileana.

Announcer:
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