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Released: 03/15/2023
Valid until: 03/15/2024
Time needed to complete: 15 minutes

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Don’t Let Hyperkalemia Get You Down When Potassium Goes Up in Your Patients with Heart Failure

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Dr. Butler:
These days, it's common to see patients with heart failure who also have progressive chronic kidney disease [CKD]. These patients are at risk for developing hyperkalemia, which compounds the challenges of medical management. The evidence is clear, however, that the suboptimal use of guideline-directed medical therapy [GDMT] leads to poorer outcomes for our patients. So how can we approach these complex cases, and what strategies can be used to make sure our patients have the best possible medical therapy and outcomes?

This is CME on ReachMD. And I'm Dr. Javed Butler.

Dr. Piña:
And I'm Dr. Ileana Piña.

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

Dr. Butler:
Well, great to have both of you. So in order to discuss this sort of complex issue, perhaps we can discuss a case that sort of illustrates all of these issues of multiple comorbidities that our patients face.

So, Dr. Piña, maybe I can ask you if you can remember some case from your practice that you would want to share with us?

Dr. Piña:
Absolutely, Javed, and thank you for the invite here. Cases are always very important because they really do illustrate what's going on. So I have this 72-year-old gentleman who used to be a stockbroker, retired. And he had an acute MI 7 or 8 months ago and ended up with an ejection fraction of 25%, but had a lot of mitral regurge, really moderate to severe. And when he was sent home from the hospital, he was put on hydralazine and a nitrate, because everybody was afraid of his renal function, and IV milrinone. His creatinine had been like about 2.5. And so when I saw him, I started him on just 2.5 of enalapril BID to see how he would do. And then his creatinine rose, as we would all expect it to do, to 3.5, and the nephrologist told him his kidneys were damaged and that he needed to stop the enalapril immediately. At that time, his potassium was 4.6.

So I cut back the diuretic, but I continued the enalapril, gradually increasing it across time, up to 15 mg twice a day. He felt so much better. His shortness of breath got better. At one point, I was even able to remove the diuretic because he was doing so well. And now he was on spironolactone; he was on an SGLT-2 inhibitor, in this case, it was dapagliflozin; and he was on metoprolol succinate, at 100.

His potassium was 5.2.

And because I had some blood pressure room still, I increased him to 20 of enalapril BID. He was still feeling better. And I tried to even drop the milrinone dose; that's my final goal, to get them off it. And his creatinine was 1.7 now, but his potassium rose to 5.7, and then 6 on repeat. I started him on patiromer 8.4 mg once a day, maintained everything else the same, I did not back off on anything, and the potassium dropped to 4.6. And he is still doing quite, quite well. And I'm going continue to try to get him off the milrinone.

Dr. Butler:
Well, what an interesting case. And the reason why I say “interesting case” is because this is not some, you know, fascinating, unusual diagnosis. I mean, this is sort of the bread-and-butter cases that we all see in our practice on a daily basis. And how to manage these patients, that's really challenging. So really, thank you very much for sharing this case.

But let me ask you a question. You know, initially, the potassium had already gone up to 5.2, but you continued to forge forward with going up on medical therapy. And then subsequently it went up. And at that point, you decided not to compromise medical therapy, but instead enable medical therapy with the use of patiromer. So as a cardiologist, can you tell me how you think about hyperkalemia? How do you define hyperkalemia? Do you have like a fixed number, or it actually depends on the patient and their risk factors that is sitting in front of you? And in general, just some idea about how hyperkalemia affects guideline-directed medical therapy.

Dr. Piña:
The thing I think we have to remember as cardiologists is that when we're so afraid of higher potassium, it's really potassiums that rise suddenly, not these chronic increases. And I think all my years of doing this, I have always tried to keep the potassium right around 5. I think it's protective of arrhythmias; maybe I'm wrong about that. But when I see a 4, then I get more worried. And so when I saw him going to 5.2, but he was looking clinically so well, and the creatinine was actually getting better, not worse, I just forged on. Lucky for me, since he's on milrinone, he gets weekly potassium checks. He gets weekly panel so that I can see it, you know, pretty continuously. And then it started to creep up. And when it crept up to 5.7 and then the repeat was 6, I said, let me calm everybody's nerves down. And he's fine. He was fine. He has an ICD [implantable cardioverter defibrillator] in. I started the patiromer, and he's doing quite well. I'm going to see him actually next week. He's doing quite well.

Dr. Butler:
Yeah, that's great, because getting somebody off of milrinone, I mean, you know, eventually, I mean, that's a huge benefit as well, not only just improving their heart failure symptoms.

Any perspective from a nephrologist, Dr. Rossignol?

Dr. Rossignol:
Yeah, sure. First of all I wish to mention that I fully agree with the approach Dr. Piña implemented on her own. One may remind that in heart failure without clinical signs of congestion, down-titration of diuretics may favor the titration of RAAS [renin-angiotensin-aldosterone system] blockers; whereas the use of patiromer was indeed repeatedly demonstrated by randomized placebo-controlled trials to enable RAASi [RAAS inhibitor] maintenance. And regarding the hyperkalemia threshold that Dr. Piña discussed, I should mention that within an international multidisciplinary panel, cardiologists, nephrologists, including Dr. Butler as senior author, recently published a paper in pharmacological research entitled “Pragmatic Diagnostic and Therapeutic Algorithms to Optimize New Potassium Binder Use in Cardiorenal Disease.” And in this setting, we recognized a serum potassium between 5.1 to 5.5, confirmed, by the way, by 2 consecutive valid samples, as a mild hyperkalemia, and we stated, consider initiation of a new potassium binder.

Dr. Butler:
You know, from my perspective, I mean, I completely agree with both of you. And also, you know, what potassium level you react to is partly a medical decision, but partly is a little bit of a social decision as well, right?

For those just tuning in, you're listening to CME on ReachMD. I am Dr. Javed Butler, and here with me today are Dr. Ileana Piña and Dr. Patrick Rossignol. We're discussing how best to address hyperkalemia in a challenging patient with CKD and heart failure.

So, Patrick, let me come back to you. Can you tell us a little bit about what the guidelines are saying about GDMT and about management of these patients and management of hyperkalemia, both from a cardiovascular and from a kidney perspective?

Dr. Rossignol:
Sure. Overall, considering the latest KDIGOs [Kidney Disease: Improving Global Outcomes] in CKD on heart failure, both ESC [European Society of Cardiology] and HFA [Heart Failure Association] treatment guidelines, they univocally recommend RAASi therapy, as you rightly mentioned, at the highest approved dose that is tolerated to improve patient outcomes because the proven benefits were achieved in trials using these doses.

Furthermore, the KDIGO CKD and ESC heart failure treatment guidelines recommend novel potassium binders to treat hyperkalemia and enable GDMT, more precisely, the ESC heart failure guidelines state that in chronic or recurrent hyperkalemia, an approved potassium-lowering agent should be initiated as soon as potassium levels are confirmed as above 5 mmol/L. One should maintain K^+-lowering agent unless alternative treatable etiology for hyperkalemia is identified. Of course, the AHA/HFSA guidelines also acknowledged that in patients with heart failure with pronounced hyperkalemia above 5 to 5.5 mmol/L while taking a RAASi, the effectiveness of potassium binders – patiromer or sodium zirconium cyclosilicate – to improve outcomes by facilitating continuation of RAASi therapy is still uncertain.

But again, as a nephrologist, I would certainly recommend the optimization of GDMT as life-saving drugs. I would implement the pragmatic diagnostic and therapeutic algorithm I referred to earlier on to get potassium normalized with choosing a new potassium binder. Monitoring is really simple and encompasses, for instance, for patiromer, after initiating or changing patiromer dose, just measure serum potassium and creatine within 3 to 7 days and repeat after 1 week. If target potassium value is achieved, measure serum potassium at 1 month, then every 3 months.

Dr. Piña:
And, Javed, another point here is you've got to look at the patient. This gentleman of mine was really, like, blossoming. He was feeling so much better. His activity level was better. Removing the diuretics for me is always important and giving him a flexible diuretic regimen. So you've got to involve the patient in some of these decisions and tell them why you're doing what you're doing.

Dr. Butler:
You know, sometimes we sort of fall into this trap of taking the average results from clinical trials that have several hundred or several thousand patients and extrapolate that result to the patient sitting in front of us. But obviously, a part of medicine practice is science and part is art, and individualizing it to the patient sitting in front of you is sort of really important.

And the other sort of message that I'm hearing both of you say sort of indirectly is that improvement of patients' symptoms is really important and a really important target, but it's not the only target. We need to sort of curb the progression of the disease and long-term trajectory of the disease beyond symptoms as well.

Ileana, let me come back to you and dig a little bit deeper on the potassium binders. So what do we know about these novel potassium binders? And then I'm going to play on words here a little bit, a little bit of nuance about the continuation of RAASi therapy in appropriate patients with these potassium binders, or an enablement of RAASi therapy with these agents. Can you tell us a little bit about potassium binders?

Dr. Piña:
Sure, absolutely. It does take a little education because this is different than a pill. This is a powder that you have to mix. And I warn them that there may be a little bit of constipation. But what we do know is that these binders are absolutely safe. That's why they're approved; they're absolutely safe. And importantly to tell the patient if you're going to stop it, that you need to tell us about it so that we can keep a close eye on that potassium. But we don't see these sudden rises in potassium, even if the patient did not take a dose or 2. The increase in potassium happens again, but it happens very, very slowly.

And now we have the DIAMOND trial added to the old PEARL trial that shows that patients not only can be sustained on their RAASi therapy, but can actually be up-titrated, which I've done in this gentleman.

So I think that the data that we have are pretty powerful for being able to continue the RAASis. And I know personally, again, after doing this for a long time, that if I am forced to back off on the RAASi, there will be a change in patients' symptoms. And that's what worries me. When the patients are doing well, they're out of the hospital, they haven't been hospitalized again, to me that's a win.

Dr. Butler:
Super. Dr. Rossignol, any perspective from a nephrology side?

Dr. Rossignol:
Sure. First of all, I fully agree with Dr. Piña's viewpoint. Maintaining RAASi was consistently found associated with better outcomes in half of our patients, while hyperkalemia in itself might be associated with worse outcomes through the discontinuation of RAASi; therefore, new potassium binder represents, from my perspective, a fantastic opportunity to maintain RAASi in the long run. And the DIAMOND trial that Dr. Piña referred to definitively ascertains that it is doable, sustainable in the long run, and safe. Again, this warrants appropriate creatine and potassium monitoring.

Dr. Butler:
Well, this has been an interesting discussion, but it's time to wrap. But before we wrap up, can you both give your final take-home

message from our discussion today? Dr. Piña?

Dr. Piña:
I think your statement of heart failure management can often be an art and that you have to balance, always, the risk and the benefits. But to me, the benefits of RAAS inhibition really supersedes, at this point, the potassium, particularly if I can control it with the new potassium-binding agents.

Dr. Butler:
And Dr. Rossignol?

Dr. Rossignol:
Sure. As a combined use of new potassium binders and the proper monitoring is a major asset to optimize the management of high-risk patients with avoiding the risk of both hypo and hyperkalemia on suboptimal RAASi therapy, just do it.

Dr. Butler:
Yeah, I would second both of those comments, that if a person meets the indication for the therapy as per the guidelines and as per the clinical trials, then we should really give it as aggressively and as up front to avoid progression of the disease and not wait for progression of the disease. And I think that's probably my biggest take-home message.

Well, unfortunately, that's all the time that we have today. It's always less time than what I can discuss with the 2 of you. So thank you so much for sharing the case, Dr. Piña, and to both of you for sharing your insights, both in terms of the science and the practice of how to manage these complex cases. And I also want to thank our audience members, and I hope that this discussion was of help to you. So thank you so much, both of you.

Dr. Piña:
Thank you both.

Dr. Rossignol:
Thank you very much. Bye-bye.

Announcer:
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