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Hyperkalemia as a Barrier to GDMT: Why “Stopping GDMT” Is Often the Wrong Reflex

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Dr. Burton:
Hi everyone. This is CE with GLC, and I'm Dr. Jim Burton. And here today with me is Dr. Nihar Desai. And in this particular episode, we're going to be talking about that dilemma of maintaining optimal guideline-directed medical therapy in patients with mild or moderate hyperkalemia.

And, Nihar, I understand that you've got a patient case to discuss around this and what we're going to need to know around it.

Dr. Desai:
Yeah, Jim, great to be with you. And yeah, I thought I would present a case that I suspect is familiar to you, familiar to our audience. It kind of centers around a patient with reduced ejection fraction heart failure, moderate sort of CKD, that then ultimately develops mild to moderate hyperkalemia. And then we're caught in that very frequent situation of what are we going to do next? And I know that comes up in your practice a lot. I feel like it comes up in my practice a lot. I think it comes up in everyone's practice, and so I hope that maybe that that discussion can be engaging for the audience and really help them think about how might they approach that when they encounter it tomorrow or the day thereafter.

So this is a case of a 68-year-old man with ischemic cardiomyopathy, an ejection fraction of 30%, and you're sort of getting acquainted with him and having an office encounter, and of course you've emphasized the importance of guideline-directed medical therapy, you've sort of charted a course based on what he's already on, which was an angiotensin receptor–neprilysin inhibitor at 24/26 BID, metoprolol succinate 100 mg daily, and an SGLT2 inhibitor. And you've elected, as I think all of us could and would and should, to add a mineralocorticoid receptor antagonist. In this case, spironolactone 25 mg daily.

And I think, as all of us then would be accustomed to doing in our clinical practice, we get some chemistries back, and sure enough, the creatinine has risen from 1.8 to 2.1, and the potassium has risen from 4.7 to 5.6.

And so maybe, Jim, I'll sort of pause there and just say again that this is probably something that happens every day in many of our practices: a decision about guideline-directed medical therapy, a plan for some surveillance laboratory testing, and then the development of hyperkalemia. And so maybe with that, I'll just pause and maybe kind of ask you for some thoughts. How might you approach that? How might you think about that? And then I might sort of share some thoughts as well.

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Dr. Burton:
Well, I guess like you, now I see this kind of thing all the time, and I get called from my colleagues to say, “Oh I've got this person, and ideally I want them to be on these core pillars of therapy for heart failure you've mentioned,” and we know that 2 of the reasons why people have their core therapies discontinued or down-titrated are, number one, a rise in creatinine or fall in GFR because they go together, and, number two, a rise in potassium. So we know that both of those things would be the main top 2 reasons for people either scaling back or even stopping those core therapies for people with heart failure. And I get asked those things a lot.

And the other thing, of course, to mention from my lens as well as yours, is that probably about 50% of people that you describe with heart failure in that way have got abnormal kidney function, and so it's a really common problem that, as you say, we both see in our core clinical practice. So what would you do then?

Dr. Desai:
It's a great point, and I think for us, there's a couple of things maybe that are worth just reinforcing. I couldn't agree more with what you kind of laid out for the audience, and I think maybe one additional point to make is just how important guideline-directed medical therapy is, that the stakes are very high. The urgency for getting patients on these sort of core pillars, these foundational therapies, is really essential. It's critical.

The morbidity and the mortality benefit of these therapies in reduced EF heart failure plus/minus the very important added dimension of kidney disease as an important kind of risk enhancer but also another important element that needs to be thought about as part of the comprehensive care of these patients really can't be overstated. And so I think it's nice that that awareness has gotten out, because, again, the stakes are very high and the urgency is very real.

And so for us I would say we've had this reflex for too long, and in some ways there were professional society recommendations and even older versions of the guidelines that actually reinforced this reflex of yes, by all means, try and get patients on the 4 pillars for reduced EF heart failure. But if and when you encounter hyperkalemia, that the next step after sort of maybe some thoughts about dietary counseling and making sure that there weren't other kind of reversible contributors to that hyperkalemia was to down-titrate or to discontinue background RAASi therapy, the ACE inhibitor, the ARB, the ARNI therapy, and the mineralocorticoid receptor antagonist.

And I think all of us felt uncomfortable in doing that because we said, well, geez, the benefits of these therapies are so substantial, and yet in the face of an electrolyte abnormality, we're going to now de-intensify that therapy. And I think obviously we've now come a long way. I think we've learned that what happens to those patients after you discontinue and de-intensify their therapy is not good, that there's an increase in hospitalizations, there's an increase in morbidity and mortality that is associated with that act.

And then relatedly, that we have another paradigm that we can follow, and that's to reach for a modern potassium binder that can address the legitimate concern over hyperkalemia and also enable and facilitate the maintenance and continuation of the very important background therapies that we have.

And so I think it's been a very interesting journey for us in our own practice of a very common issue, again, seemingly an everyday kind of occurrence, and we were sort of taught one thing, that we got accustomed to doing one thing, and now the sands have really shifted and we really have to evolve, that the evidence is really asking us to do something different. The professional societies are asking us to do something different, and increasingly, the guidelines are asking us to do something different. And I think that's really what I think is an important message for the audience as well.

Dr. Burton:
Yeah, I love what you said there about the reflex action, and perhaps as you say, the reflex action should be reaching for the potassium binders.

And I think central message from the ESC and from KDIGO is helpful, and that is that hyperkalemia should be managed with potassium-lowering measures, including potassium binders, and only as a last resort should we be down-titrating those core therapies.

So look, we're about to time. Nihar, thank you so much for sharing that case with us. We hope to the listeners that you'll keep that in mind the next time you see a similar patient. And thanks for joining us.

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