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The Binder Selection Playbook: Matching the Right Strategy to the Right Patient

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Dr. Burton:
This is of course CE with GLC, and I'm Dr. Jim Burton, and joining me today is my colleague and friend in cardiology, Dr. Nihar Desai.

So we know that multi-professional care can help to address those complex, interconnected management needs of patients with heart failure and CKD through that coordinated, individualized management. So in this episode, we're going to talk through 3 or 4 case studies and just review the relevant guidelines around that and perhaps some clinical trial evidence. We'll just talk about the things that led us to the decision-making in the examples that we're going to present to everyone today. So let's get started.

Nihar, I think you've got someone for us to have a think about first.

Dr. Desai:
Sure, Jim, that sounds great, and I think the first case is, again, one that'll be very familiar to you and to our audience: a patient with reduced ejection fraction heart failure who is hospitalized.

This happened to be a 74-year-old woman with a non-ischemic cardiomyopathy, an ejection fraction of about 25% to 30% admitted with an acute decompensation of her heart failure. Her background and contextual history is notable for breast cancer, stage 3b chronic kidney disease, type 2 diabetes, and hypertension. Sort of at baseline, has Class II/Class III symptoms, but now of course is hospitalized. Her medications are then optimized during that hospitalization. She's on losartan at 50 mg, dapagliflozin 10 mg, metoprolol succinate 100 mg daily, and spironolactone at 25 mg daily. She's euvolemic, normotensive.

About a week later, there's some routine laboratory testing that's done by her primary care physician as kind of a post-hospital check-in and just given some of the changes that were made to her medications, and now her potassium is up to 5.6 and a creatinine that's up to 1.6 from 1.4.

So at that point, the provider discontinued the spironolactone, and unfortunately, about 6 weeks later, that patient was calling our office with progressive fatigue, dyspnea on exertion, weight gain. We had her unfortunately have to go back to the emergency department with recurrent volume overload, worsening heart failure symptoms. On admission, the potassium was 4.9. The BNP was markedly elevated, and it was really during the course of that hospitalization where we got nephrology engaged. We really sort of came together to talk through a multidisciplinary care plan, just like you had mentioned, Jim, and we sort of made the decision to reach for a binder in

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this case for chronic management as part of an enabling strategy of background guideline-directed medical therapy. We opted for patiromer 8.4 g daily, and then restarted the spiro at 12.5 mg daily.

How would you have thought about this case if you saw this person in the office or in the hospital? And then how would you have made that kind of decision maybe between patiromer and sodium zirconium cyclosilicate, just given this individual's kind of history and some of the clinical context as well?

Dr. Burton:
Yeah, I mean, I think that would resonate with most people, and we see these kind of stories in our cardio-kidney-metabolic clinic where I work as well. And you've got that exactly right. How many times we've seen where particularly the MRA has been discontinued because the potassium has gone up, and then they're back with heart failure syndrome symptoms.

And from a quality of life point of view, an outcome point of view, a hospitalization point of view, continuing on that goal-directed medical therapy is absolutely right, and reaching for a binder is also there in clinical guidelines for that very reason.

I'm going to talk about a similar case, Nihar, if that's all right with you?

Dr. Desai:
Yeah, that's great.

Dr. Burton:
This is from my kind of kidney lens. Okay, so I'm going to talk about one that I get asked about a lot. So let's say for example, it's a 70-year-old man, and he's got stage 4 CKD on a background of diabetes. He's also got heart failure preserved ejection fraction and a bit of albuminuria, and he's taking 10 mg of ramipril alongside a beta-blocker and an SGLT2 inhibitor with some diuretics. And over 9 months, let's say for example, his GFR has fallen gradually from around 23 mL/min to 19 mL/min, with a creatinine increase that fits with that. And his potassium is about 5.7 mmol/L. So he's stable. He's got no other acute problems. His volume status is pretty steady. And the immediate question I get asked is, should I stop or down-titrate the ramipril or the ARB, for example, because his kidney disease has progressed from stage 3 to stage 4, and his potassium handling has therefore become a little bit more difficult?

So the only thing I'm going to say before I sort of ask your opinion on that is about the STOP-ACE trial. So the STOP-ACE trial was a really important trial in the kidney literature where we looked at people who had more advanced kidney disease, and we thought if we stop the ACE inhibitor or the ARB, they'll get that reversible change, and they'll recover a little bit of kidney function, and surely that is good in that situation, and will prevent them from continuing on to kidney failure. And of course, the trial showed that against placebo, it didn't make a difference about that progression to kidney failure, it didn't make a difference about their starting dialysis, or in fact their cardiovascular events or mortality.

So that's great from a kidney point of view. But of course, a lot of these people also have other comorbidities like heart failure, where continuing those medications is really important, right? So the message should be that if they need to stay on it, they can stay on it.

Dr. Desai:
I think this is such another important case, one that frequently is encountered in our clinical practice. And I think I guess part of the challenge that we have to confront is just how siloed and maybe organ focused we can be at times. And what the patients really want and need and deserve is for people to really come together to think about them holistically and for us to really have the discussion that you were just having with the audience around, hey, maybe from a narrow lens, stopping that background therapy might not have such an important clinical consequence. But maybe from another vantage point, or from a heart failure hospitalization, that has very important implications for a patient.

Increasingly as we develop these cardio-kidney-metabolic clinics, the more that we spend time together in the hospital or in the office thinking about these patients, I think the better, the more coordinated the care will be. I think the better the quality of care will be, and I think the better the outcomes that we'll see for our patients.

Dr. Burton:
The other thing just to say with that just when the potassium is high, again, people say, oh, why would we use a binder to continue on

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those therapies when pill burden is high and polypharmacy is a problem, and I say, well, it's around quality of life as well. We're talking about your patient, keeping people out of hospital, keeping them on their goal-directed medical therapy. Stopping those late-night admissions to the emergency department with hyperkalemia is absolutely again the right thing to do to keep their potassium in the normal range.

Dr. Desai:
Yeah, it's such a great point. And, Jim, that sort of reminds me of maybe a case that I might bounce off of you now, also of something that's incredibly common in all of our practices, and that's actually a patient with preserved ejection fraction heart failure. And I think the more we're learning about the epidemiology, that winds up being the more dominant profile and phenotype of heart failure nowadays.

And as we have this kind of rise of other cardio-kidney-metabolic contextual comorbidities, obesity and diabetes and chronic kidney disease, that I suspect that that prevalence of HFpEF will continue to rise.

And so this was of a relatively young patient, in their early 50s, but obese with diabetes type 2 and with chronic kidney disease stage 3. They also had some proteinuria, some albuminuria. I think the UACR was in the 150 to 170 range and was diagnosed with preserved ejection fraction heart failure based on their echo parameters, had some elevated pulmonary pressures and evidence of diastolic dysfunction.

And we tried to manage that patient as, again, I think the evidence would suggest that we should that for now the foundational therapy has been an SGLT2 inhibitor. They've shown to be beneficial, kind of across the full spectrum of heart failure. And then to that, we actually added a nonsteroidal mineralocorticoid receptor antagonist, in this case finerenone, to their background therapy, and that was of course studied in the FINEARTS study as well. And we then kind of confronted a very similar conundrum. One that we've been talking about through most of this educational series is, again, the development of hyperkalemia.

And so, Jim, let me ask you, have you had some experience with the nonsteroidal MRA? What's been kind of your experience with those in your own practice? And would that have changed anything in terms of your approach to the hyperkalemia that developed in this case?

Dr. Burton:
Epidemiological data would tell us that actually that 5 to 5.5 range is still worthy of consideration actually. And we have to be mindful of the fact that for years we've been telling people that potassium above 5 is worthy of caution. And guidelines tell us that we don't start particular therapies like MRAs if the potassium is above 5.

We use finerenone a lot. It's been shown to reduce the progression of albuminuric CKD in people with type 2 diabetes, and I've been using it for a number of years in my practice. And I have the same approach to initiating finerenone as I would with any medication that can impact on creatinine and potassium. And KDIGO would say to measure creatinine and potassium within 2 to 4 weeks, and I would certainly be adhering to that, and closer to 2 weeks if it was someone who was particularly at risk of hyperkalemia, where it was in the higher end of that range or they had heart failure concurrently or on other medications.

Dr. Desai:
And just like the discussion we've been having on the HFrEF side and thinking about when and how to use modern potassium binders, how to select between the 2, I think that that discussion and all the themes that we've touched on are very relevant for the HFpEF patient as well.

Dr. Burton:
And look, to round things off, there's something that we haven't necessarily touched on, and I wanted to sort of bring up a case of the hospitalized patient. So we've talked about people who've been admitted with hyperkalemia or they've had their core therapies discontinued or down titrated that's led to a hospitalization. But I'm going to talk to you about a patient that I saw.

So a 66-year-old woman, type 2 diabetes, HFpEF as it happens, and stage 3b CKD. So her GFR was estimated around 35 mL/min, and she was on losartan 100 mg, a beta-blocker, an SGLT2 inhibitor, and a loop diuretic. And she came into hospital with diarrhea, an acute kidney injury, with a potassium of 6.4. And again, I imagine we've both seen that scenario where someone comes in acutely unwell, and because of hyperkalemia, which is treated in the usual way in that emergency environment, but at the same time had some of her core

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therapies discontinued. So in that case, the losartan was completely withheld, and I guess if it had reduced ejection fraction, similarly the MRA or the ARNI might well have been discontinued for a short period of time. And in that point, when I saw her in clinic later, I could see that when she was discharged from hospital, her potassium was 4.8, and everybody was suitably relaxed about that, but she hadn't had her therapies restarted, and that's the key. There was no documented plan to restart, and no one had reached for a potassium binder to facilitate that while that patient was in hospital. And so, I guess, like many patients, that temporary discontinuation became permanent until I saw them. And I think that's a real point of vulnerability for the people that we look after with CKD and diabetes, and for you with heart failure, right?

So I guess my question is, what do we need to do slightly differently about that? Because as soon as someone's come in with hyperkalemia, we know that they're at risk of coming back, and we know that their risk of hospitalization and other things is higher in that case. So what should we do differently? What would you do in that situation if you were on rounds before someone was discharged?

Dr. Desai:
Yeah, Jim, it's such an important point, and I think that despite the fact that we all have these electronic health record systems, despite the fact that the ability to communicate with each other is seemingly much easier and simpler, that it seems to be harder and harder, and unfortunately, there's too many patients where there's a temporary plan that one provider has or something in their mind, but it never gets communicated. The follow-up never happens, and unfortunately, for too many of our patients, they wind up on low, intermediate, or no dose of important background therapies and, hey, we don't want to step on the toes of our colleagues in primary care or in endocrinology, but we've got to think about these therapies as disease-modifying therapies. These are cardio-renal-metabolic therapies. They have very important benefits for our patients.

But then there's got to be a very deliberate plan, and even if it's not all reintroduced during the hospital course, that there is a very close outpatient plan to say, hey, at that outpatient visit, if things are going okay, our plan would be to make sure that you get back on your MRA or your ARB, whatever the case may be, and that the patients are then kind of fully engaged in that discussion and have line of sight for it as well.

Dr. Burton:
Yeah. So we started off this session by talking about the multi-professional team. I think that sits very well within that. And again, with the patient in the center, you've just said, they're part of that decision-making ultimately because they're going to be taking those medications and adhering to those medications that are going to change their outcomes, and I think getting everybody involved with that and not feeling, like you say, that you're stepping on toes, but actually, just we're just facilitating and helping each other because we don't want to normalize one blood result; we just want to prevent recurrence and restore therapies that protect the heart and kidneys.

So look, we've probably gone on for long enough. Thank you so much, Nihar, for those cases and those insights. That's all the time we have today, and thanks to everybody for joining us.

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