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Mastering MRA Monitoring: The Key to Success

Announcer:
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Dr. McMurray:
This is CME on ReachMD, and I'm Dr. John McMurray.

Dr. Desai:
Yes, great, John. It's great to be with you. I'm Akshay Desai.

Dr. McMurray:
So, Akshay, we've recently discussed the results of the FINEARTS-HF trial, but of course, what we really want to do is to understand how to use the effective treatment demonstrated in that trial, finerenone, in clinical practice. So I think you've got a real-life case for us to discuss today.

Dr. Desai:
So, John, let me pose to you a patient who's 57. She has a history of hypertension, of type 2 diabetes, and obesity. Her body mass index in the clinic was measured at 32 kg/m^2. She is seen after a recent hospitalization for worsening heart failure, and during the admission she had an echocardiogram that showed that she had an ejection fraction estimated at 45% with some left ventricular hypertrophy, presumably related to her history of hypertension, and she had a coronary ischemic evaluation with a stress perfusion study that suggested no ischemia.

In clinic, she says she's still a little short of breath on climbing stairs but feels much better than when she went into the hospital and is comfortable walking on flat ground and had no difficulty getting into the clinic. Her current medicines include furosemide 40 mg once daily, empagliflozin 10 mg once daily, metformin 1,000 mg twice daily, irbesartan 150 mg daily, and amlodipine 10 mg daily.

On examination, her blood pressure is 140 over 80mm of mercury and her heart rate was 70 and regular. There was no apparent jugular venous distension. Her lungs were clear, there was no murmur or gallop, and her liver was not enlarged. There was some mild pitting edema of the lower extremities, but nothing significant. And her laboratory profile from the last measurement on discharge showed a potassium of 4.8, a creatinine of 1.7 mg/dL, which translates to an eGFR of 31 mL/min/1.73 m^2. And a urinary albumin-to-creatinine ratio of 30 mg/g. Her hemoglobin A1c was 7.7%.

Dr. McMurray:
So does this patient need more treatment? Absolutely. This patient is at very high risk of 2 types of events. One is an adverse cardiovascular outcome, and the other is, in fact, end-stage kidney disease because the patient that you just described is a patient who would have fitted in perfectly to 2 recent trials.

One that we talked about, FINEARTS, this patient would definitely have been eligible for FINEARTS and would have also been eligible for either of 2 earlier trials with finerenone in patients with type 2 diabetes and chronic kidney disease. That was the FIDELIO and the FIGARO trials. So no question this patient needs more treatment.

Patient could do with some other medication as well, so obviously the 3 trials I just mentioned used finerenone. Hopefully that will lower the patient's blood pressure a bit because I think 140 is a bit too high for a patient like that with the profile you've just described. Patient's hemoglobin A1c is not terribly well controlled. There are other glucose-lowering therapies that also reduce cardiovascular and, we now know, renal risk and also help treat obesity. So a GLP-1 receptor agonist would be another treatment I would be thinking about in this patient. So finerenone, GLP-1 receptor agonist. And if I didn't achieve better blood pressure control with those 2 treatments, I would consider additional therapy. But let's start with those 2.

Dr. Desai:
We often think about cardiovascular disease in isolation, but I think we see now the overlap with diabetes, with chronic kidney disease. And here with the data from FIDELIO, FIGARO, and now FINEARTS, we're seeing a therapy that has some efficacy across cardiometabolic spectrum.

Dr. McMurray:
We're used to thinking about cardiovascular outcomes because that's where we've had treatments that are beneficial, but it's only recently now that we've really been able to do much for kidney outcomes. Until the SGLT2 inhibitors, finerenone, and GLP-1 receptor agonists, we only had angiotensin receptor blockers and ACE inhibitors. And for many, many years, that was the only treatment to improve renal outcomes. Now we've got many more. And I think the important takeaway currently is that we now have so many more treatments than we used to have and that we now can achieve so many more goals therapeutically than we could have done in the recent past.

Dr. Desai:
One of the issues that I think always comes up when we think about applying more medical therapy is the balance of efficacy and safety. And I think we've talked a lot in the last few minutes about how effective these drugs are in isolation and in combination. And I think it's important to emphasize, as you've done in the past, that the effects seem to be additive largely of these drugs, not substitutive. So it's really the whole regimen that brings benefit to the patient.

So how do we think about use of MRAs now in this advanced CKD population with proteinuria? How carefully do we need to monitor patients? Does hyperkalemia happen less with finerenone than with spironolactone?

Dr. McMurray:
Well, we do have to monitor patients because hyperkalemia, although infrequent, does occur. So in a small percentage, just 2% or 3% of patients in FINEARTS, we did see serious hyperkalemia. There were no fatal cases of hyperkalemia. There were very few cases leading to hospital admission.

But we've always got to remember the benefits relative to the risk, and there is no question that in the 3 trials that we've talked about – FINEARTS, FIDELIO, and FIGARO – the different populations that we've talked about, the balance of benefit was clearly favorable relative to risk.

In terms of safety of spironolactone versus finerenone, I think that's difficult to make that comparison. I think the important message is serious hyperkalemia is very uncommon. And just to finish with, maybe we should also remember that hypokalemia is a problem in these patients. In fact, hypokalemia is more common than serious hyperkalemia. And hypokalemia also carries risks, particularly in patients with heart failure, I would say. And we know that MRAs substantially reduce that risk of hypokalemia.

Dr. Desai:
Yeah, I think that's a critical point, John, because I think it's often forgotten.

Dr. McMurray:
And we've also got a great synergy that maybe we should speak about, because at least when we did our analysis of the SGLT2 inhibitor trials, we found that they seem to reduce the risk of hyperkalemia in patients getting concomitant MRA therapy. So there is a synergy, not just in terms of that added efficacy that you talked about, but also in terms of safety.

Dr. Desai:
Well, I think our time is almost up. Do you want to speculate at all, John, about guidelines and how those might evolve in the light of these data?

Dr. McMurray:
I think we can speculate. I mean, certainly, FINEARTS is such a large and convincing trial that it is going to change guidelines. I think the debate will be about the class and level of evidence. So it is one large trial, but there is a meta-analysis with the other MRA trials. And of course, there is the additional experience in adjacent populations using finerenone in people with type 2 diabetes and chronic kidney disease. So collectively, does that give you enough evidence to give a level A in terms of class of recommendation? Well, the SGLT2 inhibitors got a class 1 recommendation for reducing the same composite endpoint as we saw reduced with finerenone. And so perhaps we could anticipate a similar class recommendation.

Dr. Desai:
Great. Well, I think that closes our discussion for today. It's been a pleasure joining you for this conversation.

Dr. McMurray:
My pleasure as well to speak to you.

Announcer:
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