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Released: 09/24/2025
Valid until: 09/24/2026
Time needed to complete: 1h 02m

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Guidelines Update: RAASi/MRA Therapy in CKD and HF Management

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Dr. Kelepouris:
Hello. This is CME on ReachMD, and I'm Dr. Ellie Kelepouris. I'm here with my colleague, Dr. Javed Butler. Welcome, Javed. Javed, can you share with us what are the current guideline recommendations for RAAS inhibition and MRA therapy for patients with CKD and heart failure?

Dr. Butler:
Not only is there a critical role for RAAS inhibitors and MRA in patients with CKD and heart failure, but actually, this role is evolving, and there are more and more indications coming out as we do more clinical trials, and it all sort of goes to the role of angiotensin II and aldosterone in development and progression of cardio-kidney disease.

So if you look at heart failure with reduced ejection fraction, there's a class I recommendation, in the absence of contraindication, to give RAAS inhibitors—ACE inhibitor, ARB, ARNI, and MRA therapy.

If you look at the KDIGO guidelines, again, non-steroidal MRAs and RAAS inhibitors are recommended for patients with chronic kidney disease as well.

So not only are they recommended to be used in patients with CKD and heart failure, their use is expanding as we do more clinical trials. The problem that comes up is that as our patients get sicker—which, in the world of heart failure, will be more advanced heart failure; in the world of CKD, lower eGFR—those are the patients that are at the highest risk for poor outcomes, and by virtue of that, need therapy the most, but they are at higher risk of not being able to tolerate the medications for side effects, things like hyperkalemia, that makes it a little bit tough to manage. But at the end of the day, these drugs are widely recommended across the spectrum of cardio-kidney disease.

Dr. Kelepouris:
Thank you. I can also support that statement by looking at the CKD literature, and inhibitors of the RAAS (renin–angiotensin–aldosterone) system are a critical part of clinical practice guidelines for managing patients with all stages of chronic kidney disease.

I think one of the issues that we see when we take care of patients with this cardio-kidney syndrome is that although the recommendations are to use them as first-line in advanced CKD in patients with uACRs greater than 300 with or without diabetes, we

see that their hemodynamic effect to raise the serum creatinine, and possibly also their hyperkalemia effect in CKD stage 4, makes physicians a little nervous about using them. And the doses tend to be down-titrated with really suboptimal results in terms of cardiovascular protection and renal protection as well.

So this is a very important point to make to our colleagues, that reducing or down-titrating the dose does have consequences.

What are your thoughts about that?

Dr. Butler:
Yeah, I would suggest to our audiences to see this paper, which was recently published in Journal of Cardiac Failure just last month by one of my colleagues from Duke University, Dr. Steve Greene, CARE-HK registry and subsequent abstracts and papers. Really, this is real time now, right? I mean, these are not some historical concerns, that clinicians do down-titrate the medications. Which again, makes sense, right? I mean, we are sort of in that mindset of "first, do no harm."

The problem is that when we down-titrate medications because of, say, risk of hyperkalemia, you're basically exchanging a long-term problem and a short-term problem, right? So, yes, you're now reducing the risk of hyperkalemia today, but you're increasing the risk of progression of the disease in the future and its consequences. And the question is, how can we better manage hyperkalemia as opposed to lowering the doses or stopping the drugs altogether?

Dr. Kelepouris:
And in fact, this is a real problem because 1 in 3 patients in that study—really only 1 in 3 patients received optimal therapies with renin–angiotensin system use and MRA use. The rest of the patients really were down-titrated.

And so this is something that's really an important consideration. So thanks for highlighting that study.

Dr. Butler:
Great.

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