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A multidisciplinary clinical dialogue: Optimizing long-term goals: practical approaches to novel potassium binder use in cardiorenal care

Dr. Wong:
Hi. I am Aaron Wong, one of the cardiologists from South Wales from the United Kingdom.

Dr. Burton:
And hi, my name's James Burton. I'm a nephrologist and professor of renal medicine at Leicester in the United Kingdom as well.

Dr. Wong:
So it's a great honor to be here today and discussing with Jim about how we can optimize the long-term goals and share some practical approaches to novel potassium binders in patient with cardio and renal problems.

So these were our disclosure.

So, I'm going to start out with the case Jim. I have a 77-year-old man who presented to our hospital with a typical heart and kidney failure problem. Many decompensated heart failure with a very low ejection fraction. You can see from the chest X-ray here it shows venous congestion, and the patient's kidney function show an eGFR of 40 and a potassium of 5.5 and a very raised NT-proBNP, as expected. So the patient came in with this list of medication, which includes medication for diabetes, for atrial fibrillation, and hypertension. So as a cardiologist, these were the options that's going though my mind when we see this patient.

So can I ask you a specific question with regards to congestion? Should we, as a cardiologist, worry about the potassium or the kidney function? Because clearly, we want to decongest the patient. What is your thoughts?

Dr. Burton:
So I guess this is the sort of person that we would see in our everyday practice, right. Somebody with cardiorenal metabolic syndrome, significantly impaired kidney function, raised BNP, maybe with or without diabetes, and I guess the key is that it's congestion that's the killer. And I think we worry a lot that we're over-diuresing people, push that creatinine up. We see a fall in their GFR, and we worry about that. But actually, we can see from the data that congestion and worsening renal function, of course, that's bad, but congestion without worsening renal function is a worse situation to be in than someone who is off-loaded even if their creatinine is going up. So don't be afraid to increase the dose of the diuretic irrespective of creatinine and get that congestion away.

Dr. Wong:
Yeah. I think as a cardiologist, sometimes I see patients in the heart failure clinic that were not adequately decongested. So, that's why they keep coming back to the hospital. I think you highlighted a very important point about not to worry too much about the transient worsening renal function in the context of decompensated heart failure.

So back to the case. Then we discharged the patient, the patient was adequately decongested, and the patient was started on the standard guideline-directed heart failure medication. And we were very cautious about the MRA because of the potassium issue. And this patient actually came back a bit earlier than expected because he was feeling generally unwell. I think we over-diuresed the patient. As you can see here, the creatinine has gone up to 200, and the urea has gone up as well. So the loop diuretics were discontinued. And

so in this context, patients with heart failure, we often see worsening renal function. So we should be worried about RAASi therapy in this context.

Dr. Burton:
Or should we? That's the question, isn't it?

Dr. Wong:
That's right.

Dr. Burton:
So we talked about off-loading congestion is the right thing to do. Sometimes we don't get that right. People's clinical condition changes; we've all seen that. But is discontinuing the RAASi therapy the right thing to do? Aaron, I've seen you present on this many times, and you give a really great analogy to oncology and chemotherapy and side effects from that, right?

Dr. Wong:
That's right. That's right. I think patients with severe heart failure, the prognosis is worse than a lot of common cancer. So I think we should try our best, try to preserve and maintain disease-modifying drugs in these patients.

Dr. Burton:
And that's the key. These are disease-modifying medications like chemotherapy medications. We don't discontinue chemotherapy because someone feels nauseated; we manage the side effects and continue the disease-modifying medications, and this is the same. We shouldn't discontinue for something like hyperkalemia, which we know can be modified in other ways.

Dr. Wong:
Yeah. That's right. That's right. And the data already is very clear, is to show that the RAASi dose reduction is just as harmful as stopping the RAASi altogether. So you almost double up the adverse events in this group of patients.

So recently, I came across a STOP-ACE trial. Can you just briefly talk through that?

Dr. Burton:
Yeah. So I guess this was a key study looking at that question about whether we should stop the RAASi therapy if the GFR is below 30 mL/min because you know we get back a little bit of kidney function because it's a reversible decline in GFR. And I think what we saw from the data was, from a kidney point of view, it doesn't make any real clinical difference whether we continue or discontinue the ACE inhibitor from a kidney progression point of view, but there are many other reasons why we might be on a RAASi therapy, including people with heart failure. So I guess what this tells us is there's no harm from a kidney point of view in continuing the ACE when the GFR drops, but there could be really clear benefit from a heart failure point of view or other reasons why they might be on an ACE inhibitor.

Dr. Wong:
Okay. Thank you. So it's very nice and clear that, I think, in the context of CKD, we should maintain RAASi as much as possible.

So let's go back to the case. We saw this patient again in our clinic. The BNP has risen a little bit more. The patient remains very symptomatic, and now the kidney function has improved since we cut back the loop diuretics. Well, now we get another problem. It's a potassium issue. The potassium is 5.3, and the patient is not yet on the optimal medical therapy for heart failure. So you can see throughout, in many, many months, the patient's kidney function is kind of around with the GFR just between 30 to 40, and the potassium being a recurrent problem.

So in this context, what would you do with regards to thinking about a potassium binders in this context?

Dr. Burton:
Well, I guess we know that kidney function is going to improve. People with heart failure, 40% have impaired GFR, we know their kidney function is not going to improve, so there's no point waiting. And actually, we also know the longer you wait, the more likely they are to have poor clinical outcomes, hospitalization for heart failure, so the key is to get a therapy started as soon as we can. And a novel potassium binder is one of those therapies that's recommended in guidelines in the UK and internationally to facilitate maximal goal-directed medical therapy for people with cardiorenal disease.

But one of the questions that I get asked a lot is how long should we continue with a novel potassium binder once we've started it? So, I mean, what do you do in your clinical practice in that situation, Aaron?

Dr. Wong:
So in the patient, as you said, this potassium and the renal function is unlikely going to improve, and hyperkalemia is a recurrent

problem that needs a long-term solution. So from clinical trials, we show that once these binders were stopped, there was a rebound hyperkalemia. Just to highlight that this patient needs a long-term solution. So in my clinical practice, the majority of my patients would maintain on the binders as well as optimize RAASi therapy.

So this is a very interesting to share this study with you about unraveling the relationship between hyperkalemia and RAAS inhibition therapy. And this paper highlighted that hyperkalemia, initially, was thought to be associated with worse outcome. But when adjusting for discontinuation of RAASi therapy, hyperkalemia is no longer the culprit; it's actually the discontinuation of the RAASi therapy. I think that kind of echos what we've been thinking for many, many years that, I think, is the discontinuation of RAASi and hyperkalemia is a risk factor for not prescribing RAASi and optimize a MRA in this situation.

So I know you been involved a lot in the Delphi consensus. Can you just give us some highlights from that paper?

Dr. Burton:
Yeah. I guess the main thing from the Delphi consensus, which was a consensus using the Delphi methodology across Europe and the United States, really highlighting that it's predictable. We know those individuals that are going to be developing hyperkalemia. It's predictable, and it is manageable. And actually, we should be really thinking along the lines of the reduction of RAASi being one of the final parts of the pathway, and using novel potassium binders should really be part of our standard menu of things that we would do to reduce the potassium before we think about reducing RAASi therapies. And the other thing that came from that Delphi consensus piece was that actually it's conversations between cardiologists, nephrologists, and other members of the multiprofessional team that's key to really good outcomes for people with cardiorenal disease, so heart failure and CKD.

Dr. Wong:
Wow. Excellent. So what is your take-home message for the audience who watched this video?

Dr. Burton:
I guess it would be optimize RAASi therapies in our patients with heart failure as well as thinking about decongestion. If potassium is a problem, then there are other ways to manage that other than discontinuing RAASi therapy, and conversations like this about patients that we share are absolutely the best thing that we can do for people with heart failure and CKD.

Dr. Wong:
Yeah, I echo that. Thank you very much, Jim.