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Optimizing clinical care in patients with cardiorenal disease for achieving long-term goals

Dr. Bonanad:
Hello. I'm Clara Bonanad, cardiologist from Spain, and we are here in a multidisciplinary team.

Dr. Burton:
Hi, I'm James Burton. I'm a nephrologist a kidney consultant from Leicester in the UK. It's nice to be here with you.

Dr. Bonanad:
We are going to present a challenging case report that is Julia. She is 76 years old. She has hypertension, type 2 diabetes, CKD stage 3. She had a small ischemic stroke with no sequelae, and she's obese, and she's on Metformin, candesartan with hydrochlorothiazide, and aspirin. She goes to her primary care physician due to a recent event of palpitation and a slight shortness of breath while walking. And she is certainly referred to cardiology. At the cardiology outpatient clinic, she was diagnosed with new onset atrial fibrillation. The ejection fraction was 47%. And cardiologist added furosemide, bisoprolol, and apixaban, and removed aspirin; 72 hours later she began to experience sudden dyspnea and she went to the emergency department.

At this moment James would you have done anything differently with Julia?

Dr. Burton:
So I think at this point in time, from guidelines and evidence, we know that for people with type 2 diabetes and CKD as measured by abnormal kidney function, that they would benefit from being on an SGLT-2 inhibitor. And in fact, even if she didn't have diabetes we know with impaired kidney function people almost certainly should be, if they have high cardiovascular risk, should be on an SGLT-2 inhibitor.

But here's my plug to make sure we're measuring ACR levels so that not only are we sure that they should be on an SGLT-2 inhibitor, but also whether they might benefit from something like finerenone.

Dr. Bonanad:
Thank you. I agree with you. So Julia was admitted to the cardiology department for decompensated heart failure in the context of respiratory infection and the emergency echocardiogram shows an ejection fraction of 42%. The analysis, the creatinine and glomerular filtration rate was similar to previous. Electrolytes were normal, and NT-proBNP was very high. So we performed also a stress CMR and we confirmed that the left ventricular ejection fraction was 42% with no ischemic etiology.

So at this moment, Julia was diagnosed of having cardiorenal syndrome. We know that risk factors for developing cardiorenal syndrome are diabetes, obesity, metabolic syndrome, hypertension, and another physiopathological ways that sympathetic neurohormonal activation, inflammation, endothelial dysfunction, fibrosis, and oxidative stress, all that produces organ damage and activates RAASi pathway and also profibrotic pathways that affect the kidneys and the heart.

So at the start, we only add up-titrate bisoprolol to 2.5 mg per day, and we up-titrate furosemide to 100 mg per day, but we didn't do anything with candesartan and the other medications.

So James, what do you think about that treatment?

Dr. Burton:
So again, I think you highlighted something really important, which is about that cardiorenal axis. And are we thinking from both lenses. What can we do to optimize the treatment from a cardiac and a renal perspective? You highlighted around the ACE inhibitor. We know that that is a disease-modifying therapy not just an antihypertensive. It's good for people with proteinuric renal disease, as well as being good for cardiac protection and, of course, for hypertension, but also that opportunity to start someone on a SGLT-2 inhibitor. And I think now we will probably all be doing that in any case. But I think this just highlights the importance of taking those opportunities as a multiprofessional team to make sure that we optimize therapy from more than one angle, whether that's heart failure or kidney disease or diabetes.

Dr. Bonanad:
I agree with you. I think we lost an opportunity in these admissions. So 6 months later, Julia was admitted for decompensated heart failure and hyperkalemia. Hyperkalemia was treated with standard care at the emergency department and potassium binders, and she was admitted to the cardiology department. She has a worsening heart failure, and the potassium was 6.7. After Julia improves in her heart failure decompensation, we discharged Julia with potassium binders and we up-titrate furosemide and we add curiously, hydralazine plus nitrates. We referred Julia at the cardiac renal unit with an analysis in 1 week.

So what do you think, or would you have done anything differently? And what do you – why hydralazine plus nitrates?

Dr. Burton:
So I can understand the rationale behind that, which is to reduce or stop the ACE inhibitor or the ARB, thinking that that would reduce the risk of recurrent hyperkalemia. And I understand that. But actually, we know from observational data that that increases the risk of cardiovascular events, and it doesn't necessarily reduce the risk of renal events.We know that from the STOP-ACE trial. So actually, what we should be doing is optimizing their cardiorenal care with an ACE inhibitor, knowing that that will reduce the risk of cardiovascular events, and managing the hyperkalemia in a different way.

Dr. Bonanad:
I agree with you again, and I think we lost another opportunity. But at the cardiorenal unit they added sacubitril valsartan, SGLT-2 inhibitor, they up-titrated bisoprolol, and they make an appointment in 2 weeks with an analysis to try to think in starting MRA in this patient. So we know as hyperkalemia associated risk of hospitalization is increased in cardiorenal patients, 3.8 higher in CKD patient and 2.8 higher in heart failure patients.

Dr. Burton:
And I think what you've shown really nicely there is that once you got to that multiprofessional meeting, that there was a much more robust way of optimizing treatments from both sides. They were on an SGLT-2 inhibitor, they were on an ARNI, which includes an angiotensin receptor blocker, you're thinking about an MRA, and we're thinking much more cohesively about that care from not just a cardiovascular point of view, but also a renal angle. And that's been highlighted more recently in many guidelines, including KDIGO, just about the importance of stewardship and thinking about that multiprofessional approach, not just as doctors, but also other members of the multiprofessional team, like pharmacists.

Dr. Bonanad:
Thank you very much, Jim, for your and your opinion as a nephrologist.

I wanted to mention the STRONG-HF, that is a multinational, open-label, randomized trial. And this study, mentioned in the 2023 Heart Failure Guidelines, showed a greater reduction in cardiovascular event in the arm of a rapid optimization of these four foundational therapies within 6 weeks. So we have to do it, and we have to use tools such as potassium binder to do it. And the main reasons to do not optimize treatment are renal dysfunctional hyperkalemia that were known as the major causes to further reduce RAASi, particularly MRA. So these guidelines highlight that new potassium binders potentially could make us enable RAASi initiation and up-titration with a potassium level more than 5 mEq/L. So in American guidelines for the first time, give a class 2b recommendation of these drugs.

So I wanted to mention this study that is very recently, the ZORA study, is a comparative observational cohort study with hospital records and administrative databases for U.S., Japan, and Spain to assess the likelihood of maintaining treatment with RAASi 6 months after an hyperkalemia episode in two cohort; one of patients with new potassium binder for at least 120 days versus those without a prescription of a potassium binder and they were followed up at 6 months. So patients who received the potassium binder had at least 2 to 2.5 times the likelihood of maintaining RAASi treatment compared to those that did not receive a potassium binder. And achieving goals is cost effective. And these two articles demonstrated one with sodium zirconium cyclosilicate in Norway and Sweden, and the other one in Spain with patiromer. And why we have to optimize the foundational therapy is because the effect of these four medication, reduced mortality by 73% in 2 years, and prolonged survival for 6 years. And we have the fear of the risk of hyperkalemia in certain

situations, but hyperkalemia was an unmet need that is now resolved in cardiorenal patients with the new potassium binders.

And as key messages I think we have to avoid to de-escalation of RAASi or discontinuation of this therapy. We have to use novel potassium binders, and we have to work in multidisciplinary teams. And to conclude my presentation, I wanted to say that coming together is the beginning, keeping together is progress, but working together is the success.

Thank you very much.