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# Breaking Silos With STAMP! Detecting Cardiorenal Risk and Integrating Nonsteroidal MRA Therapy in Patients With HF and CKD

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**Dr. Morales:**
This is CE on GLC. Today, we'll be speaking about heart failure and chronic kidney disease often traveling together, but care doesn't always follow. When cardiorenal risk emerges, who recognizes it? Who acts? And who owns the next step? So today, we'll be using the practical STAMP approach to follow one patient across the journey of primary care, cardiology, and nephrology from early detection with the GFR and UACR to coordinated heart and kidney protection.

My name is Dr. Javier Morales, and I am a practicing internist that's based out of Long Island, New York, and very involved in cardio-kidney-metabolic health, as well as continuing education. I am privileged to be joined by 2 experts from across the pond, Professor Nikolaus Marx and Professor Brendon Neuen.

So would you please identify yourselves and tell us a little bit about your practices and where you're from. We'll start with Dr. Marx.

**Dr. Marx:**
Yes, I'm Niko Marx. I'm a cardiologist at the University of Aachen in Germany, heading the cardiology department, and I have a longstanding clinical and research interest in cardiovascular disease and comorbidities, especially diabetes and kidney disease, and I've been involved in various guidelines from the European Society of Cardiology.

**Dr. Morales:**
And Dr. Neuen?

**Dr. Neuen:**
Well, thanks very much, Javier and Nikolaus. Great to join you both. My name is Brendon Neuen. I'm an associate professor of medicine, and I'm a nephrologist at Royal North Shore Hospital in Sydney, Australia, and I'm the program lead for renal medicine at the George Institute for Global Health. I have a particular interest in the intersection between kidney disease and cardiovascular risk and how we can improve outcomes for patients with CKD by addressing both CKD and heart failure risk simultaneously.

**Dr. Morales:**
So let's start with a patient that we all typically see in clinical practice, and this is the type 2 diabetic patient who suffers with obesity, hypertension, worsening shortness of breath, and edema, declining in their eGFR with no recent UACR.

So, Niko, what catches your attention first, and what needs to happen now?


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**Dr. Marx:**
Well, thank you. First of all, it's important to recognize that this patient is at risk for heart failure. Hypertension, diabetes, CKD, obesity all are drivers and put the patient at risk for heart failure. If I see this patient, I have the suspicion that heart failure could be there, and this patient needs a particular cardiology workup, so I would start with an NT-proBNP to get an idea, but then—which is probably elevated; keep in mind, in those with obesity it may be low.

This patient deserves an echocardiogram to see whether he or she has heart failure, and I would assess LV function. I would assess the valves. I would, of course, do an ECG to see whether the patient has atrial fibrillation, for example. But given the symptoms and the high risk, this is something that would be my first differential diagnosis.

And it's important to mention, as cardiologists, to be honest, we never cared about the kidney as long as we didn't send the patient to the cath lab. But we need to recognize that CKD is not only a driver for cardiovascular risk, including heart failure, but also has an impact on the prognosis. And I think this is something that we as cardiologists learned and have to recognize and translate into our daily practice.

**Dr. Morales:**
So, Dr. Neuen, give us a little bit of a nuance about how this particular patient plays in your practice and how you would screen for this particular patient.

**Dr. Neuen:**
Yeah, I agree completely with what Niko said. These patients are often—if they don't have heart failure, established heart failure, they're certainly at high risk of it. And the other important point to note is that combination of reduced kidney function and heart failure signals at a heightened risk not only of kidney outcomes but also adverse cardiovascular outcomes. So this is a high-risk individual if they are losing kidney function and have abnormal GFR.

The next thing that's important to note is that from our perspective, what is really critical in this scenario is an assessment of their kidney risk, and it's important that GFR only gives you a part of the picture.

What we really want to know is what degree of albuminuria do they have? Do they have no albuminuria, mildly increased albuminuria, or moderately increased albuminuria? And that's important because it tells us lots of things. It tells us what degree of risk they have. It informs our treatment options, and increasingly, it's a treatment response biomarker that we can use to guide the use of foundational and other guideline-directed therapies for kidney and cardiovascular disease.

At this stage, it's also really important to establish the cause of kidney disease. Now, most of these patients will have kidney disease related to cardio-kidney-metabolic syndrome, but it's important to not forget there are other treatable causes of kidney disease. So assessing for microscopic hematuria, getting a renal tract ultrasound is important in all of these individuals.

**Dr. Morales:**
You know, and for me, from a primary care standpoint, and again, just to circle back on this discussion that we're having with you is, well, for the most part, this is a pretty sick patient. This particular patient class that we identified has a myriad of things that are going on stemming from obesity, from their diabetes and their overall volume status, and all of this is having significant end organ impact.

So for us in primary care, I think it's important to ascertain where the problem with a defect actually might lie. So with all due respect, gentlemen, what I want to do is—we had this beautiful meeting recently, the European Society of Cardiology over at Munich, Germany, and they actually have launched a new campaign called this STAMP on CKD campaign.

So it's important to first recognize that CKD may be existing, but as you just mentioned a little while ago, it's about triage as well. And there are significant screening gaps that actually exist as well as addressing some of these modifiable risk factors that could lead to end organ progression.

So, Professor Marx, I'm going to come back to you for a second. For the most part, if the GFR is declining but we don't have a recent UACR for the patient, then what are we missing, and how does this get that information changed? And what happens next?

**Dr. Marx:**
Well, it's so kind of you to ask the cardiologist, but since we have a nephrologist on board, Brandon, I hand over this question to you and maybe give you the cardiologist perspective at the end.

**Dr. Neuen:**
Yeah. So it is just so critical. I think the first thing to establish is chronicity, Javier. Chronic kidney disease means that we need to make sure it's not acute, and typically that means that the GFR is sustained and low for greater than 3 months, and also that albuminuria is


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sustained.

Now, albuminuria—if there's one thing clinicians remember from this particular series and this conversation is that albuminuria is woefully undertested and tells us so much about the risk of these individuals. It tells us about their risk of kidney failure. It tells us about what treatments we should offer them.

It tells us the response to treatments in terms of kidney prognosis, and as Niko will talk about, it tells us about cardiovascular risk. And that's important because for the most part, most of these individuals will experience a cardiovascular event or die due to heart disease before they reach dialysis.

Now, what we need to do is recognize that albuminuria tells us—and we can categorize it into 3 categories, A1, A2, and A3, representing what used to be called normo, micro, and macroalbuminuria, and that tells us about the risk of kidney disease progression and other complications. And we should measure that at least annually in people with diabetes, like this particular case, but also in people with established heart failure or hypertension, obesity, and other risk factors.

So what that means for the practicing general practitioner or cardiovascular clinician is that essentially everyone who you are seeing should be screened with albuminuria.

**Dr. Morales:**
So as one of the messaging that I've been giving to some of my colleagues from the primary care sector is that the GFR is really a measure of kidney function, whereas a UACR is a measure of kidney damage. And the impact of proteinuria on cardiovascular health was never as well defined as it was by the cardiology community historically.

So, Dr. Marx, give us your take on this UACR.

**Dr. Marx:**
Well, I think, and as Brendon and you both mentioned, let's take a patient that we've seen in the cath lab this morning, eGFR 55. Nobody cares. But if he has an A3 UACR, high albuminuria, that means he's not only at risk for kidney failure but also for heart failure, cardiovascular death, and other cardiovascular manifestations.

And this is something that we learn as cardiologists but might be different in primary care and of course in nephrology, but measuring it needs strong forces to implement it, but it really helps for the sake of the better for the patient. I think this is important.

**Dr. Neuen:**
And often by the time patients get to see nephrologists, they've lost a substantial amount of kidney function, and those opportunities to modify prognosis, really, those opportunities come to cardiologists and primary care. So that's really where some of the biggest gains can be made in improving kidney health, but also cardiovascular prognosis by implementing those therapies.

**Dr. Morales:**
So what's really interesting is Dr. Marx had mentioned for the most part that you could have an abnormal UACR with a normal glomerular filtration rate. So it's a very important parameter that flies under the radar that really no one is paying attention to or at least should be paying attention to.

And I do get it because sometimes patients are a little bit squeamish about surrendering a urine sample in the office, but it's probably the easiest barometer that we have with respect to heart failure because it's just a spot urine that's collected right there, and you could submit it for quantitative analysis in the laboratory.

**Dr. Neuen:**
Moving on to talk about this case, Niko, we have identified you've got the echo back, you've got someone perhaps with heart failure with preserved ejection fraction and high NT-proBNP. They might have seen me or you've ordered a UACR and they've got a moderate chronic kidney disease.

We've spoken about how this individual is a high-risk patient who needs a number of therapies to modify their prognosis. Can you talk us through how you would implement a treatment plan for someone like this? Because I often feel like this is an area where nephrology and heart failure cardiologists, sometimes there's a tension in terms of the speed and sequencing of therapy.

So value your perspective on this.

**Dr. Marx:**
So for this given patient, let's assume he or she has HFpEF. It's important to implement therapies that have been shown to improve the prognosis. And if we go 10 years ago, we didn't have such therapy. We gave them a diuretic and full stop. Now, I think it's clear this


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patient benefits from an SGLT2 inhibitor, both for the kidney as well as for the heart, and the same holds true for the nonsteroidal MRA finerenone.

We have very good data from FINEARTS, and this is in addition for HFpEF. In addition, this patient has diabetes and CKD, and we have data from FIGARO and FIDELIO demonstrating not only a cardiovascular but a benefit with respect to progression of kidney disease, and I think this goes hand in hand.

If this patient had HFrEF, the nephrologist is sometimes more important because certain therapies that we have for HFrEF, for example, ARNIs, are not approved if the eGFR is less than 30, and here we need the nephrologist to help us, or we discuss this. I can speak about our department here. We, 12 years ago, established a cardiorenal unit, so cardiologists, nephrologists working on the same ward, seeing the patients.

And I can tell you, we learned a lot from your colleagues, Brendon. Initially we didn't give ARNI if the eGFR was 25. Now we learned, okay, be careful, look at the potassium, but give it because the patient—it's not studied in a trial, but the patient benefits from it. And I think this is very, very important to not only focus, let's say, on the heart here or on the kidney, but make sure that you interact and learn from each other.

**Dr. Neuen:**
The relationship between these 2 organs is bidirectional. So if you protect the heart, you end up preserving kidney function and vice versa. If you institute kidney protective therapies, you also reduce heart failure risk.

So the epidemiological is bidirectional, and the treatments are also now converging as well. So we have similar treatments for both heart failure and kidney disease.

**Dr. Morales:**
From a primary care standpoint, I think it is important to recognize the fact that heart failure with preserved ejection fraction is probably far more common and under recognized compared to heart failure with reduced ejection fraction. So we need to start paying attention a little bit to some of these patients that might be coming in with minimal edema perhaps, or maybe no edema whatsoever, but exertional dyspnea and coughing and so forth.

That being the case, we've talked about using barometers and particularly the UACR to help to predict whether or not there's any existing kidney damage. But there's also other parameters and barometers that we could use to help us along the journey to identify heart failure with preserved ejection fraction.

I think, Niko, you can talk a little bit about echocardiograms and how that might be helpful.

**Dr. Marx:**
Yeah, I think it's very important. The first screening tool in primary care, for example, if you don't have an echo available, is the NT-proBNP. Keep in mind it might be low, false low in someone with obesity, but it gives you a first hint. But then echo needs to be performed because you need, in particular for HFpEF, very kind of sophisticated parameters to make the diagnosis.

There are certain scores, and just assessing LV function, showing that LV function is not reduced, is not enough. And therefore echo—and if you as a primary care physician send the patient to a cardiologist for an echo, ask in particular for HFpEF. Do the scores, do the measurements because now we have therapies to improve the prognosis.

**Dr. Neuen:**
You bring up an important point. People with kidney disease are underdiagnosed with heart failure, particularly heart failure with preserved ejection fraction. So what I'm hearing from you in terms of requesting the echo is that information is really important, asking the specific question: Are there features of heart failure with preserved ejection fraction?

Is that what you're telling us?

**Dr. Marx:**
Absolutely. You need a complete echo. Ejection fraction, diastolic dysfunction, LA size, E/e′, various very specific cardiology parameters. But at the end, you want a definite answer: Is this compatible with HFpEF, yes or no? And that's important. And in particular, in individuals with advanced CKD, they have hypertrophy and fibrosis.

These are the hallmarks of uremic cardiomyopathy, and the first manifestation is HFpEF. And on the long run, LV function might deteriorate and go down, but that's very important. So screening NT-proBNP but then refer for an echo. And all these patients, they say, "I go to the stairs and everything's fine," and all of a sudden they get shortness of breath, and it might not be obesity, age, or whatever. It might be heart failure. Just think about it.


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Once cardiology and nephrology are both involved, what does good co-management actually look like from your perspective as a primary care physician?

**Dr. Morales:**
Oh, man, that's a very, very loaded question. But I think before I start talking a little bit about that and start tackling what the co-management looks like from the primary care standpoint, I'm just listening to this really eloquent discussion that you gave us, Niko, about echocardiograms and E/e′ ratios and so forth.

But how complicated it is it from a nephrology standpoint to assess what the status of the kidney is? It's pretty simple. Just check the urine albumin-to-creatinine ratio, and that's the simplest parameter that you have in terms of prognostic indication, what it has to offer.

But now that we've established the fact that the kidney affects the heart and the heart affects the kidney, Brendon, you being the nephrologist, so what do we have in the toolbox that helps to preserve the kidney?

**Dr. Neuen:**
Yeah, absolutely. And the real advantage we have now is that there is a convergence of treatments for both heart failure and kidney disease. It makes life simpler from an implementation perspective. We've heard about these therapies from Niko, SGLT2 inhibitors, and nonsteroidal MRAs in people with heart failure with preserved ejection fraction and CKD that have shown dual benefits on both organs. So in someone like this, and Niko's gone through the evidence, clear indication for therapy with both of these agents.

The other thing to note is that whilst albuminuria gives you important prognostic information, we then—what we do is we measure it longitudinally, and reductions in albuminuria also correlate with treatment response.

We've got good data showing that for every 30% reduction in albuminuria we reduce the risk of kidney failure by 20%. And so what that has meant is that if you look at the American Diabetes guidelines, they recommend an albuminuria reduction of 30% specifically as a treatment target.

So longitudinally now, we're implementing these therapies then serially measuring their UACRs to assess treatment response. We've also got data now with the nonsteroidal MRAs in non-diabetic kidney disease from FIND-CKD, so it actually doesn't matter now whether this patient has diabetes or does not have diabetes. Both an SGLT2 inhibitor and a nonsteroidal MRA has proven benefit.

So again, what we're seeing is simplicity, and what that means from an implementation perspective is whether you're a cardiologist, primary care physician, or nephrologist, at least from my perspective, it doesn't matter. I don't care whether Niko or you, Javier, initiates the finerenone or an SGLT2 inhibitor. Bottom line, all I really care is that this patient is on those therapies.

**Dr. Morales:**
Yeah, and as you had mentioned before, I mean, really the cornerstone therapies are largely going to be centered around the nonsteroidal mineralocorticoid receptor antagonist, both in the diabetic population and, as you just mentioned, in that FIND-CKD study in the non-diabetic population. And historically, when you're looking at the SGLT2 trials specifically for chronic kidney disease, they specifically had populations of patients that were involved that did not have diabetes.

So this is really good, and I mean, Brendon, you couldn't have said it nicer by saying simplicity, because these agents work very, very well in terms of curtailing the progression of chronic kidney disease. But far important is how well these 2 classes of agents can actually play in the sandbox together because both in combination will lead to significant reductions in the UACR, and there might be some additional protection that the SGLT2 inhibitors offer for or towards the trend of developing hyperkalemia.

**Dr. Marx:**
Maybe, Javier, can I add one point? I think it's important. This patient has diabetes, obesity, CKD, and heart failure with preserved ejection fraction, and I think the third drug class that needs to be on board is the GLP-1 receptor agonist because, from FLOW, we have very good data, diabetes and CKD, kidney and cardiovascular benefit. And with respect to heart failure, very nice data from STEP-HFpEF diabetes that this patient benefits with respect to improvement of symptoms.

So this is something. And we talked about how fast to implement these therapies, and cardiologists have good evidence for the REF patients, and I think from the from the nephrology part, we are getting more and more evidence that early implementation is crucial. Don't wait for 3 months. Don't wait for the next UACR because the patients benefit early, and so that's what we would put in right away.

Brendon, probably you agree, I hope.

**Dr. Neuen:**
Yeah. Niko, perhaps we've held your hand historically treating some of the more advanced CKD patients, but now it's your turn to hold our hand and institute these therapies in a rapid fashion.


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To understand the nephrology perspective, we have always been somewhat conservative and risk-averse. I think that's well known. And so we've been typically slower in how we initiate therapies, and part of that is born out of the historical fact that we haven't had very many therapies for kidney disease. But I think now the equation is very different. And what I say to both patients and to my colleagues is if you institute therapy as rapidly as possible without delay, and we can talk about the kind of support and infrastructure that you might need to do that, the earliest benefit for the patient is reduction in heart failure events, and the long-term benefit is the reduction in kidney failure.

So there are multiple benefits. They occur at different times, but that helps us and the patients and the clinicians just understand why we're trying to do this quickly, because the benefits accrue very rapidly with respect to heart failure.

**Dr. Morales:**
So it's kind of interesting because oftentimes the patient is seeing multiple practitioners, multiple clinicians, and oftentimes some clinicians tend to be quite territorial when it comes towards managing particular outcomes. So for instance, I'm sitting here and we're talking about our patient journey, our type 2 diabetic obese individual with a little bit of shortness of breath and edema, but this patient in particular may have chronic kidney disease, they may have heart failure, but who's watching it? Who's watching the stove cooking? Who's actually taking ownership in terms of all of these parameters? Should it be the nephrologist looking at the kidney? Should it be the cardiologist looking at heart failure? Who basically owns it? From an internal medicine standpoint, I think this is pretty much holistic. We have to look at it in general.

But I think we've learned the lesson, particularly during the pandemic, about constant communication between the multiple specialists. So at least here in the United States with electronic medical records that are oftentimes shared, then we could see the thought process that might be going on from an interdisciplinary approach.

But nothing is easier than just picking up your mobile device and saying, "Hey, Niko, what do I do with this particular patient? How are we going to modify this patient's therapy?"

**Dr. Neuen:**
I think that's a key message. This has to be multidisciplinary, and no matter what your resources are, you've got to find a way to make it work with all of these other clinicians because the bottom line is there are just far too many patients like this for a cardiologist and a nephrologist and, to be frank, primary care to look after, and we need to find ways to do it with our colleagues.

The other thing I would say also is that many of these therapies that we've spoken about, especially when they're instituted in the setting of heart failure, can cause fluctuations in kidney function. But those should not dissuade us from implementing these therapies and continuing them.

Too often they're discontinued for temporary or short-term fluctuations in kidney function when these therapies are lifesaving. That's a really important aspect.

**Dr. Morales:**
And that's a really, really, very important message that was made because so many people tend to peel away therapies just because of the decline in glomerular filtration. But when you're looking at all of these historic studies, with initial treatment, there's always going to be a little bit of a dip in that GFR, and the question is how much of a dip is considered acceptable?

I think some of the literature says about maybe 30% or so was expected.

**Dr. Neuen:**
Yeah. No, that's absolutely right. And the other thing where the interface between all our specialties is incredibly important is that post-discharge period, and I'm sure it's the same in the US and in Germany, where communication is often suboptimal. But that's a high-risk period for our patients, and we're often trying to get people onto guideline-directed therapies.

If we can work together to follow up patients early and assess and titrate therapies or guideline-directed or foundational therapies, assess side effects, monitor potassium and kidney function, that's how we're going to get the best outcomes for these individuals. So concentrating on that early post-discharge period and then assessing for side effects.

**Dr. Morales:**
Yes, and remember that a pillared approach oftentimes is necessary because the additive use of these agents ultimately translate into a much better outcome and potentially less adverse events that can happen. So taking into consideration the kidney, its effect on the heart, I mean, nobody said it really nicer than the STAMP campaign that was introduced in the European Society of Cardiology this year.


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And just to remind our audience about the acronym of what STAMP actually means, because it's quite practical to the discussion we're having here today, and that is we need to **S**creen for chronic kidney disease and don't be GFR-centric. We need to**T**riage and stage that chronic kidney disease by looking at that UACR, looking at the clinical patient, and then we need to **A**ddress that cardio-kidney risk by implementing appropriate therapies with **M**odification if needed based on the need for additional intensification. And remember to **P**lan ahead for some of the health services that the patients may actually need on this journey of the marriage between the kidney and the heart and overall well-being.

Anyway, that's all the time that we have for today. So I want to thank our audience for listening in and thank you, Professor Niko, for joining us, and Dr. Brendon, and it's been a really fruitful discussion. Thank you so much for your participation and for your valuable insight to our audience.

**Dr. Marx:**
Thank you very much.

**Dr. Neuen:**
Thanks so much.

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