Episode Title: Cardio-Kidney-Metabolic (CKM) Syndrome in HFpEF & HFmrEF: From Visceral Fat to Guideline-Directed Therapy

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Sabra Lewsey: Welcome to the American Heart Association's Heart Failure Podcast Series. This Podcast is titled cardio, kidney, metabolic, syndrome, and heart failure with preserved and mildly reduced ejection fraction from visceral fat to guideline directed therapy. I'm Dr. Sabra Lewsey, a heart failure cardiologist at Johns Hopkins, and this program has been created and directed by a volunteer planning committee, and is made possible by support from Bayer. I would like to give my co-moderator and colleagues an opportunity to introduce themselves before we get started in this conversation

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Sabra Lewsey: Dr. Sauer.

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Andrew J. Sauer: Thanks, Sabra Andrew Sauer. I'm a heart failure and transplant cardiologist at Saint Luke's Midamerica Heart Institute. I also focus on Cardio kidney metabolic disease, and I direct our cardiometabolic center as well as the Cardiometabolic Center Alliance, a consortium of several sites that do this work. And you know we're excited to talk about heart failure preserved ejection fraction at the intersection of CKM. And I'm going to hand off to my co-discussants to introduce themselves.

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Sabra Lewsey: Dr. Ho!

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Jennifer Ho: Hi, I'm Jen. Ho! It's really nice to be here today. I am a heart failure cardiologist, and also serve as director of research in cardiology at Beth Israel Deaconess Medical Center, so super excited to join today.

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Sabra Lewsey: Thanks so much, and Dr. Vaduganathan.

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Muthuah Vaduganathan: Thanks so much, Muthu Vaduganathan. I'm a cardiologist at Brigham and Women's Hospital and co-director of the center of cardiometabolic implementation science, and so excited to join this panel.

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Sabra Lewsey: This is surely going to be a great discussion. So before we get this started, it's important to give this disclaimer the recommendations and opinions presented by our faculty today may not represent the official position of the American Heart Association, and the materials are for educational purposes only, and do not constitute an endorsement or instruction by the AHA. The AHA does not endorse any product or device to get us centered for our conversation. I think we should get started with the case. I know we grew up in the practice of

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Sabra Lewsey: using a case to anchor us. So this is a case of Miss D. She's a 57 year old woman. She's presenting with worsening shortness of breath that has gone from mild to a more significant limitation in the past couple of months, in

spite of the use of diuretics which she's already on, she did have an echocardiogram. It showed preserved systolic function and a dilated left atrium, and she did have lab testing where natriuretic Peptides were only borderline elevated.

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Sabra Lewsey: She does have type 2 diabetes, and she's had that since she's been about 39. She's been trying her best to lose weight. She was recently diagnosed with sleep apnea, and has a body mass index around 40, but really she's too limited to do exercise now, and often doesn't have any flexibility in her work hours. She's making better food choices, but feels she has limited, healthy food options in her immediate neighborhood.

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Sabra Lewsey: She does have some concerns that there's protein in her urine because her primary care just told her that. And she is scheduled to see a kidney specialist. So for Ms. D. Does she have HFpEF, or does she have CKM Syndrome or both? And where should we start in understanding her risk, and where to implement necessary interventions as her treating provider. Dr. Ho! How would we even define CKM.

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Jennifer Ho: Yeah, this is a great case, because it's so complex and has so many related comorbidities in there. And yet it's so representative of the types of patients who we see so commonly now. So I think to get to the 1st questions, how do we stage this patient? This is a patient with known obesity. And so they meet at least CKM. Stage one criteria, and if we go by the

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Jennifer Ho: ACC/AHA heart failure stages, she's also been Stage A added on top of that, this patient has type 2 diabetes and albuminuria, and so that would actually move us up in the CKM stages to stage 2 still qualifies as heart failure stage A, and then, with her structural heart disease in a dilated left atrium.

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Jennifer Ho: Actually, she moves us up to CKM stage 3, or sort of pre heart failure with evidence of cardiac remodeling. And then we know she has dyspnea. I guess I would say that we don't know whether her dyspnea is attributable to heart failure or elevated filling pressures or not at this time.

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Jennifer Ho: so there's perhaps more evaluation needed there. But if her dyspnea is really thought to be in the setting of elevated, left-sided filling pressures, then she would certainly also meet HFpEF criteria, and in this case that would solidly put her into the CKM Stage 4 category along with heart failure Stage C.

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Jennifer Ho: I think a couple of other things. We know that there's sort of other complex overlay of some of her health behaviors and and other things that are playing into some of the challenges in this case, that I think were sort of part of the rationale of maybe putting together a CKM initiative. We also know that HFpEF overall is under recognized in patients with obesity. So those are sort of the initial thoughts for this case.

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Andrew J. Sauer: Thanks for those thoughts, Jen, I think it's really important to kind of reiterate that we see these patients all the time. And as you point out, Jen. You know we don't even have a full assessment of the HFpEF question, because we don't have any data here for this patient on markers of congestion or remodeling. So you know, we see these

patients in clinic, and they may have seen 4 or 5 cardiologists before us, and oftentimes it's unfortunate. They've not even had Natriuretic Peptides checked.

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Andrew J. Sauer: And perhaps there's evidence of elevated PA pressures. There's already evidence in this echocardiogram for left atrial enlargement. So there's probably some evidence that there's some heart failure. And yet that diagnosis oftentimes is not even made. So I do think that's a really important point is that we need to do a better job, actually diagnosing both the CKM. As well as the heart failure as well as documenting their ejection fraction.

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Andrew J. Sauer: So Muthu, tell us a little bit more, I mean a little bit of planned redundancy with the American Heart Association CKM presidential statement. So Jen kind of gave us a thread of an introduction to the CKM stages. But can you help us? Maybe kind of compare and contrast CKM staging with heart failure staging so that the clinicians can kind of put a framework in their mind. To Jen's point.

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Muthuah Vaduganathan: Yeah. And I thought, Jen really laid out that foundation related to this case really, really, nicely, and that these really are intended to evolve in parallel that AHA stages move from early risk factors to ultimately subclinical disease and clinical disease and manifestations of heart failure.

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Muthuah Vaduganathan: And similarly, the CKM framework put forth by the American Heart Association presidential advisory moves through stages 0 1, 2, 3, and 4. So stage 0 is really people without any risk factors, including no evidence of adiposity, and represents less than 10% of the American population.

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Muthuah Vaduganathan: Now, stage one is individuals with excess adiposity, so-called. These angry adipocytes or dysfunctional adiposity. And then stage 2 is when we start to see metabolic risk factors manifest that are associated with obesity, including high triglyceride levels, hypertension, diabetes, metabolic syndrome, as well as earlier manifestations of chronic kidney disease. Early evidence of albuminuria, for instance.

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Muthu Vaduganathan: stage 3 is evidence of subclinical cardiovascular disease, including based on imaging such as

coronary calcification or coronary plaque buildup. Furthermore, more advanced manifestations of chronic kidney disease are also included in Stage 3 stage 4. Individuals with truly clinically manifest cardiovascular disease. This is patients presenting

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Muthu Vaduganathan: instance with myocardial infarctions, or presenting with heart failure or atrial fibrillation. As a part of the CKM syndrome, so as you can see this is presented in parallel to the staging system that

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Muthu Vaduganathan: We know about and used in clinical practice for heart failure. And CKM truly does evolve alongside heart failure staging. I think this is actually a very nice way for clinicians to frame risk trajectories of disease as well as communicating that to patients.

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Sabra Lewsey: Thanks so much, Muthu, that that's really helpful to to go over the stages. Can we think about with the CKM stages? And this concept of dysfunctional adipocytes as you mentioned.

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Sabra Lewsey: what is the underlying pathophysiology really for visceral adiposity and insulin resistance?

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Sabra Lewsey: How does CKM and heart failure intersect in this pathophysiology, Muthu?

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Muthu Vaduganathan: Yeah. So I think this is really, really, critically important. And there's a lot of crosstalk between adiposity and heart failure risk, for instance, adipocytes themselves express

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Muthu Vaduganathan: locally as well as systemically, on the heart, to influence risk of progression of heart failure. Furthermore, adiposity can manifest in different locations or depots all across the body, including around the pericardium, the chest wall, as well as in visceral tissue beds.

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Muthu Vaduganathan: Much of that culminates to actually increasing degrees of diastolic dysfunction and pericardial constraint, as well as impairing distensibility of the heart.

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Muthu Vaduganathan: Furthermore, adiposity can increase circulating plasma volume that also contributes to the manifestations of HFpEF but I don't know if I know that there are many, many intersecting pathways here. I don't know if any of the other speakers as well might had their own favorite pathways that might contribute to heart failure.

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Sabra Lewsey: Yeah. Dr. Ho, doctor, Sauer.

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Jennifer Ho: I'm happy to jump in. I think Muthu's done a fantastic job. I think there are so many pathways involved. It's hard to attribute. Maybe one or the other, you know, if we step back, and we even recognize that 80% of patients with HFpEF have overweight or obesity, it just makes it so plainly obvious how big of a problem this is. We know that there have been really big shifts in

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Jennifer Ho: cardiovascular risk really from predominantly driven by hypertension and hypercholesterolemia

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Jennifer Ho: in the last 25 years. Really, now, to be more obesity, CKD, hyperglycemia related. So I think all of those pathways you mentioned probably play a role, including inflammation, metabolic dysfunction, and probably also kidney disease that then set someone up to be much higher risk for heart failure in general.

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Jennifer Ho: I don't know, Andrew, if you have other thoughts here.

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Andrew J. Sauer: Yeah, I think just to add a few things that haven't already been mentioned is just remember that adipose itself is essentially active.

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Andrew J. Sauer: It's not inert. It has implications for inflammatory adipokines, activation of the Renin-angiotensin aldosterone system. So we do get a lot of contribution for example, from visceral adiposity to the pathophysiology of hypertension itself.

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Andrew J. Sauer: So, and as we think, we've learned a lot about this intersection that both Jen and Mutu are talking about as we think about disease modifying therapies which we'll talk about in a moment. As we've learned about how these disease modifying therapies impact many of these pathways, we realize that this is really predominantly HFpEF.

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Andrew J. Sauer: As already stated, 80% of HFpEF is metabolically oriented disease. And so thinking about how all these pathways intersect is really part of being a good clinician, and that's why cardiologists who take care of HFpEF now have to think like

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Andrew J. Sauer: about the kidneys and have to think about the liver and the fatty liver disease, for example, metabolic liver disease. Because all of these pathways are intersecting and the therapies we use modify these pathways at multiple levels.

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Andrew J. Sauer: So I do think the Pathophysiology is really important to keep in mind, as we think about how to care for these patients.

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Sabra Lewsey: So I hear, go ahead. Dr. O.

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Jennifer Ho: Sorry to jump in. Yeah, I guess as an epidemiologist, there are sort of 2 super interesting things that are probably worth

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Jennifer Ho: bringing up. One is that we know that HFpEF, clinically is a female, predominant syndrome. Right?

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Jennifer Ho: We know that women with obesity are at greater susceptibility of developing future HFpEF compared with men. And I think there's a lot of things that we can learn about disease pathophysiology. There, for example.

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Jennifer Ho: we've previously shown that visceral, adipose tissue which we think is really central, like Andrew pointed out at driving CKM. In general. When we look at visceral, adipose tissue in women, for example, that visceral, adipose tissue is associated with greater elaboration of adipokines.

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00:14:18.960 --> 00:14:41.890
Jennifer Ho: inflammation, and fibrosis markers compared with visceral, adipose tissue in men. So I do think there are many things that are still unknown but really interesting ways in which we can potentially better understand these disease mechanisms. The other thing I'll point out is that there are really important geographic differences that I think are so fascinating. So we know in Asian populations, for example.

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Jennifer Ho: HFpEF can occur even at much lower BMIs, and it turns out that at a lower given BMI Asian populations tend to have higher prevalence of diabetes, and just as much HFpEF. So the question is, is this really obesity driven? Or is this metabolically driven? And probably the answer is a combination of both.

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Andrew J. Sauer: That's a really important point to segue into kind of the next discussion point, Muthu. If you could help us, how do we

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Andrew J. Sauer: identify patients sooner in the staging, whether it be CKM stages earlier stages before overt disease, clinical disease as well as for heart failure. You know, we'd like to start identifying

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Andrew J. Sauer: patients with HFpEF, for example, in Stage B, because we know that there are therapies that can modify that disease trajectory. So what do you think about when you're seeing a patient as ways to kind of screen essentially for markers of more advanced CKM or heart failure.

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Muthu Vaduganathan: So the AHA Presidential advisory for CKM recommends early screening as early as in individuals of the age of 30 years or older for CKM risk factors and obesity guidelines even recommend screening in adolescents for the early management of overweight and obesity. However, we know heart failure is largely a condition that affects middle aged and older adults.

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Muthu Vaduganathan: and so screening for heart failure largely takes place at other touch points in care when new risk factors develop. For instance, the type 2 diabetes guidelines recommend annual screening with natriuretic peptide levels in people. Even after 1st diagnosis of type 2 diabetes.

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Muthu Vaduganathan: Similarly, when patients initially present for chronic kidney disease, adjacent evaluation for heart failure is also recommended. And so that's my own clinical perspective as well, is that when these risk factors or pathways to where heart failure commonly occurs

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Muthu Vaduganathan: is seen in clinical practice, those are the time points that we should be screening. Now that we have cost effective biomarkers like natriuretic peptides and point of care imaging tools, think that screening for heart failure should become more top of mind for clinicians.

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Sabra Lewsey: Thanks so much with regards to how we evaluate patients across the CKM spectrum earlier on in life.

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Sabra Lewsey: and even when heart failure is then present? What is the best means of risk stratification? Should we be just using their renal function labs? Should we be thinking about quantifying albumin in the urine.

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Sabra Lewsey: What would you say, Andrew, with regards to this.

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Andrew J. Sauer: Yeah, I mean, I think in our practice now we, you know, when our when our patients with HFpEF are intersecting with CKM, which is, we've talked about quite a bit here. It's quite a few of them.

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Andrew J. Sauer: We need to be more comprehensive in our screening. And, in other words, we need to keep the nephropathy in mind. For example, you know, cardiologists are not used to routinely checking urine-albumin-creatinine ratio UACR. But they need to start becoming more comfortable doing that because it's not just about the risk to the kidneys as part of comprehensive

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Andrew J. Sauer: KDIGO guideline oriented nephropathy screening. It's also about the cardiovascular risk that comes with states of elevated UACR, be it micro or macro albuminuria. So UACR. Over 30, for example, is an opportunity to start treating patients, particularly those with diabetes, treating them differently and treating them early. So I think thinking broader about CKM in the construct of treating HFpEF because most of our patients with HFpEF

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Andrew J. Sauer: have Metabolic Associated disease.

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Andrew J. Sauer: And then I think the other space to think about more that we're doing in our clinic now is the liver space so MASH and MASLD. There are therapies now for MASH and MASLD to help prevent the progression of cirrhosis, which has intersections with CKM and cardiovascular disease, and so we use basic FIB-4 screening, for example, which you can just check the calculator. And all you need is an AST ALT, a platelet and an age. And basically every patient has this. So

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Andrew J. Sauer: when FIB-4 is elevated enough, it can prompt checking elastography like basically a fiber scan to then look for markers of steatosis and deranged liver disease. And this all intersects, as we've said, and we have therapies like GLP-1's, which we'll talk about in a second that have been shown to help reduce the progression of disease and liver disease as well as the kidney disease.

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Andrew J. Sauer: I think it's just important to take home that CKM. Involves looking beyond just the heart, because this has implications for the heart, even though these other pathways are outside of the heart.

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Sabra Lewsey: Getting us all to be that holistic provider taking care of these patients.

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Andrew J. Sauer: We've been going back to being medicine docs again, and I think it's great. I think a lot of us have jumped in this space. Willingly. We're excited about it.

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Sabra Lewsey: Yeah. So going back to our patient, taking us back to the focus. So we recognize that she has. CKM related HFpEF. And she has these related conditions, diabetes, obesity, sleep apnea, kidney disease. So what therapies are really essential for her to be started on right now by guideline recommendation and by

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Sabra Lewsey: really what's evolving in terms of current available evidence? And would we call these disease modifying therapies.

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Muthu Vaduganathan: Yeah, so perhaps I can start I think the foundation of CKM management CKM, Associated risk is

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Muthu Vaduganathan: with RAAS inhibitors. And we've known this for now decades that RAAS inhibitors, especially in albuminuric patients with chronic kidney disease especially associated with type 2 diabetes really is disease modifying not only reduces early cardiovascular risk, but also longitudinal kidney disease progression. In addition, SGLT-2 inhibitors are now foundational and perhaps are considered one of the 1st true CKM therapies

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Muthu Vaduganathan: SGLT-2 inhibitors have been shown in dedicated clinical trials to reduce clinical events in people with type 2 diabetes and cardiovascular risk

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Muthu Vaduganathan: in people with chronic kidney disease, with and without diabetes and with heart failure across the ejection fraction spectrum. And so, in a person

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Muthu Vaduganathan: like this case, here we have SGLT 2 inhibitors should really be an early choice in how we manage her cardiovascular risk and CKM. Risk.

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Andrew J. Sauer: Yeah. And I think so. You've got RAASi. You've got SGLT 2 inhibitors. I think. The next class that we talk about more recently is the MRA space, and this is a little bit muddy. I think it gets a little confusing for folks, particularly when you look at Spironolactone, for example, which is a drug, a medication I clearly love for heart failure in general, very powerful in HFrEF, but unfortunately, TOPCAT had a neutral result, and you know, even when you look at the North Americas, there's some.

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Andrew J. Sauer: There's some trickiness to trying to decipher that. And the guidelines have given very lackluster recommendations for steroidal MRA for appropriate reasons. And so we've seen non-steroidal MRA emerge as maybe a potential, true dedicated pillar therapy, particularly when you look at the combined evidence that we have from the diabetes and CKD data that we have from FIDELIO and FIGARO, and

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Andrew J. Sauer: FIDELITY, and then, combined with FINEARTS, with the FINE-HEART studies we now see that for patients in particular with HFpEF, who also have diabetes and elevated UACR over 30, these are really perfect patients to see a likely benefit from non-steroidal MRA, particularly FDA approval for Finerenone most recently, I think, is relevant.

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Andrew J. Sauer: and I think that we have to be thinking about this more and try to parse out some of the differences between traditional steroidal MRAs, which have not yet risen to the level of evidence to provide the same level of recommendation.

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Jennifer Ho: I guess I'll build on both of those. So we know that cornerstone of therapy in this individual would be ARNI based SGLT 2 inhibitors, and then consideration of mineralocorticoid receptor antagonists. The last class that really deserves attention here are incretin-based therapies. Of course, these have really evolved with the recent trials, including STEP-HFpEF STEP-HFpEF diabetes

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Jennifer Ho: that looked at Semaglutide in patients with heart failure, with midrange and preserved ejection fraction, as well as the SUMMIT trial that was published last year, really showing that tirzepatide in patients with pEF and mid-range EF

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Jennifer Ho: was able to reduce primary outcome combined heart failure, hospitalization and cardiovascular death. And so I think, in this particular patient case, who comes in with

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Jennifer Ho: all of the comorbidities obesity plus diabetes plus obstructive sleep apnea plus

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Jennifer Ho: likely HFpEF that hasn't quite been diagnosed yet. I think it really behooves us to think about initiation of incretin-based therapies in this patient.

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Andrew J. Sauer: Yeah. And let's also remember what we've been talking about. It's not just about the heart. All these therapies we've talked about also reduce the progression of chronic kidney disease, and, you know, have been shown to reduce UACR, for example, and and for the incretin based therapies. We're seeing emerging evidence as we talked about with reducing the progression of metabolically associated liver disease. So a lot of power in treating the larger CKM syndrome

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Andrew J. Sauer: in these therapies, it's not just about the HFpEF. If the HFpEF sits within the CKM disease construct. And all these pathways that we've been talking about, it's important to see that these are pillars of therapy that do more than just target the heart.

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Andrew J. Sauer: And I think that's a really important take home.

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Muthu Vaduganathan: And dovetailing on that. I think that really underscores the importance of the CKM. Syndrome itself.

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Muthu Vaduganathan: You know it's remarkable that in 2025. The treatment paradigm for HFpEF almost entirely parallels that

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Muthu Vaduganathan: for chronic kidney disease. And while we've thought about these as siloed models of care, siloed specialties, now, we have the same therapeutic armamentarium, or nearly the same therapeutic armamentarium to combat 2 growing chronic conditions.

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Muthu Vaduganathan: Furthermore, because each of these therapies really not only treat the individual condition, but have adjacent effects on each of the other entities. For instance.

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Muthu Vaduganathan: a therapy like the non-steroidal MRA Finerenone can prevent diabetes. Can slow kidney disease progression by lowering albuminuria levels, and so that actually reinforces the importance of CKM that these therapies have actually taught us about the interconnectedness and interrelatedness of these conditions.

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Andrew J. Sauer: Yeah. So turning back to our patient,

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Andrew J. Sauer: Jen, what do you recommend if this patient's seeing you? And you've got all these tools now. And, as Muthu points out, 5 years ago we were not talking about HFpEF and CKM the same way. We're talking about it today. It's amazing, you know, to stop and think about how far we've come and how much we're learning.

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Andrew J. Sauer: So is there a sequencing? Is there an order? You know these patients are already on 7, 8, 9, 10 medicines. When they see you, how do you decide what to add? And when to add it.

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Jennifer Ho: It's a great question, and something that I think we, as clinicians, really struggle with right, because there's not a lot of guidance here in terms of how to sequence these medications, and I think, really being sensitive to to all of

the other polypharmacy, or many other medications that are ongoing, I think. Really.

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Jennifer Ho: I think we have to be very thoughtful about it. I guess in this patient they're already on a loop diuretic. I think it would be easy to say. Well, can we actually get rid of something? Can we get rid of their loop diuretic by putting them

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Jennifer Ho: on an SGLT? 2 inhibitor and an MRA potentially with those diuretic effects. So that may be one way to kind of think about it in this particular individual. And then, I think, with a BMI of 40, you know, really thinking about what their long-term risk is of other

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Jennifer Ho: complications. I would really be sort of focused on weight management therapy with GLP-1 receptor agonist, but.

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Andrew J. Sauer: Yeah, I think.

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Jennifer Ho: To see yours.

111
00:28:46.820 --> 00:29:04.909
Andrew J. Sauer: Yeah, just as an editorial comment on the GLP-one, we use a lot of these in our clinic. It's really important to have a fair amount of time devoted to talking about Gi side effect mitigation. Because if you look at the clinical trials, less than 10% of patients in pretty much. Any of these trials

112
00:29:04.910 --> 00:29:18.650
Andrew J. Sauer: have significant enough Gi side effects that they have to come off drug. But if you don't warn patients about how to initiate how to up titrate, start low, go slow. For example, we start at low dose. We up titrate once a month.

113
00:29:18.650 --> 00:29:26.730
Andrew J. Sauer: you know we do this sort of automatically with our nurse navigators. And we tell patients it's going to take 4 to 6 months to get you to target dose.

114
00:29:26.730 --> 00:29:42.389
Andrew J. Sauer: and you need to keep your meal size to. Maybe with the size of your fist, you need to eat lots of fiber hydrate. You need to focus on protein. You need to focus on nourishment and avoid some super fatty foods as well as super glycemic enriched foods.

115
00:29:42.390 --> 00:29:59.599
Andrew J. Sauer: and I think that if you don't do that ahead of time, the patients have a bad experience, and then they blame the drug, and oftentimes that becomes a barrier to up titration. So it's important to realize that there's an art to this

Muthu? Do you have other thoughts on sequencing, or titration, or how and when you add things.

116
00:30:00.290 --> 00:30:26.909
Muthu Vaduganathan: No, I think we've adopted this paradigm in HFrEF of rapid sequence implementation, especially around the hospitalization. I think the HFpEF patients a bit more complex, and I actually am with Jen here. I think a bit more of a tailored approach is probably necessary for most individuals because of the spectrum and diverse presentations of HFpEF that we see. But for that very high risk patient that

117
00:30:26.910 --> 00:30:41.660
Muthu Vaduganathan: especially who's now been hospitalized for HFpEF, I do adopt a very similar kind of rapid sequence approach and try to get these core pillar sort of therapies on as quickly as possible. Now, in terms of

118
00:30:41.800 --> 00:31:06.720
Muthu Vaduganathan: other sort of practical tips. Andrew, you discussed the GLP-one receptor agonists and Gi side effects. It's important to note some of the other key issues related to the other therapies, for instance, SGLT-2 inhibitors are often well tolerated in most individuals we do need to be mindful about euglycemic DKA that's most relevant in a patient with type 2 diabetes on

119
00:31:06.860 --> 00:31:34.920
Muthu Vaduganathan: insulin or sulfonylureas, especially with poor glycemic control. Furthermore, they don't have indications for type one diabetes, and so we should be very very careful about use along with ketone monitoring in those individuals. For MRA’s. Really, hyperkalemia is the most important issue, and so closely monitoring potassium levels after initiation is critical and using risk mitigation methods

120
00:31:35.090 --> 00:31:58.979
Muthu Vaduganathan: alongside MRAs. It's also important, for instance, dietary counseling use and appropriate titration of diuretics as well as use alongside SGLT-2 inhibitors, and ARNI’s might lower the risk of hyperkalemia, and then finally, for RAAS inhibitors and ARNI blood pressure lowering is the most substantial, but I think some of the practical tips that Jen shared. For instance.

121
00:31:58.980 --> 00:32:13.190
Muthu Vaduganathan: discontinuation of diuretics or down titration of diuretics or adjustment of non-essential antihypertensive therapies like calcium channel blockers, might allow for continued RAAS inhibition, and ARNI use.

122
00:32:14.030 --> 00:32:15.420
Andrew J. Sauer: Really excellent points.

123
00:32:15.740 --> 00:32:44.639
Sabra Lewsey: Fantastic. I have to take the opportunity with such experts in the field, with regards to implementation, you know, when we think about our care models. This patient is seeing a primary care Physician hasn't quite yet made it to the cardiologist, or maybe is getting diagnostics, for HFpEF. Is referred to a kidney specialist who is in charge of these therapies and initiation? And do we need to really start thinking about how we harmonize our care models for these patients that are dealing with all these things at once. Jennifer.

124
00:32:45.980 --> 00:33:05.840

Jennifer Ho: Yeah, Sabra, you bring up a really important point. How do we even think about implementation of these medications and these sort of interconnected comorbidities. I think, Andrew said it really nicely. We have to really be internal medicine docs and think holistically about our patients. I do think.

125
00:33:06.250 --> 00:33:17.329
Jennifer Ho: You know, the AHA sort of thought about, how do we link care of individuals with CKM to community-based programs to really address sort of these.

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00:33:17.330 --> 00:33:36.549
Jennifer Ho: these fragmented care and silos toward a more coordinated care model. So I think there has to be more coordination to help patients navigate through multiple subspecialties. How do we sort of bring it all together. And so I think there is a lot of effort.

127
00:33:36.560 --> 00:33:40.269
Jennifer Ho: sort of dedicated to trying to improve

128
00:33:40.370 --> 00:33:47.500
Jennifer Ho: where we are currently sort of working across these interdisciplinary teams, I would say in CKM. Health.

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00:33:49.320 --> 00:33:50.649
Andrew J. Sauer: Muthu, other thoughts?

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00:33:54.840 --> 00:34:13.990
Muthu Vaduganathan: No, I I you know this is something that's rapidly evolving. We're trying to learn about best strategies. And I think ultimately, while there are many, many such models, it will be tailored to your individual local health infrastructures and local available resources, and so

131
00:34:13.989 --> 00:34:36.279
Muthu Vaduganathan: some might benefit from virtual care models. Some might leverage local, for instance, allied health professionals or pharmacist-based care models, others might have more navigator programs where there truly are guided, but I think that ultimately we do need to rethink our own local care patterns.

132
00:34:36.280 --> 00:34:46.140
Muthu Vaduganathan: because now we have a new entity. CKM, that brings us together. And I think it's a wonderful framework that we can actually move forward. Our care in parallel.

133
00:34:47.299 --> 00:34:59.069
Andrew J. Sauer: Yeah, we've shown that best ways to implement is to it's really not sexy. It's it's checklist protocol driven up titration initiation of therapies. And it centers on

134
00:34:59.069 --> 00:35:28.709
Andrew J. Sauer: the clinician. The cardiologist, for example, needs to be willing to take charge and really work and do shared care with primary care. Endocrine nephrology, but recognize that at the center of this patient's risk is their cardiovascular risk that drives most of the concern for these patients. They develop atrial fibrillation. They develop

pulmonary hypertension, they develop RV failure. You know, this is what's coming for these patients if we don't intervene. So, being willing to work outside of our silos and say.

135
00:35:28.799 --> 00:35:48.509
Andrew J. Sauer: as I'm up titrating the GLP-1's. For example, I'm going to have to down titrate your insulin, and that's good for you. But we're going to have to work with maybe your endocrine doctor or your primary care specialist to make sure that we're doing this together and doing it safely. So this is the daily grind that we all go through in our efforts to care for our patients. But

136
00:35:48.509 --> 00:35:58.809
Andrew J. Sauer: it's changing the philosophical approach. You know. We don't get to just sit in this cardiovascular lane anymore. And that's actually, I think, a good thing if we can kind of think beyond that.

137
00:35:59.390 --> 00:36:11.239
Sabra Lewsey: Agreed. We all have to bend towards the center and the center being our patients. And really we are the point of the wedge now trying to align this care for them. So today, I think we really talked about

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00:36:11.240 --> 00:36:36.390
Sabra Lewsey: really recognizing the risk that our patients are at with regards to CKM, and how that can progress to include cardiovascular disease, especially including heart failure. How to risk, stratify these patients with biomarkers, when to think about risk stratifying these patients when any of these comorbidities come up, and then how to really get them on evidence-based therapies. And we know that that evidence base is quickly evolving

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00:36:36.390 --> 00:36:46.719
Sabra Lewsey: and reminding ourselves that we all need to be active in that role and reevaluate their risk along their life course. So I just want to thank

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00:36:46.720 --> 00:37:10.289
Sabra Lewsey: all of you for this fantastic conversation I have learned so much. I know that all of our colleagues that will be listening will benefit from this, and their patients as well. And so, Dr. Sauer, Dr. Ho. Dr. Vaduganathan, thank you. Thank you for joining us for this conversation, and a reminder that this episode is a part of the American Heart Association's heart failure Podcast series.

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00:37:10.290 --> 00:37:15.099
Sabra Lewsey: More episodes can be found at learn.heart.org. Thank you.