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The Critical Interplay: CKD, HF, and HyperkaleAmia

Announcer:
Welcome to CE on ReachMD. This activity is provided by Medtelligence and is part of our MinuteCE curriculum.

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Dr. Rossignol:
This is CME on ReachMD, and I'm Dr. Patrick Rossignol. Today, we will review the pathophysiology of chronic kidney disease, heart failure, and hyperkalemia.

CKD and heart failure are intertwined conditions. And in a white paper, Faïzan and I published in Circulation in 2018, we proposed to revisit cardiorenal syndrome with considering a single umbrella. Indeed, these patients are exposed to the same cardiovascular and renal risk factors: hypertension, diabetes, obesity, dyslipidemia, tobacco use, which may trigger the same pathophysiological pathways, including fibrosis.

This may lead to damage to both the heart and the kidneys, leading to some degrees of kidney function impairment, decreased eGFR, and increased microalbuminuria, and heart damage, left ventricular hypertrophy, diastolic dysfunction, ultimately leading to overt CKD and/or heart failure. We proposed that all patients should be treated the same way, with the same drugs—ACE or ARBs, MRAs, and SGLT2 inhibitors.

Some years later, as major randomized clinical trials in heart failure and in CKD were completed, and compellingly successful with SGLT2 inhibitors and the non-steroidal mineralocorticoid receptor antagonist, finerenone, the American Heart Association endorsed the concept of CKM—cardiac-kidney-metabolic—syndrome.

While all guidelines across medical specialties now recommend to implement ACE, ARB, SGLT2 inhibitors, and, in increasing settings, non-steroidal mineralocorticoid receptor antagonism.

CKD is highly prevalent in heart failure. Worsening chronic kidney disease and worsening renal function are strong prognostic factors in heart failure. CKD, heart failure, and diabetes are common causes of impaired potassium excretion, which may lead to hyperkalemia. While hyperkalemia is a pharmacologically inherent risk associated with the use of renin–angiotensin–aldosterone system antagonists. Furthermore, CKD may trigger hyperkalemia through potassium redistribution mechanisms in case of metabolic acidosis.

All these features contribute to the frequent recurrence of hyperkalemia and are strong hurdles for the implementation of RAASi life-saving drugs. Indeed, hyperkalemia is a trigger to RAASi discontinuation.

In conclusion, the increased use of ACE, ARBs, and/or MRAs has resulted in increased awareness of risk of serious worsening of renal function and/or hyperkalemia. The fear of inducing worsening of renal function or hyperkalemia has limited the initiation or increase in dose of those life-saving drugs in patients with heart failure and CKD.

Therefore, there is a clinical need for chronic hyperkalemia treatments that are effective, safe, and well-tolerated to enable use and optimization of RAASi therapies.

Well, that's our time. Thank you for listening.Bottom of Form

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