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Released: 10/24/2025
Valid until: 10/24/2026
Time needed to complete: 1h 00m

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CKD-aP: Beyond the Surface

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

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Dr. Kraft:
This is CME on ReachMD, and I'm Dr. Leonie Kraft. Today, we'll take a closer look at chronic kidney disease-associated pruritus, or in short CKD-aP, and we will focus on its prevalence and pathophysiology.

Despite advances in dialysis care, moderate to severe pruritus continues to affect over 30% of patients on hemodialysis. And there's data from 2 large observational studies, like the DOPPS trial from 2009 to 2018 with almost 8,000 patients and the more recent CENSUS-EU trial by 2024 with percentages ranging from 26% to almost 40% of dialysis patients.

So if we look at the numbers, this shows that almost every third patient on hemodialysis is affected by moderate to severe CKD-aP. And this data just underscores how common and persistent and chronic disease or symptoms of CKD-aP is, despite the fact that we often just ignore it and don't talk about it.

And why it is important to talk about CKD-aP is that it is associated with adverse clinical outcomes. Studies have linked it to increased mortality, higher hospitalization rates, and increased healthcare costs, but also as important—or if you ask me, even more important—is the reduced quality of life reported by the patients. Almost all individuals with CKD-aP report a substantial impact on their daily lives with poor sleep quality and depressive symptoms.

And as the severity of pruritus increases from none to mild, moderate, or severe, so do sleep disturbances, depressions, and so do the impact on their daily lives with leisure, errands, work, or school.

The pathophysiology of CKD-aP is complex, not yet fully understood, and it is certainly multifactorial, with opioid imbalance, skin conditions, toxin removals with uremia on dialysis, neuropathy, and immune modulatory reasons that all have an impact on CKD-aP.

And all those different underlying causes of CKD-aP have different therapeutic approaches. They've listed a lot of them here, but we will explore a lot of these treatment strategies later today. This is just meant to show you the complexity of the issue and the complexity of the pathomechanisms, to be honest.

And if we look a bit closer, this diagram just illustrates the interplay of the different triggers I showed you that are contributing to CKD-aP.

In recent years, particular attention has been given to the role of an opioid imbalance, specifically increased mu-opioid receptor expression, alongside with a downregulation of kappa-opioid receptors, which is the target of the new kappa-opioid receptor agonist difelikefalin. But in addition, a systemic immune response involving proinflammatory cytokines and inflammation has been recognized as a factor in the pathogenesis of CKD-aP.

And I want to show you an interesting recent study from 2025 investigating the association between inflammatory markers and CKD-aP. And the authors found significantly elevated levels of proinflammatory cytokines in patients suffering from moderate to severe pruritus compared to those without symptoms or with only mild symptoms. And notably, patients that responded to the kappa-opioid receptor agonist difelikefalin, they showed a greater reduction in inflammatory markers compared to nonresponders, which again supports that systemic inflammation plays a key role in CKD-aP.

So to conclude—and if there's something I want you to take away from this, then there are 3 points—CKD-aP is more common than we think. It greatly impacts the quality of life of your patients. And if we understand the different pathomechanisms of CKD-aP, we might be able to treat our patients more adequately.

That's our time. Thank you for listening.

Announcer:
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