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Advances in Heart Failure Care: FINEARTS-HF Findings on Finerenone

Announcer:
You're listening to *Audio Abstracts* on ReachMD. Here's Dr. Ankeet Bhatt.

Dr. Bhatt:
Good morning. My name is Dr. Ankeet Bhatt. I'm an Associate Physician, practicing cardiologist, and Clinical Investigator at the Kaiser Permanente San Francisco Medical Center in Division of Research in San Francisco, California. Today, I'll be talking about the FINEARTS-Heart Failure study, which explored the role of the nonsteroidal mineralocorticoid antagonist, or MRA for short, in patients with mildly reduced and preserved ejection fraction.

For context, MRAs are known to be beneficial in patients with heart failure and reduced ejection fraction. However, their role in treating patients with mildly reduced or preserved ejection fraction has not yet been fully explored—until the results of the FINEARTS-Heart Failure study.

This study, published in the *New England Journal* in October 2024, assessed the efficacy and safety of finerenone, which is a nonsteroidal mineralocorticoid antagonist, in a patient population with mildly reduced and preserved ejection fraction, asking the question of whether this may be a new, beneficial therapeutic avenue in these patients with mildly reduced and preserved ejection fraction.

So, let's dig into the study. The study enrolled over 6,000 patients from 37 countries. Eligible patients had symptomatic heart failure and a left ventricular ejection fraction of 40 percent or higher. Eligible patients were then randomly assigned to receive either finerenone or placebo. Finerenone was dosed based on baseline renal function and up-titrated to 20 to 40 milligrams once daily.

The primary outcome was a composite of total worsening heart failure events, which included hospitalizations or urgent visits for heart failure, and cardiovascular death.

What did the study show? The study results indicated that those randomized to finerenone experienced a statistically significant 16 percent reduction in the primary outcome as compared to placebo over a median follow-up of 32 months. This also included an 18 percent reduction in total worsening heart failure events alone. While cardiovascular death rates were modestly lower in the finerenone group—and numerically lower—this difference did not reach statistical significance.

Additionally, patient-reported outcomes as measured by the Kansas City Cardiomyopathy Questionnaire showed a modest but statistically significant improvement in symptom burden amongst those randomized to finerenone, with a 1.6-point advantage as compared to those randomized to placebo. However, there were no meaningful differences in New York Heart Association class or a composite kidney outcome. All-in-all, these data suggest that finerenone, as compared to placebo, improves both clinical events and health-related quality-of-life.

In terms of safety, finerenone was associated with a higher incidence of hyperkalemia and serum creatinine levels as compared to those randomized to placebo, though rates for hospitalization or death from hyperkalemia were low, and the total incidence of hyperkalemia was lower than that seen in prior trials of a steroidal mineralocorticoid antagonist, spironolactone.

Conversely, rates of hypokalemia, which is known to be associated with adverse effects, were lower amongst those randomized to finerenone as compared to those randomized to placebo.

All-in-all, these results from the FINEARTS-Heart Failure trial demonstrate that finerenone reduces heart failure-related clinical events in patients with mildly reduced and preserved ejection fraction, suggesting that this may be a new therapeutic avenue for a condition that

has a high residual risk and limited therapeutic options to date.

I thank you for your time and for your attention, and I hope that this provided a synopsis of the FINEARTS-Heart Failure trial.

Announcer:
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