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Finerenone in Patients With IgAN: A FIND-CKD Analysis

Announcer:
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Dr. Neuen:
Hello, everyone. My name is Brendan Neuen. I'm a nephrologist and director of kidney trials at Royal North Shore Hospital in Sydney, Australia, and program lead for the Renal Division at the George Institute for Global Health. I'm coming to you here from ASN Kidney Week in Denver, Colorado, where we've just presented new data on the effects of finerenone in patients with IgA nephropathy, which was a subgroup analysis from the FIND-CKD trial.

As you might already know, FIND-CKD evaluated the effect of the nonsteroidal MRA finerenone in patients with nondiabetic kidney disease. There were about 400 patients—or 415 patients—in the trial that had IgA nephropathy, and in this analysis that I presented, we looked at the efficacy and safety of finerenone in this population, including on effects on proteinuria thresholds and targets recommended by the KDIGO guidelines, as well as a range of exploratory biomarkers both in the plasma and in the urine that might help us understand how the drug exerts its kidney-protective effects in patients with IgA nephropathy.

Almost all of these 400 or so patients had their disease confirmed on biopsy. The mean eGFR was about 49 mL/min, and the median UPCR was 1.1 g/day. About 21% of patients were on an SGLT2 inhibitor at baseline. We also had detailed histological data, including MEST-C scores in about half of the patients who had a kidney biopsy.

What we found was that finerenone reduced the primary outcome of total GFR slope by 0.7 mL/min/year in the overall population, and this effect was consistent or similar in patients with IgA nephropathy compared to other forms of kidney diseases. When we looked at the effect on chronic GFR slope, so excluding that acute effect, what we found was a 1.2 mL/min/year improvement in GFR, and again that was similar in patients with IgA nephropathy compared to other forms of kidney diseases.

In terms of reductions in proteinuria, we observed a 50% reduction in proteinuria and 50% reduction in albuminuria over 12 months in patients with IgA nephropathy. What this translated to was an almost sevenfold higher odds of achieving the KDIGO guidelines targets of less than 0.5 g/day of proteinuria, and ideally even less than 0.3 g /day of proteinuria. Again, a sevenfold higher likelihood in those patients who are allocated to finerenone versus placebo.

In terms of the biomarker data, these were really interesting and informative. What we found was that finerenone did not modulate biomarkers related to the immunopathogenesis of the disease. It didn't meaningfully alter APRIL, BAFF, Gd-IgA1, or total immunoglobulins. But in contrast, what we did see were clear reductions in a range of kidney damage biomarkers, particularly kidney injury molecule one or KIM-1, and matrix metallopeptidase 7, or MMP-7, both in the plasma and in the urine. And we know these markers are implicated in particular in interstitial injury and fibrosis in the kidney. What this suggests is that finerenone is exerting benefits through mechanisms that are likely to be distinct and complementary to those of immunological drugs in patients with IgA nephropathy.

Overall, the safety profile in patients with IgA was also in keeping with the overall trial population.

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So in summary, we've got now clear data in the IgA patients that finerenone exerts beneficial effects on GFR decline, proteinuria, increases the likelihood of achieving KDIGO targets in terms of proteinuria, and we have a better understanding of how it is exerting its beneficial effects through nonimmunological mechanisms that are distinct and complementary to our immune-related drugs.

That's it from me. Thanks very much for joining us today, and I look forward to future conversations and speaking soon.

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