TH-PO1253

Finerenone in patients with IgA nephropathy: A FIND-CKD analysis

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Background

- Immunoglobulin A nephropathy (IgAN) is the most common primary glomerular disorder worldwide and is a major cause of chronic kidney disease (CKD)[1,2]
- Remission of proteinuria is the key treatment goal in IgAN because proteinuria is strongly associated with the long-term risk of kidney failure; consequently, the Kidney Disease: Improving Global Outcomes (KDIGO) guidelines recommend a target of <0.5 g/day, ideally <0.3 g/day[3,4]
- In the FIND-CKD trial (NCT05047263), finerenone, a nonsteroidal mineralocorticoid receptor antagonist, was shown to reduce urinary albumin-to-creatinine ratio (UACR), attenuate estimated glomerular filtration rate (eGFR) decline, and reduce the risk of kidney and cardiovascular events in patients with nondiabetic CKD[5]
- This prespecified analysis of the FIND-CKD trial aimed to examine the efficacy and safety of finerenone in patients with IgAN and to explore its potential mechanisms in the kidney by analyzing blood and urine biomarkers

Methods

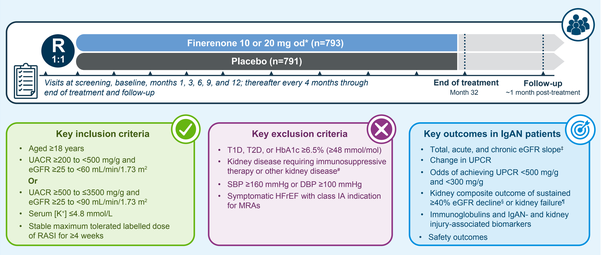
FIND-CKD study

- FIND-CKD was a double-blind, placebo-controlled trial that randomized eligible adults with CKD (eGFR 25 to <90 mL/min/1.73 m^2 and UACR ≥200–3500 mg/g) without diabetes 1:1 to receive finerenone (10 or 20 mg) or placebo[5]
- Study design, eligibility criteria, and key outcomes are shown in **Figure 1**

IgAN analyses

- CKD etiology, including IgAN, was categorized based on investigator-reported diagnoses, with most participants with IgAN having their diagnosis confirmed by biopsy
  - Oxford MEST-C classification was extracted from biopsy pathology reports, where available
- Biomarker analyses were performed in participants with biopsy-proven IgAN
  - Biomarkers were selected a priori based on their roles in IgAN pathophysiology or kidney damage. Serological markers were measured using Roche Cobas analyzers or by enzyme-linked immunosorbent assay; other biomarkers were measured using Olink ExploreHT in EDTA plasma or urine samples. Urine biomarker levels were creatinine-normalized

Figure 1. Study design



*For 10 mg od: eGFR ≥25 to <60 mL/min/1.73 m^2, for 20 mg od: eGFR ≥60 mL/min/1.73 m^2 #lupus nephritis or anti-neutrophil cytoplasmic antibody–associated vasculitis within 6 months prior to screening, or known autosomal recessive or dominant polycystic kidney disease; †total slope defined as the mean annual change in eGFR from baseline to month 32, acute slope defined as the mean change from baseline to month 3, chronic slope defined as the mean annual change from month 3 to the end of treatment; ‡sustained ≥40% eGFR decline from baseline over 4 weeks; ¶defined as an eGFR <15 mL/min/1.73 m^2, initiation of chronic dialysis, or kidney transplantation

DBP, diastolic blood pressure; eGFR, estimated glomerular filtration rate; HbA1c, glycated haemoglobin; HFrEF, heart failure with reduced ejection fraction; IgAN, immunoglobulin A nephropathy; [K⁺], potassium concentration; MRA, mineralocorticoid receptor antagonist; od, once daily; R, randomized; RASi, renin–angiotensin system inhibitor; SBP, systolic blood pressure; T1D, type 1 diabetes; T2D, type 2 diabetes; UACR, urinary albumin-to-creatinine ratio; UPCR, urinary protein-to-creatinine ratio

Results

- Of 1584 participants in FIND-CKD, 416 (26%) had an investigator-reported diagnosis of IgAN
  - Of these, 93% had a diagnosis confirmed by kidney biopsy

Baseline characteristics

- Baseline characteristics of participants with IgAN were generally balanced across treatment groups (**Table 1**)
- Mean age was 48 years, 45% were female, and 75% were Asian
- Mean eGFR was 49 mL/min/1.73 m^2 and median urinary protein-to-creatinine ratio (UPCR) was 1122 mg/g
- At baseline, 21% of participants were receiving a sodium-glucose co-transporter-2 inhibitor (SGLT-2i)

Table 1. Baseline characteristics of participants with IgAN

Characteristics	Overall (N=416)
Demographics	
Age, years, mean ± SD	47.9 ± 12.7
Sex, female, n (%)	189 (45.4)
Race,* n (%)	
Asian	310 (74.5)
Black	2 (0.5)
White	99 (23.8)
Other#	5 (1.2)
Medical evaluations and history	
BMI, kg/m^2, mean ± SD	26.2 ± 5.2
Systolic blood pressure, mmHg, mean ± SD	126.0 ± 12.9
Medical history, n (%)	
Hypertension	337 (81.0)
Atherosclerotic disease	20 (4.8)
Heart failure	3 (0.7)
Medication use, n (%)	
SGLT-2is	89 (21.4)
Diuretics	35 (8.4)
Kidney characteristics	
eGFR, mL/min/1.73 m^2, mean ± SD	49.5 ± 16.0
UPCR, mg/g, median (Q1–Q3)	1122.1 (807.9–1635.9)
IgAN etiology confirmed by biopsy, n (%)	387 (93.0)

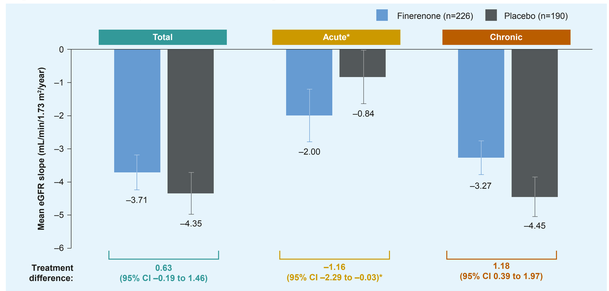
*Participant race was self-reported; #included American Indian or Alaska native, native Hawaiian or other Pacific islander, or multiple; the multiple-race category included participants who reported belonging to more than one racial group

BMI, body mass index; eGFR, estimated glomerular filtration rate; IgAN, immunoglobulin A nephropathy; SD, standard deviation; SGLT-2i, sodium-glucose co-transporter-2 inhibitor; UPCR, urinary protein-to-creatinine ratio

eGFR slope

- In participants with IgAN, finerenone attenuated eGFR decline over 32 months, with a total slope of –3.7 mL/min/1.73 m^2/year (95% confidence interval [CI] –4.2 to –3.2) in the finerenone group versus –4.3 mL/min/1.73 m^2/year (95% CI –5.0 to –3.7) in the placebo group (**Figure 2**)
- While finerenone resulted in greater acute reduction in eGFR in the first 3 months compared with placebo, thereafter, finerenone attenuated chronic slope by 1.2 mL/min/1.73 m^2/year (95% CI 0.4 to 2.0) versus placebo
- Effects on total, acute, and chronic slope were consistent in IgAN versus other etiologies of CKD (all *p*-interaction >0.05)

Figure 2. eGFR slope in patients with IgAN



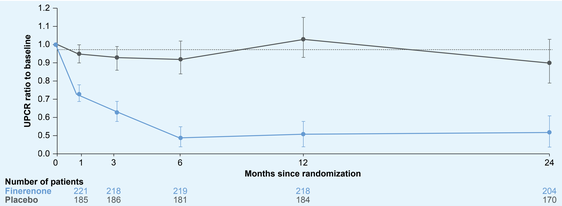
*Reported in mL/min/1.73 m^2/3 months

CI, confidence interval; eGFR, estimated glomerular filtration rate; IgAN, immunoglobulin A nephropathy

UPCR

- At month 12, finerenone reduced UPCR by 51% (95% CI 42–59%) versus placebo in participants with IgAN (**Figure 3**)
  - This effect was similar in those with other etiologies of CKD (*p*-interaction=0.07)
- Finerenone was associated with a nearly 7-fold increase in the odds of achieving a UPCR <500 mg/g at month 12 (odds ratio [OR] 6.82; 95% CI 3.71–13.14) (**Figure 4**)
  - Similarly increased odds were observed for achieving UPCR <300 mg/g (OR 6.68; 95% CI 3.27–14.85)

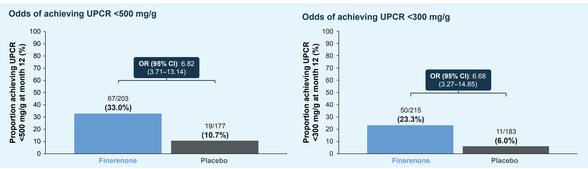
Figure 3. Change in UPCR over time



Error bars represent 95% CI

CI, confidence interval; UPCR, urinary protein-to-creatinine ratio

Figure 4. Odds of achieving UPCR <500 and <300 mg/g at month 12

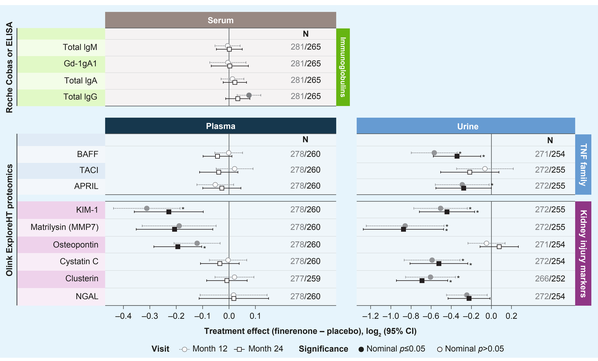


CI, confidence interval; OR, odds ratio; UPCR, urinary protein-to-creatinine ratio

Biomarkers

- Compared with placebo, finerenone did not alter circulating galactose-deficient immunoglobulin A1 (Gd-IgA1) or consistently modify total serum immunoglobulins A, G, or M levels (**Figure 5**)
  - Finerenone also did not affect plasma concentrations of a proliferation-inducing ligand (APRIL), B-cell activating factor (BAFF), and transmembrane activator and calcium-modulator and cyclophilin ligand interactor (TACI), although APRIL and BAFF were reduced in the urine
- Finerenone did reduce levels of several established biomarkers of kidney injury compared with placebo, particularly circulating and urinary kidney injury molecule-1 (KIM-1) and matrix metallopeptidase-7 (MMP7) (**Figure 5**)

Figure 5. IgAN and kidney injury biomarkers in participants with biopsy-confirmed IgAN



Filled markers, nominal *p*-value ≤0.05; asterisk (*) indicates significance (*p*≤0.05) after adjusting for multiple testing for all detectable proteins (plasma/serum: 2516; urine: 1030). Estimates were derived from linear mixed models adjusted for baseline biomarker levels, age, and sex. Urine data were creatinine-normalized. Analysis was restricted to participants with biopsy-proven IgAN

APRIL, a proliferation-inducing ligand; BAFF, B-cell activating factor; CI, confidence interval; ELISA, enzyme-linked immunosorbent assay; Gd-IgA1, galactose-deficient immunoglobulin A1; Ig, immunoglobulin; IgAN, immunoglobulin A nephropathy; KIM-1; kidney injury molecule-1; MMP7, matrix metallopeptidase-7; NGAL, neutrophil gelatinase-associated lipocalin; TACI, transmembrane activator and calcium-modulator and cyclophilin ligand interactor; TNF, tumor necrosis factor

Safety

- The safety profile of finerenone in the IgAN population was generally consistent with that of the overall trial population (**Table 2**)
  - While the incidence of hyperkalemia was higher with finerenone (18.1%) than with placebo (13.7%), the absolute incidence of hyperkalemia events leading to permanent discontinuation was low

Table 2. Safety outcomes

AE, n (%)	Finerenone (n=226)	Placebo (n=190)
AEs		
Any AE	155 (68.6)	119 (62.6)
Drug-related	59 (26.1)	36 (18.9)
Leading to permanent discontinuation	6 (2.7)	9 (4.7)
SAEs		
Any SAE*	48 (21.2)	33 (17.4)
Drug-related	0	1 (0.5)
Leading to permanent discontinuation	0	3 (1.6)
Leading to death	0	1 (0.5)
Hyperkalemia#		
Any hyperkalemia	41 (18.1)	26 (13.7)
Drug-related	28 (12.4)	18 (9.5)
Leading to permanent discontinuation	5 (2.2)	0
Serious‡	1 (0.4)	1 (0.5)
Leading to hospitalization	0	0

AEs that started or worsened after the first dose of study intervention up to 3 days after any temporary or permanent interruption of study intervention were considered treatment-emergent; *all but one SAE in the placebo group required hospitalization; #hyperkalemia includes AEs with MedDRA codes 10020646 (Hyperkalaemia) and 10005725 (Blood potassium increased); ‡both cases of serious hyperkalemia (n=1 in each arm) required hospitalization

AE, adverse event; MedDRA, Medical Dictionary for Regulatory Activities; SAE, serious adverse event

Conclusions

- In FIND-CKD, finerenone attenuated eGFR decline irrespective of whether patients had IgAN or a different form of CKD
- Finerenone reduced proteinuria in patients with IgAN and increased the likelihood of achieving proteinuria targets of <500 mg/g or <300 mg/g
- The safety profile of finerenone in patients with IgAN was consistent with that of the overall trial population, and the incidence of hyperkalemia leading to permanent discontinuation was low
- Finerenone did not modify plasma or serum markers of immunological disease activity but did reduce several kidney injury biomarkers implicated in tubulointerstitial inflammation and fibrosis,[6] suggesting its mechanism is likely distinct and complementary to those of immunological therapies for IgAN

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References

1. Stoneman S, *et al. JAMA* 2026;335:799–813
2. Neuen BL, *et al. JAMA* 2026;336:224–235
3. Bagchi S, *et al. Kidney Int Rep* 2021;6:1661–1668
4. Rovin BH, *et al. Kidney Int* 2025;108:S1–S71
5. Heerspink HJL, *et al. N Engl J Med* 2026;395:533–545
6. Ravindra KC, *et al. Toxicol Sci* 2024;201:206–215

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