NEWSLETTER 10

Finerenone and Reduced Sudden Death Across CKM Stages

A prespecified FINE-HEART analysis of 18,991 adults published in JACC

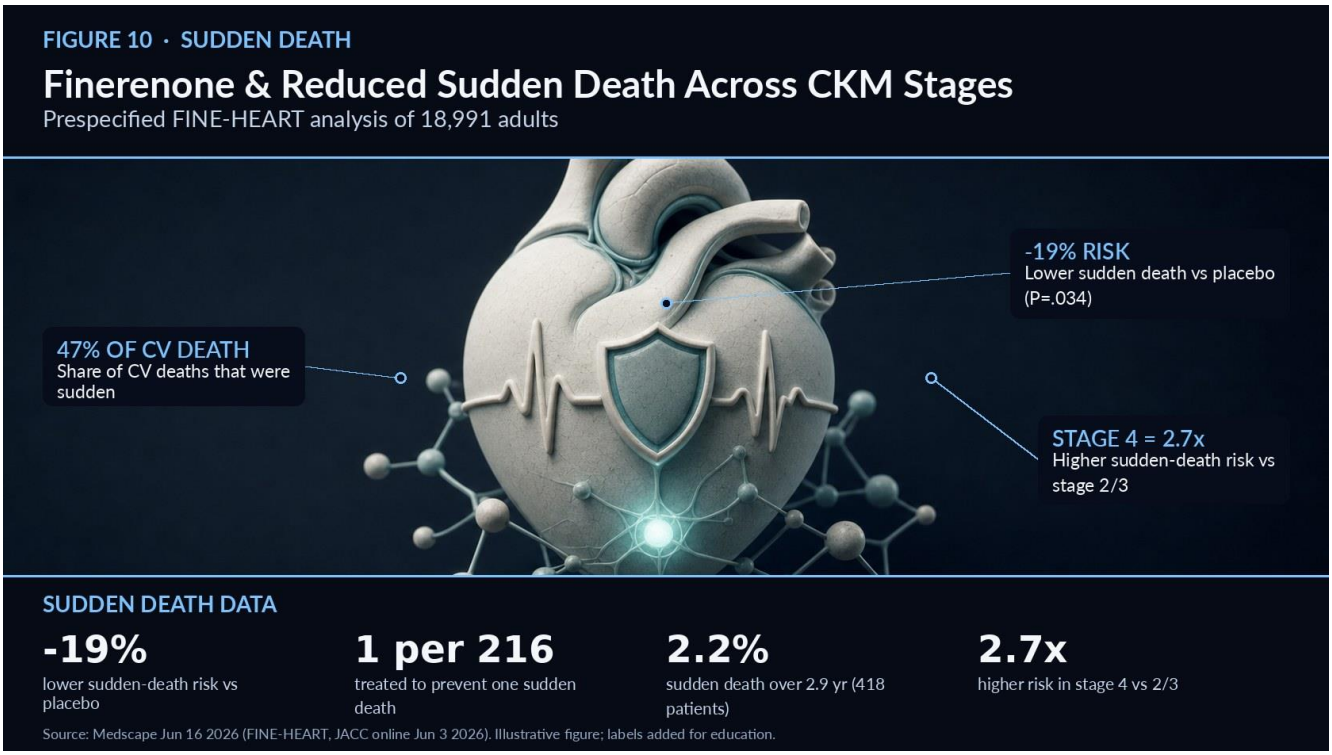

Figure 10. Finerenone and Reduced Sudden Death Across CKM Stages — mechanism overview (illustrative; labels added for education).

A prespecified analysis from the FINE-HEART programme, covering 18,991 adults, examined sudden death across the CKM spectrum and was published online June 3, 2026 in the *Journal of the American College of Cardiology*, with lead author Alberto Foà.[1] Sudden death is a particularly feared outcome in cardiometabolic disease, and this analysis is among the first to quantify it stage by stage.

The burden proved substantial. Over a median 2.9 years, sudden death occurred in 418 patients (2.2%), accounting for 47% of all cardiovascular deaths—nearly half.[1] That fraction underscores how much of the cardiovascular mortality in CKM is potentially arrhythmic and abrupt.

Finerenone meaningfully reduced this risk. It was associated with a 19% lower risk of sudden death versus placebo (P = .034), translating into one sudden death prevented for every 216 patients treated.[1] While the number needed to treat is large, the outcome prevented is catastrophic and otherwise difficult to anticipate.

Risk was strongly stage-dependent. Patients with CKM Stage 4 had a 2.7-fold higher risk of sudden death than those with Stage 2 or 3 (P < .001), reinforcing the value of staging for prognosis and for targeting intensive therapy.[1] Independent predictors of sudden death included older age, heart-failure history, atrial fibrillation, prior myocardial infarction, higher UACR, lower systolic blood pressure, and reduced kidney function (all P < .05)—a profile most clinicians will recognise immediately.[1]

The stage-dependence of sudden death is clinically actionable. Knowing that Stage 4 patients face a 2.7-fold higher risk than those at Stage 2 or 3 helps target the most intensive monitoring and therapy to those who stand

to gain most.[1] The recognisable predictor profile—older age, prior heart failure or myocardial infarction, atrial fibrillation, higher UACR, lower systolic pressure, and reduced kidney function—maps neatly onto data most clinics already collect, so risk can be flagged without new tests.[1]

Practical dosing mirrored the trials: finerenone was given once daily, starting at 10–20 mg and titrated to 20–40 mg by eGFR, with the analysis excluding the earliest and most advanced CKM stages.[1] Corroborating pooled datasets—including the INFINITY analysis and the *Lancet* CKD pooled analysis—support finerenone's consistent cardiovascular and kidney benefit, positioning sudden-death reduction as one more reason to consider it across appropriate CKM patients.[3,4]

References

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