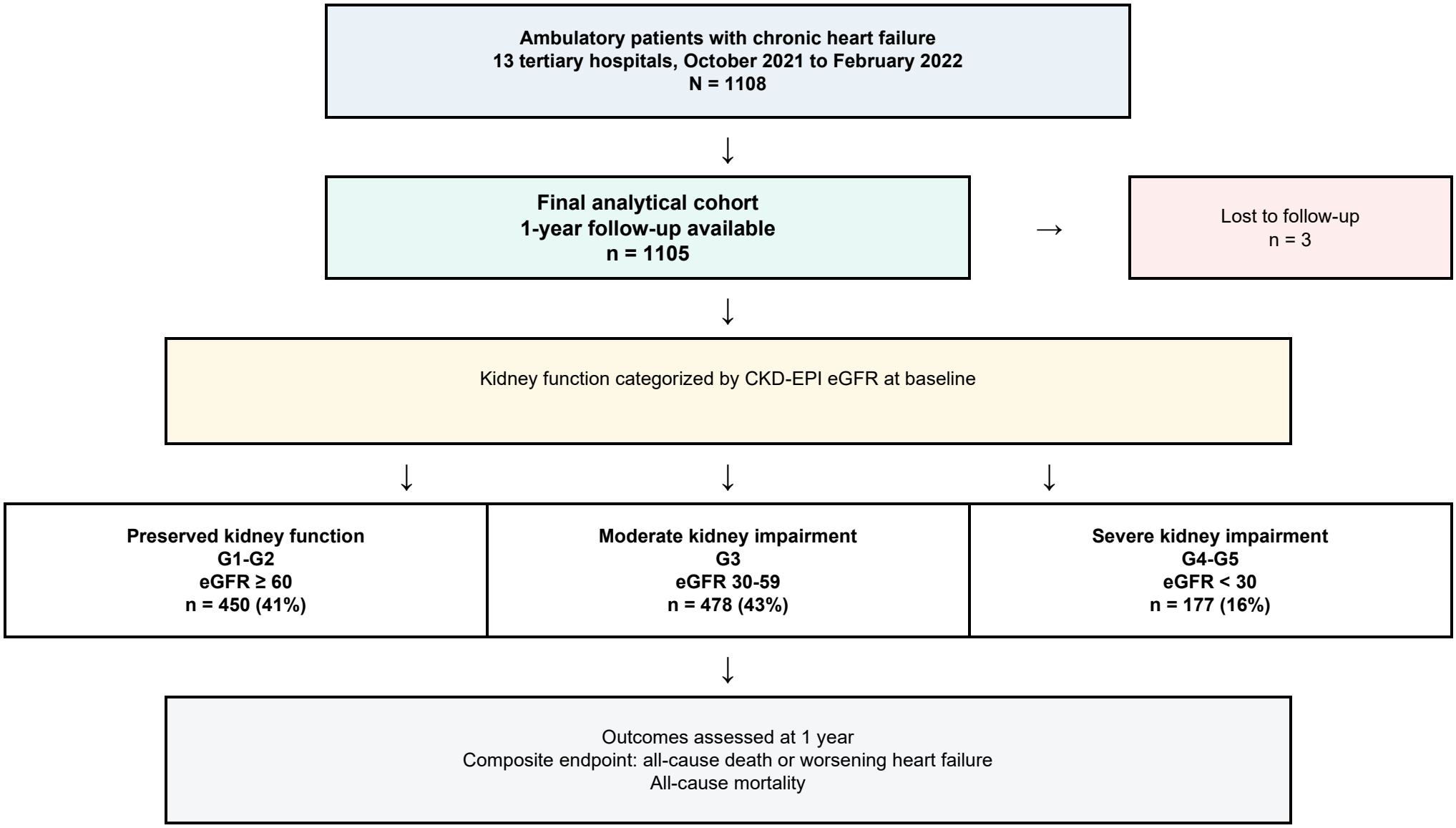
Figure S1. STROBE flow diagram: study population.



CKD-EPI, Chronic Kidney Disease Epidemiology Collaboration; eGFR, estimated glomerular filtration rate; WHF, worsening heart failure.

Table S1. Guideline-directed medical therapy across eGFR categories, stratified by HF phenotype

Therapy	eGFR ≥ 60	eGFR 30–59	eGFR < 30
HFrEF / HFmrEF (LVEF < 50%)			
RASi	302/315 (96)	253/289 (88)	58/83 (70)
MRA	254/315 (81)	185/289 (64)	35/83 (42)
SGLT2i	232/313 (74)	187/284 (66)	47/82 (57)
Beta-blocker	288/315 (91)	239/289 (83)	71/83 (86)
HFpEF (LVEF ≥ 50%)			
RASi	89/135 (66)	100/189 (53)	39/94 (41)
MRA	59/135 (44)	69/189 (37)	19/94 (20)
SGLT2i	51/135 (38)	80/189 (42)	35/93 (38)
Beta-blocker	96/135 (71)	118/189 (62)	60/94 (64)

eGFR, estimated glomerular filtration rate in mL/min/1.73 m²; HFrEF, heart failure with reduced ejection fraction; HFmrEF, heart failure with mildly reduced ejection fraction; HFpEF, heart failure with preserved ejection fraction; LVEF, left ventricular ejection fraction; RASi, renin-angiotensin system inhibitor; MRA, mineralocorticoid receptor antagonist; SGLT2i, sodium-glucose cotransporter 2 inhibitor.

The data are expressed as n/N (%).

Table S2. Sequential Cox regression models for the association between estimated glomerular filtration rate (per 10 mL/min/1.73 m² decrement) and 1-year outcomes

Model (covariates entered)	Composite endpoint		All-cause mortality	
	Full cohort	Complete-case (n = 946)	Full cohort	Complete-case (n = 946)
Model 1 unadjusted	1.26 (1.19-1.34) *P* < .001	1.27 (1.20-1.35) *P* < .001	1.39 (1.28-1.51) *P* < .001	1.40 (1.28-1.54) *P* < .001
Model 2 + age, sex	1.21 (1.14-1.29) *P* < .001	1.22 (1.14-1.31) *P* < .001	1.32 (1.20-1.45) *P* < .001	1.34 (1.21-1.49) *P* < .001
Model 3 + comorbidities, LVEF[a]	1.21 (1.13-1.29) *P* < .001	1.22 (1.13-1.31) *P* < .001	1.35 (1.22-1.49) *P* < .001	1.37 (1.22-1.53) *P* < .001
Model 4 + congestion & severity[b]	1.05 (0.97-1.13) *P* = .26	1.05 (0.97-1.13) *P* = .26	1.13 (1.00-1.27) *P* = .052	1.13 (1.00-1.27) *P* = .052

CA125, carbohydrate antigen 125; CI, confidence interval; eGFR, estimated glomerular filtration rate; HR, hazard ratio; LVEF, left ventricular ejection fraction; NT-proBNP, N-terminal pro–B-type natriuretic peptide; NYHA, New York Heart Association.

Values are hazard ratios (95% confidence intervals). eGFR was modelled as a continuous linear term, expressed per 10 mL/min/1.73 m² decrease. Models were fitted with Cox proportional-hazards regression (Breslow method for ties). The complete-case columns show models 1-3 refitted in the 946 patients with complete data for all model 4 covariates, to confirm that the attenuation reflected covariate adjustment rather than differential missingness.

[a] Comorbidities: diabetes mellitus, atrial fibrillation, hypertension, valvular heart disease, and chronic obstructive pulmonary disease.

[b] Congestion and severity markers: NYHA functional class, composite congestion score, NT-proBNP, CA125, hemoglobin, and furosemide-equivalent dose.

Table S3. Sensitivity analyses of the associations of eGFR and baseline furosemide-equivalent dose with 1-year outcomes

A. Association between eGFR and each endpoint definition

Endpoint definition	Events, No.	Unadjusted model	Fully adjusted model
Primary composite endpoint: all-cause death or worsening HF	266	1.26 (1.20-1.34) *P* < .001	1.04 (0.96-1.13) *P* = .33
Composite endpoint excluding ambulatory intravenous-diuretic episodes: death, HF hospitalization, or urgent visits	226	1.28 (1.20-1.36) *P* < .001	1.03 (0.94-1.12) *P* = .50
All-cause mortality	143	1.39 (1.28-1.51) *P* < .001	1.12 (0.99-1.26) *P* = 0.07*

B. Association between baseline furosemide-equivalent dose and outcomes in the fully adjusted model

Outcome	Events, No.	Fully adjusted model: furosemide-equivalent dose per mg/d
Primary composite endpoint	266	1.008 (1.006-1.011) *P* < .001
All-cause mortality	143	1.009 (1.005-1.012) *P* < .001

C. Association between eGFR in fully adjusted models with vs without furosemide-equivalent dose

Outcome and model specification	Events, No.	Furosemide-equivalent dose included	Fully adjusted model: eGFR per 10 mL/min/1.73 m² decrease
Primary composite endpoint	266	Yes	1.04 (0.96-1.13) *P* = 0.33
Primary composite endpoint	266	No	1.08 (1.00-1.17) *P* = .04
All-cause mortality	143	Yes	1.12 (0.99-1.26) *P* = .07

All-cause mortality	143	No	1.21 (1.08-1.35) *P* < .001

CA125, carbohydrate antigen 125; COPD, chronic obstructive pulmonary disease; eGFR, estimated glomerular filtration rate; HF, heart failure; HR, hazard ratio; LVEF, left ventricular ejection fraction; NT-proBNP, N-terminal pro–B-type natriuretic peptide; NYHA, New York Heart Association.

Values are hazard ratios (95% confidence intervals) from Cox proportional-hazards models.

The fully adjusted model included age, sex, valvular heart disease, chronic obstructive pulmonary disease, hypertension, atrial fibrillation, left ventricular ejection fraction, NT-proBNP, CA125, hemoglobin, NYHA functional class, composite congestion score, and furosemide-equivalent dose, unless otherwise stated.

*For all-cause mortality, the linear eGFR term is shown. In the primary fractional-polynomial model, eGFR was independently and nonlinearly associated with mortality ($P = .013$), consistent with a disproportionate risk concentrated at very low eGFR values that may not be fully captured by a linear term.

Table S4. Per-covariate associations in the final multivariable fractional-polynomial (MFP) models, with Benjamini-Hochberg correction

Covariate	Raw *P*	BH-FDR
Composite endpoint (all-cause death or worsening heart failure)		
NT-proBNP	**< .001**	**< .001**
Hemoglobin	**< .001**	**< .001**
Furosemide dose	**< .001**[a]	**< .001**
Atrial fibrillation	**.005**	**.020**
Congestion score	.020	.064
Male sex	.047	.123
CA125	.054	.123
NYHA class	.090[a]	.167
Hypertension	.094	.167
MRA	.357	.571
COPD	.438	.637
SGLT2i	.592	.789
Prior HF hospitalization	.719	.885
Valvular disease	.840	.922
RASi/ARNI	.870	.922
Beta-blocker	.922	.922
All-cause mortality		
Hemoglobin	**< .001**	**.002**
CA125	**.002**	**.015**

NT-proBNP	**.006**	**.030**
Male sex	**.011**	**.033**
Furosemide dose	**.011**	**.033**
eGFR	**.013**	**.033**
Atrial fibrillation	.027	.058
COPD	.038	.071
RASi/ARNI	.113	.188
Prior HF hospitalization	.203	.305
Valvular disease	.224	.305
Beta-blocker	.430	.519
NYHA class	.450[a]	.519
Hypertension	.750	.804
MRA	.842	.842

BH-FDR, Benjamini-Hochberg false-discovery-rate-adjusted *P* value; CA125, carbohydrate antigen 125; COPD, chronic obstructive pulmonary disease; eGFR, estimated glomerular filtration rate; MFP, multivariable fractional polynomial; MRA, mineralocorticoid receptor antagonist; NT-proBNP, N-terminal pro–B-type natriuretic peptide; NYHA, New York Heart Association; RASi, renin-angiotensin system inhibitor; SGLT2i, sodium–glucose cotransporter-2 inhibitor.

[a] *Joint 2-degrees-of-freedom Wald test.*

For each covariate, the raw P value is the global Wald test of its term(s) in the final MFP Cox model (the same model used to derive figures 3 and 4): a single z test for covariates entered with 1 term, and a joint test of both terms for variables modeled with a second-degree fractional polynomial (NT-proBNP, CA125, hemoglobin, eGFR and furosemide dose, as applicable) or with 2 indicator levels (NYHA class). The Benjamini-Hochberg false-discovery-rate correction was applied to these raw *P* values within each model. Both models were fitted in the MFP complete-case sample (n = 850). Bold rows are significant after BH-FDR. Only covariates retained in the final MFP model are shown; eGFR was eliminated by the selection procedure for the composite endpoint and therefore does not appear in that panel.