Supplemental Table 1: General linear mixed effects regression model of the systolic blood pressure (exposure) and eGFR.

Variable	Estimate	P
Intercept	15.54	<0.001
SBP	-0.02	<0.001
Time1	-0.56	0.58
Time2	-99.80	0.09
Time3	164.98	0.06
Time4	-81.83	0.01
SBP x time1	0.14	0.004
SBP x time2	-6.97	0.02
SBP x time3	10.22	0.02
SBP x time4	-3.50	0.04
Hemoglobin	-0.04	<0.001
Bicarbonate	-0.06	0.003
Phosphate	5.27	<0.001
Potassium	0.05	0.26
Albumin	0.04	0.02
Proteinuria		
None	0.03	0.86
Minimal	0.15	0.34
Moderate	referent	
Severe	-0.08	0.64
ACE/ARB	1.18	<0.001
Male	-0.71	0.02
Age	0.03	0.02
CAD	0.42	0.26
CHF	1.05	0.006
PVD	0.26	0.52
Diabetes	0.48	0.23
Malignancy	0.59	0.17
Cause of CKD		
Diabetes	-1.19	0.02
Ischemia	0.27	0.55
Glomerulonephritis	0.51	0.22
Other		

SBP systolic blood pressure, ACE angiotensin converting enzyme inhibitor, ARB angiotensinogen receptor blocker, CAD coronary artery disease, CKD congestive heart failure, PVD peripheral vascular disease, CKD chronic kidney disease. The time values (labelled time1-time4) represent splines of time at values of 1, 48, 168, 411 and 952 days of follow up.

Supplemental Table 2: General linear mixed effects regression model of the diastolic blood pressure (exposure) and eGFR.

Variable	Estimate	P
Intercept	15.34	<0.001
DBP	-0.06	<0.001
Time1	-0.06	0.95
Time2	-124.44	0.04
Time3	200.91	0.02
Time4	-93.89	0.005
DBP x time1	0.32	<0.001
DBP x time2	13.08	0.01
DBP x time3	18.49	0.02
DBP x time4	-5.43	0.06
Hemoglobin	-0.03	<0.001
Bicarbonate	-0.06	<0.001
Phosphate	5.24	<0.001
Potassium	0.04	0.29
Albumin	0.04	0.01
Proteinuria		
None	0.03	0.86
Minimal	0.14	0.37
Moderate	Referent	
Severe	-0.08	0.67
ACE/ARB	1.16	<0.001
Male	-0.67	0.03
Age	0.02	0.07
CAD	0.44	0.23
CHF	1.03	0.007
PVD	0.30	0.46
Diabetes	0.53	0.18
Malignancy	0.62	0.15
Cause of CKD		
Diabetes	-1.20	0.01
Ischemia	0.28	0.54
Glomerulonephritis	0.54	0.19
Other	Referent	

DBP diastolic blood pressure, ACE angiotensin converting enzyme inhibitor, ARB angiotensinogen receptor blocker, CAD coronary artery disease, CKD congestive heart failure, PVD peripheral vascular disease, CKD chronic kidney disease. The time values (labelled time1-time4) represent splines of time at values of 1, 48, 168, 411 and 952 days of follow up.

Supplemental Table 3: General linear mixed effects regression model of the pulse pressure (exposure) and eGFR.

Variable	Estimate	P
Intercept	15.69	<0.001
PP	-0.01	0.24
Time1	-1.19	0.24
Time2	-68.92	0.24
Time3	119.77	0.18
Time4	-66.39	0.05
PP x time1	0.05	0.34
PP x time2	-3.56	0.26
PP x time3	5.51	0.24
PP x time4	-2.27	0.20
Hemoglobin	-0.04	<0.001
Bicarbonate	-0.06	<0.001
Phosphate	5.30	<0.001
Potassium	0.04	0.29
Albumin	0.03	0.02
Proteinuria		
None	0.17	0.82
Minimal	0.18	0.26
Moderate	Referent	
Severe	0.11	0.56
ACE/ARB	1.18	<0.001
Male	-0.70	0.02
Age	0.03	0.02
CAD	0.42	0.26
CHF	1.01	0.01
PVD	0.26	0.51
Diabetes	0.47	0.24
Malignancy	0.60	0.17
Cause of CKD		
Diabetes	-1.15	0.02
Ischemia	0.27	0.55
Glomerulonephritis	0.53	0.20
Other	Referent	

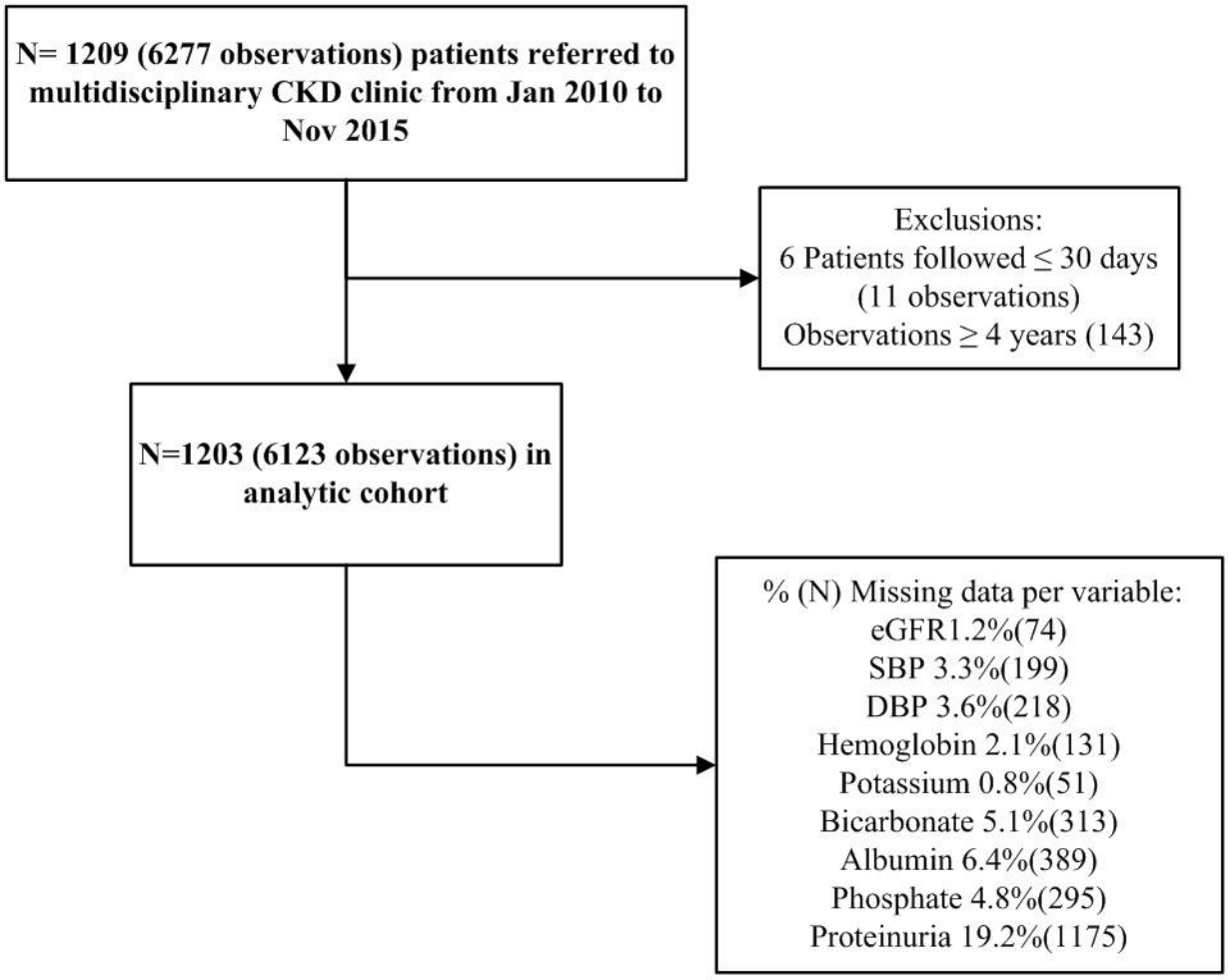
PP pulse pressure, ACE angiotensin converting enzyme inhibitor, ARB angiotensinogen receptor blocker, CAD coronary artery disease, CKD congestive heart failure, PVD peripheral vascular disease, CKD chronic kidney disease. The time values (labelled time1-time4) represent splines of time at values of 1, 48, 168, 411 and 952 days of follow up.

Supplementary Table 4: Results of sensitivity analyses for non-linearity and changes over time on the association of individual blood pressure components and eGFR.

	Non-linearity		Change over time	
Model	**Difference in -2 log likelihood between models ***	**P value**	**Difference in -2 log likelihood between models #**	**P value**
SBP:				
Full model (N=1203)	40.1	<0.001	27.1	<0.001
ESKD/death excluded (N=681)	36.3	<0.001	11.1	0.03
First eGFR value excluded (N=1203)	30.2	<0.001	10.6	0.03
DBP:				
Full model (N=1203)	47.3	<0.001	31.4	<0.001
ESKD/death excluded (N=681)	46.8	<0.001	29.6	<0.001
First eGFR value excluded (N=1203)	29.4	<0.001	12.0	0.02
PP:				
Full model (N=1203)	32.4	<0.001	3.4	0.49
ESKD/death excluded (N=681)	28.5	<0.001	2.1	0.72
First eGFR value excluded (N=1203)	30.1	<0.001	2.8	0.60

All models adjusted for age, sex, cause of chronic kidney disease, malignancy, coronary artery disease, congestive heart failure, peripheral vascular disease, diabetes mellitus at baseline, ACE/ARB use and time-updated measures of hemoglobin, bicarbonate, phosphate, potassium, albumin and proteinuria. * degrees of freedom =6, # degrees of freedom =4. SBP systolic blood pressure ESKD end stage kidney disease eGFR estimated glomerular filtration rate DBP diastolic blood pressure PP pulse pressure

Supplemental Figure 1: Study cohort.



Additional statistical methods:

Time-varying associations of blood pressure and eGFR

The time-varying associations between continuous eGFR and each of SBP, DBP and PP were analyzed using separate general linear mixed effects regression models, estimated using Restricted Maximum Likelihood (REML)(20). Fixed effects of interest in each model were: time, defined in years since the first clinic visit; continuous measures of SBP, DBP and PP; and their interactions with time. To allow for non-linear trends in eGFR, time was modelled using restricted cubic splines with 5 knots fitted at the 5^{th}, 25^{th}, 50^{th}, 75^{th} and 95^{th} percentiles of time corresponding to values of 1, 48, 168, 411, and 952 days, respectively. Statistical significance testing for non-linearity versus linearity were conducted using likelihood ratio tests. Denominator degrees of freedom were calculated using the Kenward Roger method(20). Random coefficients were specified for subject and time to account for correlations in repeated measures on the same subjects. Repeat measures of each blood pressure component were tested for associations of changes over time with repeat measures of eGFR using likelihood ratio tests. All analyses were adjusted for age at cohort entry, gender, and baseline comorbidities (coronary artery disease, congestive heart failure, malignancy, hypertension, PVD, diabetes,), as well as repeat measures of hemoglobin, albumin, phosphate, potassium, bicarbonate and proteinuria. All continuous covariates were grand mean centred prior to analysis. Multi-collinearity among baseline predictors were assessed using a variable clustering algorithm with a cut point for proportion of variation explained set at 70% (22). To avoid exclusion of subjects due to missing covariates, multiple imputation was performed prior to analysis using a Markov Chain Monte Carlo algorithm (the data augmentation algorithm) (21). Ten multiple

imputation datasets were generated with all variables included in analytical models specified as predictors in the multiple imputation model. Analyses were carried out for each multiple imputation dataset and pooled across datasets using Rubin's rules (23).

To illustrate the associations of individual BP components and eGFR over time, modelled eGFR trajectories were plotted with blood pressure variables set at the 5th, 50th and 95th percentiles (SBP:105, 140, 170, DBP:50, 70, 90, PP: 35, 60, 100)(20). All remaining continuous covariates were set to their median values, while categorical covariates were set to their mode. In a sensitivity analysis, models were repeated excluding observations from patients who reached ESKD prior to a ≥30% decline in GFR or who died (n=681) for the linear mixed models. This was to avoid bias in our estimates as a number of participants may have been referred to clinic immediately prior to ESKD initiation.

Blood pressure indices and the risk of a GFR decline > 30%

The association of SBP, DBP and PP with time to eGFR decline ≥ 30% was examined using Cox proportional hazards models for all participants (N=1203). For simplicity of interpretation, blood pressure components were categorized at approximately the 5th, 25th, 50th, 75th and 95th percentiles and modelled at baseline upon cohort entry and with time-updated values. The interval considered as a normal range was classified as the reference category. Both crude and adjusted hazard ratios were estimated, adjusting for the same covariates as in the linear mixed models. The proportional hazards assumption was checked by examining Schoenfeld residuals(24). Patients were censored at study end (n=287), at ESKD (n=540), at death (n=141), at loss to follow-up (n=20), if they moved out

of province (n=33) or received a pre-emptive transplant (n=8). To assess the potential influence of informative censoring, we employed the method by Allison where additional models were created censoring the competing event (ESKD or death) i.) at the time of the event of interest (eGFR decline≥30%) or ii.) the longest event time (4 years) and comparing the two(24).