Kidney Medicine

Population-Level Risk Factors for Kidney Outcomes in IgA Nephropathy: The CURE-CKD Registry

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Rationale & Objective: Although IgA nephropathy (IgAN) therapies are advancing quickly, therapeutic interventions are hampered by a lack of kidney disease identification and risk assessment. The study aim was to use population-level data from health systems to identify IgAN and assess risks.

Study Design: A longitudinal and real-world cohort study.

Setting & Participants: Electronic health record data for patients ≥18 years old with IgAN at Providence and University of California Los Angeles health systems during 2016-2022.

Predictors: Health insurance and care utilization along with age, gender, race, ethnicity, estimated glomerular filtration rate (eGFR), urine albumin/creatinine ratio (UACR) or urine protein/creatinine ratio (UPCR), diabetes, hypertension, and medications.

Outcomes: Time to first major adverse kidney event (MAKE): ≥40% eGFR decline; eGFR <15 mL/min/1.73 m2; administrative codes for kidney failure, dialysis, or transplant; and death.

Analytical Approach: Kaplan-Meier survival curves and Cox proportional hazards models.

Results: Patients with IgAN (n = 2,571) were 50% (n = 1,277) women and 58 ± 18 (mean ± SD) years old. At baseline, eGFR was 78 ± 27 mL/min/1.73 m^2 (chronic kidney disease epidemiologic 2021 equation); median UACR and UPCR were 166 (interquartile range 25-795) mg/g and 0.7 (0.2-1.8) g/g, respectively, among those with baseline measurements (n = 669). MAKE occurred in 22% of the cohort by 3 years. In Cox proportional hazards models, MAKE was predicted by noncommercial (Medicare or Medicaid) health insurance, hospitalization, more frequent outpatient encounters, lower eGFR, and a higher UACR or UPCR.

Limitations: Missingness, miscoding, and retrospective data.

Conclusions: Substantial loss of kidney function, kidney failure, and death were common events over a short period of time in patients with IgAN. Within health system populations, noncommercial health insurance and greater care utilization augmented risk prediction and could help to identify those who may benefit from closer monitoring and implementation of therapeutic interventions.

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IgA nephropathy (IgAN) is a glomerular disease found in populations worldwide.[1] Incidence estimates for IgAN vary from 0.76 to 2.5/100,000 persons annually, with higher rates observed in the Asia-Pacific region than elsewhere.[2] For example, the point prevalence for IgAN in France was recently reported as 3.1/100,000 versus 4.5/100,000 in Japan and 10.5/100,000 in Australia.[3,4] IgAN is traditionally described as causing kidney failure in 25% to 50% of cases over 10 to 30 years.[5] However, more recent data predict higher lifetime risk, such that almost all patients with IgAN are expected to progress to kidney failure.[6] Risk prediction is central to clinical decisions that depend on prognostication. Low estimated glomerular filtration rate (eGFR), albuminuria or proteinuria, and histologic features are established clinical predictors of major adverse kidney events (MAKE) in IgAN.[7-13] However, population-level characteristics such as attributes related to health insurance and care utilization have not been previously evaluated for risk prediction in IgAN.

IgAN therapies have advanced quickly, with several showing efficacy and safety in clinical trials of immunomodulation and a dual endothelin-angiotensin receptor antagonist.[14-17] However, deploying interventions is hampered by low rates of kidney disease identification and risk assessment.[18,19] Studies of real-world cohorts provide health information at a population level to identify disparities and gaps in care that can be addressed to improve clinical outcomes. The Center for Kidney Disease Research, Education, and Hope (CURE-CKD) Registry provides curated electronic health record (EHR) data from the Providence and University of California Los Angeles (UCLA) health systems.[20,21] We hypothesized that types of health insurance and care utilization, along with demographic and clinical variables available in EHRs, would augment risk prediction for IgAN within a large population. The study's aim was to identify IgAN and assess risks of MAKE based on population-level data from CURE-CKD.

METHODS

Study Design, Participants, and Setting

This real-world cohort study identified participants from EHR data in the CURE-CKD Registry. Demographics, clinical characteristics, and prescription medications were

PLAIN LANGUAGE SUMMARY

IgA nephropathy therapies have advanced quickly. However, therapeutic interventions are hampered by lack of disease identification and risk assessment. We identified patients with IgA nephropathy at 2 United States health systems and assessed predictors of risk for major adverse kidney events (major adverse kidney event [MAKE]—substantial loss of kidney function, kidney failure, or death). More than one in 5 patients experienced MAKE by 3 years. In addition to demographic and clinical predictors, MAKE was predicted by noncommercial health insurance, hospitalization, and more frequent outpatient encounters. A population health approach within health systems could improve outcomes by identification of IgA nephropathy and assessment of health insurance status and care utilization to help risk stratify patients for closer monitoring and implementation of therapeutic interventions.

obtained for patients ≥18 years old with IgAN in 2016 through 2022. International classification of diseases (ICD) 10 codes (Table S1) were used to identify IgAN. Exclusion criteria were kidney failure (eGFR <15 mL/min/1.73 m^2 or ICD 10 codes indicative of kidney failure, dialysis, or kidney transplant; Table S1) and missing eGFR data. The sample size was determined by the number of patients with IgAN meeting these criteria (Fig 1). Diabetes and hypertension were identified by established CURE-CKD criteria.[20,21] All-cause death was ascertained by the Social Security Death Index for patients in the Providence health system and from the California State Death Index for patients at UCLA Health. A 1 year look-back before IgAN identification was used as the baseline period for EHR data collection. Because of missingness, the look-back period for albumin/creatinine ratio (UACR) or urine protein/creatinine ratio (UPCR) was extended to 3 years to increase data capture. Providence and the UCLA Health institutional review boards approved the study with a determination that written informed consent was not required for analyses of the limited dataset. The study was conducted according to reporting of observational studies in epidemiology guidelines.[22]

Statistical Analyses

Univariate statistics described the study population at baseline. Frequency distributions were applied for categorical variables, mean ± standard deviation for normally distributed continuous variables, and median and interquartile range (IQR) for skewed variables. The Chronic Kidney Disease Epidemiologic 2021 equation was used to calculate eGFR from serum creatinine.[23] Medications were considered as used if an active prescription was present in the EHR within the baseline period. The index time for study observations began at the time of IgAN identification and continued to a MAKE event or last encounter. Time-to-event analysis evaluated the first event in a primary composite MAKE outcome (≥40% eGFR decline from baseline, eGFR <15 mL/min/1.73 m^2, ICD 10 codes indicative of kidney failure, dialysis or kidney transplant, and death; Table S1). Kaplan-Meier survival curves were analyzed for the primary composite MAKE outcome and individual components.

The main Cox proportional hazards models for the primary composite MAKE outcome examined health insurance and care utilization, demographic, and clinical variables in the full cohort. Primary health insurance type (noncommercial—Medicaid, Medicare, and unknown insurance versus commercial), hospitalization (yes or no) and outpatient visits (number) in the 1 year baseline were included as variables. Demographics were categorized as age (per decade), gender (women or men), and racial identity (Asian or non-White versus White, non-Hispanic) because IgAN is more common in these groups than the reference group.[2,4,5] Medication variables (yes or no) were angiotensin-converting enzyme (ACE) inhibitors, angiotensin receptor blockers (ARBs), corticosteroids, and other immunomodulators (biologics, calcineurin inhibitors, cytotoxic agents, mammalian target of rapamycin inhibitors, corticotropin agents, and pyrimidine synthesis inhibitors). Clinical variables were diabetes and hypertension (yes or no) and eGFR (per 10 mL/min/1.73 m^2). Because of missingness, UACR or urine UPCR (above or below medians) were included in a secondary model for the subset with these measurements. Alpha was selected as < 0.05 a priori to define statistical significance. Statistical analyses were performed in R (v4.2.2) with the survival (v3.5-5) and prodlim (2023.03.3) packages.[24-26]

RESULTS

Study Participant Characteristics

The Providence health system cared for 74% (1,905/2,571) of the full cohort with IgAN, whereas 26% (666/2,571) were at UCLA Health (Table 1). Hospitalization occurred in 25% (646/2,571) of study participants during the one-year baseline period. The median (IQR) number of outpatient visits during the baseline period was 15 (7-29). The proportion of patients with commercial health insurance as the primary payer was 53% (1,354/2,571) in the full cohort.

Patients with IgAN were 50% (1,277/2,571) women and 58 ± 18 (mean ± SD) years old at baseline (Table 1). Asian race was 13% (325/2,571) and 22% (577/2,571) were non-White race overall. At baseline, the mean eGFR was 78 ± 27 mL/min/1.73 m^2 (n = 2,571) and median (IQR) UACR and UPCR were 166 (25-795) mg/g and 0.7 (0.2-1.8) g/g (n = 669), respectively. Diabetes was present in 28% (708/2,571), whereas 75% (1,919/2,571) had hypertension. ACE inhibitors or ARBs were used by 49% (1,250/2,571). Corticosteroids and other immunomodulators were used by 39% (1,006/2,571) and 3% (77/2,571),

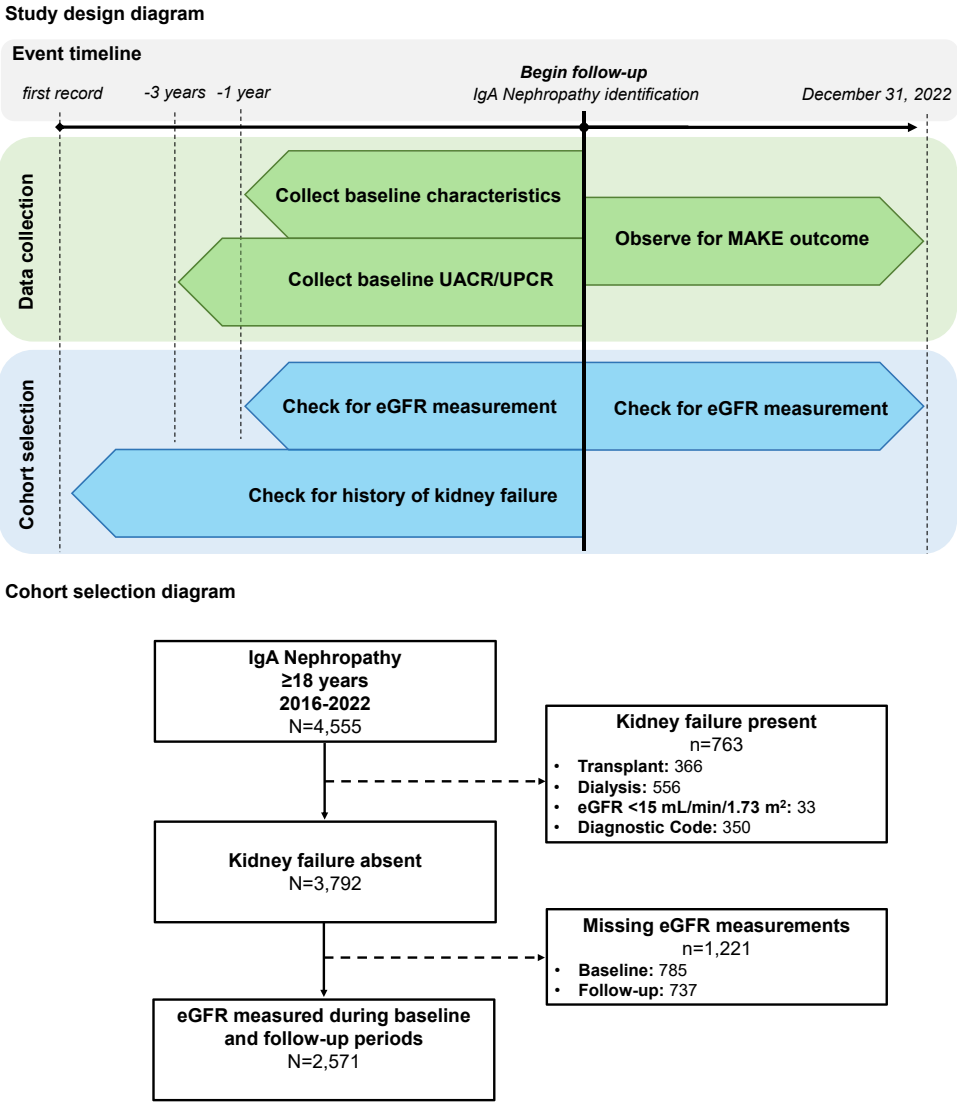


Figure 1. STROBE diagram for IgAN cohort selection. IgAN, IgA nephropathy; eGFR, estimated glomerular filtration rate; UACR, urine albumin/creatinine ratio; UPCR, urine protein creatinine ratio; MAKE, major adverse kidney events.

respectively. Use of sodium-glucose cotransporter 2 inhibitors was documented in 1.6% of patients (42/2,571). Patients with UACR or UPCR measures differed from those without these measures by distributions of race and age, higher prevalence of diabetes or hypertension, more common use of ACE inhibitors or ARBs, other immunomodulators, and a lower eGFR (Table 1).

Rates and Predictors of MAKEs Over Time

Based on the Kaplan-Meier survival analysis, the primary MAKE outcome occurred in 22% (Fig 2A) at 3 years. Of patients who experienced a MAKE (n = 536), events included: ≥40% eGFR decline in 61% (327/536); kidney failure with eGFR <15 mL/min/1.73 m^2 in 20% (108/536), dialysis in 4% (23/536), or transplant in 3% (17/536); and all-cause death in 11% (61/536; Fig 2B; Table 2,). The median (IQR) follow-up time was 2.8 (1.3-4.4) years.

In the main adjusted Cox model of the full cohort, predictors with significantly increased hazard of MAKE were hospitalization (hazard ratio [HR] = 2.59; 95% CI, 2.14-3.12), other immunomodulators (HR = 1.59; 95% CI, 1.09-2.30), noncommercial health insurance (HR = 1.46; 95% CI, 1.19-1.80), lower eGFR per 10 mL/min/1.73 m^2 (HR = 1.28; 95% CI, 1.24-1.33), diabetes (HR = 1.30; 95% CI, 1.07-1.57), ACE inhibitor or ARB use (HR = 1.26; 95% CI, 1.04-1.53), and outpatient visits (HR = 1.01; 95% CI, 1.00-1.03; Fig 3A; Table S2). Hypertension (HR = 1.33; 95% CI, 0.99-1.78) and women (HR = 0.86, 95% CI, 0.72-1.03) were borderline significant predictors.

MAKE occurred more often in those with levels above versus below the medians for UACR (166 mg/g) or UPCR (0.7 g/g), 33% (113/341) versus 20% (65/328), in the subset with baseline measurements (n = 669). In the secondary adjusted Cox model, including patients with baseline

Table 1. Baseline Characteristics of the Population With IgAN in the Years 2016-2022

	Full Cohort	Subset With UACR or UPCR	Subset Without UACR or UPCR
Patients, N (% of total)	2,571 (100.0)	669 (26.0)	1,902 (74.0)
Health care system, utilization, and insurance			
Hospitalization, n (%)	646 (25.1)	142 (21.2)	504 (26.5)
Outpatient visits, median (IQR)	15 (7-29)	18 (9-35)	14 (6-28)
Primary insurance, n (%)			
Commercial	1,354 (52.7)	392 (58.6)	962 (50.6)
Medicaid	236 (9.2)	45 (6.7)	191 (10.0)
Medicare	929 (36.1)	215 (32.1)	714 (37.5)
Unknown	51 (2.0)	16 (2.4)	35 (1.8)
System, n (%)			
Providence	1,905 (74.1)	513 (76.7)	1,392 (73.2)
UCLA Health	666 (25.9)	156 (23.3)	510 (26.8)
Demographics			
Gender, n (%)			
Men	1,294 (50.3)	356 (53.2)	938 (49.3)
Women	1,277 (49.7)	313 (46.8)	964 (50.7)
Race and ethnicity, n (%)			
American Indian or Alaska Native	33 (1.3)	8 (1.2)	25 (1.3)
Asian	325 (12.6)	150 (22.4)	175 (9.2)
Black	83 (3.2)	20 (3.0)	63 (3.3)
Hispanic or Latino(a)	99 (3.9)	28 (4.2)	71 (3.7)
Native Hawaiian or Pacific Islander	18 (0.7)	5 (0.7)	13 (0.7)
White	1,669 (64.9)	359 (53.7)	1,310 (68.9)
Other[a] or missing	344 (13.3)	99 (14.8)	245 (12.9)
Age, (y), mean ± SD	58 ± 18	57 ± 17	58 ± 18
Age category, y			
18-39	461 (17.9)	131 (19.6)	330 (17.4)
40-59	870 (33.8)	229 (34.2)	641 (33.7)
60-79	978 (38.0)	257 (38.4)	721 (37.9)
≥80	262 (10.2)	52 (7.8)	210 (11.0)
Medications			
Medication use, n (%)			
ACE inhibitor/ARB	1,250 (48.6)	451 (67.4)	799 (42.0)
Corticosteroids	1,006 (39.1)	267 (39.9)	739 (38.9)
Other immunomodulators[b]	77 (3.0)	35 (5.2)	42 (2.2)
SGLT2 inhibitor	42 (1.6)	31 (4.6)	11 (0.6)
Clinical characteristics			
Hypertension, n (%)	1,919 (74.6)	557 (83.3)	1,362 (71.6)
Diabetes, n (%)	708 (27.5)	285 (42.6)	423 (22.2)
eGFR, mL/min/1.73m^2, n (%)	2,571 (100.0)	669 (100.0)	1,902 (100.0)
Mean ± SD	78 ± 27	71 ± 29	80 ± 26
UACR, mg/g, n (%)	531 (20.7)	531 (79.4)	0 (0.0)
Median (IQR)	166 (25-795)	166 (25-795)	-
UPCR, g/g, n (%)	218 (8.5)	218 (32.6)	0 (0.0)
Median (IQR)	0.7 (0.2-1.8)	0.7 (0.2-1.8)	-
Systolic blood pressure, mmHg, n (%)	2,435 (94.7)	637 (95.2)	1,798 (94.5)
Mean ± SD	128 ± 16	130 ± 15	128 ± 16

Abbreviations: IgAN, IgA nephropathy; UACR, urine albumin/creatinine ratio; UPCR, urine protein/creatinine ratio; UCLA, University of California, Los Angeles; IQR, interquartile range; SD, standard deviation; ACE, angiotensin-converting enzyme; ARB, angiotensin II receptor blocker; SGLT, sodium-glucose cotransporter; eGFR, estimated glomerular filtration rate; BMI, body mass index; Hb, hemoglobin.
[a]Includes patients that did not identify with main census categories.
[b]Includes biologics, calcineurin inhibitors, cytotoxic agents, mammalian target of rapamycin inhibitors, corticotropin agents, and pyrimidine synthesis inhibitors.

UACR or UPCR measurements, levels above versus below the medians predicted greater MAKE hazard (HR = 1.46; 95% CI, 1.02-2.08; Fig 3B; Table S2). Hospitalization, lower eGFR, and outpatient visits had similar directions and sizes of effect as the main model. Unadjusted and adjusted estimates for the main and secondary models are shown in Table S2.

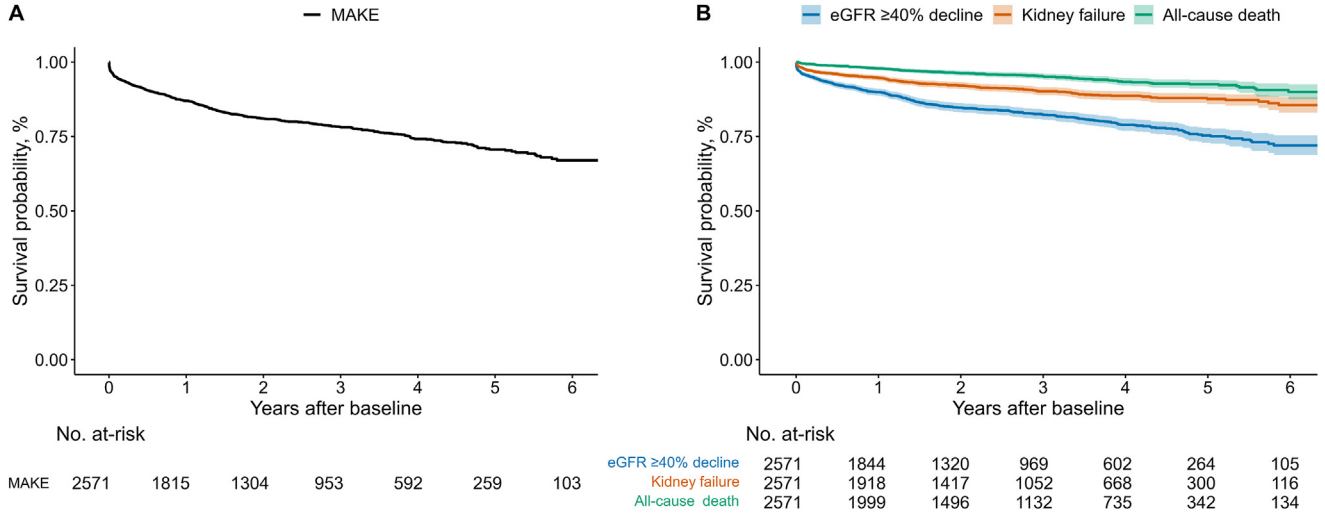


Figure 2. Survival probability of MAKE in patients with IgAN. Kaplan-Meier survival analyses. (A) Primary composite MAKE outcome ≥40% eGFR decline, eGFR < 15 mL/min/1.73 m^2, dialysis or transplant, and death in patients with IgAN (n = 2,571). (B) Individual components for the primary composite MAKE outcome. MAKE, major adverse kidney events; eGFR, estimated glomerular filtration rate.

DISCUSSION

More than one in 5 patients with IgAN from 2 United States health systems experienced MAKE defined as eGFR decline ≥40%, kidney failure (eGFR <15 mL/min/1.73m^2, dialysis, or transplant), or death after 3 years of follow-up. Notably, kidney failure and death comprised 27% and 11% of MAKE, respectively. In addition to established clinical predictors, MAKE was predicted by having noncommercial health insurance, hospitalization, and more frequent outpatient encounters.

A recent study from the United Kingdom Registry of Rare Diseases projected that most patients with IgAN will progress to kidney failure during their lifespan, irrespective of baseline kidney function or proteinuria.[6] Risk prediction is a central tenet of detecting and stratifying patients for clinical decision-making. In addition to identifying patients at higher risk, lower risk patients may be spared unnecessary treatments, side effects, and costs. Our study provides new knowledge about types of health insurance and care utilization that have not been part of previous predictive models for IgAN.[9,10] Insurance status is one measure of social determinants of health. The noncommercial health insurance group was predominantly comprised of patients with Medicare and a smaller proportion with Medicaid. As such, this group may have had higher risks related to unmeasured variables such as disabilities and low income. An important issue for further investigation is whether their insurance coverage was sufficient to support optimal care for IgAN, inclusive of clinical assessments and recommended therapies. Hospitalization, an indicator for severity of illness by acuity, was the strongest predictor of MAKE with risk increased by more than 2-fold in either the main or secondary models. More frequent outpatient encounters also point to illness severity and health seeking behaviors. Health insurance and care utilization patterns may help health systems to take a population-level approach to identify patients with IgAN who may benefit from close surveillance and nephrology specialty care to optimize management.

Diabetes and use of ACE inhibitors, ARBs, or other immunomodulators were significant clinical predictors in the main model, but did not predict MAKE in the

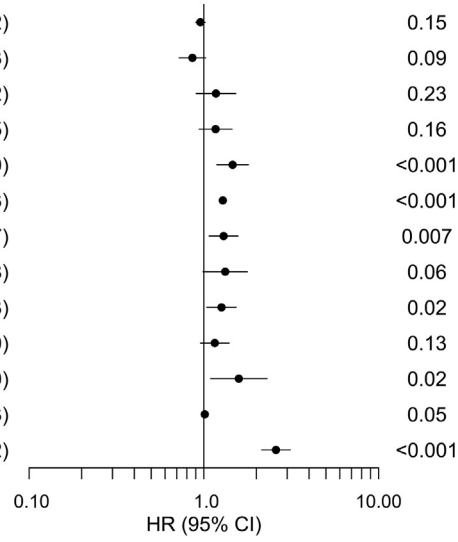
Table 2. Description of Events Among Study Participants With IgAN and MAKE During 2016-2022

	Total	Subset With UACR or UPCR	Subset Without UACR or UPCR
	N = 536	n = 178	n = 358
Event type, n (% of MAKE)			
eGFR ≥40% decline	327 (61.0)	105 (59.0)	222 (62.0)
Kidney failure	108 (20.1)	43 (24.2)	65 (18.2)
Dialysis	23 (4.2)	7 (3.9)	16 (4.5)
Transplant	17 (3.2)	11 (6.2)	6 (1.7)
All-cause death	61 (11.4)	12 (6.7)	49 (13.7)

Abbreviations: IgAN, IgA nephropathy; MAKE, major adverse kidney events; UACR, urine albumin/creatinine ratio; UPCR, urine protein/creatinine ratio; eGFR, estimated glomerular filtration rate.

A

Predictors	Comparison	Reference	HR (95% CI)	P
	No. outcome/No. in group		*Lower risk of MAKE — Higher risk of MAKE*	
Age (per 10 years)	-	-	0.96 (0.90-1.02)	0.15
Women (reference: men)	221/1277	315/1294	0.86 (0.72-1.03)	0.09
Asian (reference: White)	73/325	342/1669	1.17 (0.90-1.52)	0.23
Non-White (reference: White)	121/577	342/1669	1.17 (0.94-1.45)	0.16
Noncommercial vs commercial insurance	335/1217	201/1354	1.46 (1.19-1.80)	<0.001
eGFR (per − 10 mL/min/1.73 m²)	-	-	1.28 (1.24-1.33)	<0.001
Diabetes (yes/no)	230/708	306/1863	1.30 (1.07-1.57)	0.007
Hypertension (yes/no)	474/1919	62/652	1.33 (0.99-1.78)	0.06
ACE inhibitor/ARB (yes/no)	343/1250	193/1321	1.26 (1.04-1.53)	0.02
Corticosteroid (yes/no)	272/1006	264/1565	1.16 (0.96-1.40)	0.13
Other immunomodulator (yes/no)	35/77	501/2494	1.59 (1.09-2.30)	0.02
Outpatient Visits (average per quarter)	-	-	1.01 (1.00-1.03)	0.05
Hospitalization (yes/no)	253/646	283/1925	2.59 (2.14-3.12)	<0.001



B

Predictors	Comparison	Reference	HR (95% CI)	P
	No. outcome/No. in group		*Lower risk of MAKE — Higher risk of MAKE*	
Age (per 10 years)	-	-	1.02 (0.90-1.15)	0.79
Women (reference: men)	80/313	98/356	0.88 (0.64-1.20)	0.43
Asian (reference: White)	42/150	100/359	1.27 (0.86-1.87)	0.22
Non-White (reference: White)	36/160	100/359	0.98 (0.66-1.46)	0.92
Noncommercial vs commercial insurance	99/277	79/392	1.22 (0.85-1.77)	0.28
eGFR (per − 10 mL/min/1.73 m²)	-	-	1.25 (1.17-1.34)	<0.001
UACR ≥166 mg/g or UPCR ≥0.69 g/g (yes/no)	113/341	65/328	1.46 (1.02-2.08)	0.04
Diabetes (yes/no)	96/285	82/384	1.25 (0.88-1.77)	0.21
Hypertension (yes/no)	159/557	19/112	0.85 (0.49-1.46)	0.55
ACE inhibitor/ARB (yes/no)	119/451	59/218	0.82 (0.59-1.15)	0.25
Corticosteroid (yes/no)	89/267	89/402	1.10 (0.78-1.55)	0.57
Other immunomodulator (yes/no)	14/35	164/634	1.25 (0.68-2.31)	0.47
Outpatient Visits (average per quarter)	-	-	1.02 (1.00-1.05)	0.04
Hospitalization (yes/no)	63/142	115/527	1.84 (1.29-2.63)	<0.001

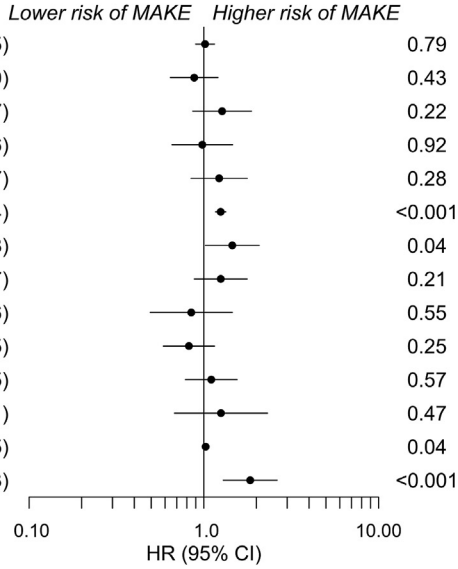


Figure 3. Predictors of MAKE in patients with IgAN. Adjusted Cox proportional hazards models. (A) Main model with full cohort. (B) Secondary model including subset with baseline UACR or UPCR measurements. HR, hazard ratio; CI, confidence interval; UCLA, University of California Los Angeles; eGFR, estimated glomerular filtration rate; UACR, urine albumin/creatinine ratio; UPCR, urine protein creatinine ratio; ACE, angiotensin-converting enzyme; ARB, angiotensin II receptor blocker.

secondary model, suggesting that the differences may be explained by albuminuria or proteinuria. Asian race was not a significant predictor of MAKE, which may reflect low statistical power considering the limited sample size, especially in the secondary model. Alternatively, Asian race could be a risk factor for developing IgAN, but not necessarily worse prognosis than the reference group when taking into account other demographic, clinical, and health care variables. Previous predictive models of IgAN prognosis have included proteinuria and histologic features according to the Oxford classification.[7-13] However, baseline proteinuria measures and prognostic features of kidney biopsies were not routinely available in the EHR of patients that comprised the CURE-CKD Registry cohort. The lack of proteinuria data is well-recognized in health system datasets for chronic kidney disease in general.[21] Although proteinuria measures may exist in clinical records outside of health systems, their lack of availability

limits the usefulness of risk prediction by these measures at a large population level. The CURE-CKD model enables a population health approach to risk stratification for IgAN based on routine information within health systems, eg health insurance, care utilization, medications, and available laboratory reports.

Risk prediction anchored in clinical practice is timely and important because IgAN therapies are rapidly expanding. A genome-wide association study of >10,000 patients and nearly 29,000 controls from 17 different international cohorts found that polygenic risk scores predicted high-risk of kidney failure associated with abnormal IgA production and pathogenic inflammatory pathways.[27] In response to immunologic injury, endothelin and angiotensin II are downstream proinflammatory mediators that damage intrinsic glomerular cells and the filtration barrier.[28,29] Based on the underlying biology, immunomodulating and anti-inflammatory agents have become key therapeutic targets.[30] Systemic and gut-specific corticosteroid therapies and a dual endothelin-angiotensin receptor antagonist have received regulatory approval for high-risk patients with IgAN.[14-17] Sodium-glucose cotransporter 2 inhibitors also reduce risk of kidney failure in IgAN.[31-33] Several other agents are currently in development for IgAN by systemic or kidney tissue immunomodulation.[34-37] CURE-CKD and other real-world cohort studies will be central to understanding uptake of new therapies and identification of patients who may benefit and assure access.

Our study has many strengths, such as curated patient data and clinical event outcomes. The CURE-CKD cohort of nearly 2,600 patients with IgAN is one of the biggest to-date and a first from clinical practice at 2 different health systems.[4,6,9,10] Nevertheless, EHRs are limited by missingness, miscoding, and retrospective data.[38] To address these limitations, we defined IgAN and disease status by available laboratory tests and by administrative codes.[20,21] However, the baseline UACR or UPCR measurements were present in the EHRs for only 26% of patients. Thus, the secondary analysis must be interpreted cautiously because of susceptibility to reduced power and unmeasured biases. IgAN classification by kidney biopsy features was not available. However, IgAN is a diagnosis made by biopsy, so the likelihood of over-diagnosis is probably small. The median duration of follow-up was nearly 3 years, but more observation time is needed to assess longer-term risks. Although CURE-CKD represents 2 different health systems in the United States, our data may not be generalizable in other settings or geographic regions.

In conclusion, substantial loss of kidney function, kidney failure, and death occurred commonly over a short period of time in patients with IgAN at 2 United States health systems. In addition to clinical predictors, MAKE was predicted by noncommercial health insurance, hospitalization, and more frequent outpatient encounters. Type of health insurance and care utilization may help to risk stratify patients with IgAN and enable a population health approach within health systems for monitoring and therapeutic interventions to reduce risks.

SUPPLEMENTARY MATERIALS

Supplementary File (PDF)

Table S1: ICD 9 and 10 Codes for IgA Nephropathy and Kidney Failure With or Without Kidney Replacement Therapy.

Table S2: Unadjusted and Adjusted Estimates for Variables Predicting MAKE in Patients With IgA Nephropathy.

ARTICLE INFORMATION

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