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Research article

A nomogram to predict cognitive function impairment in patients with chronic kidney disease: A national cross-sectional survey

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ABSTRACT

Background: Cognitive function impairment (CFI) is common in patients with chronic kidney disease (CKD) and significantly impacts treatment adherence and quality of life. This study aims to create a simplified nomogram for early CFI risk detection.

Methods: Data were obtained from the National Health and Nutrition Examination Survey cycles spanning from 1999 to 2002 and again from 2011 to 2014. Stepwise logistic regression was used to select variables and construct a CFI risk prediction model. Furthermore, C-statistic and Brier Score (BS) assessed model performance. Additionally, Kaplan–Meier survival curves were utilised to assess risk group-death prognosis relationships.

Results: Of the 545 participants in the CKD model development cohort, a total of 146 (26.8 %) had CFI. The final model included the variables of age, race, education, annual family income, body mass index, estimated glomerular filtration rate, serum albumin and uric acid. The model had a C-statistic of 0.808 (95 % confidence interval (CI): 0.769–0.847) and a BS of 0.149. Furthermore, the 5-fold cross-validation internal C-statistic was 0.764 (interquartile range: 0.763–0.807) and BS was 0.154. Upon external validation, the model's C-statistic decreased to 0.752 (95 % CI: 0.654–0.850) and its BS increased to 0.182. The Kaplan–Meier survival curves demonstrated that intermediate-to-high-risk participants had shorter overall survival time than low-risk participants (log-rank test: $p = 0.00042$).

Conclusions: This study established an effective nomogram for predicting CFI in patients with CKD, which can be used for the early detection of CFI and guide the treatment of patients with CKD.

1. Introduction

Chronic kidney disease (CKD) is a global health problem with high prevalence and mortality rates and is associated with several

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cardiovascular and cerebrovascular diseases [1–6]. While medical advancements have potential to prolong the lifespan of patients with CKD, the extended lifespan could result in an increased risk of cognitive function impairment (CFI), which can affect memory, language, executive function, and attention [7]. Furthermore, studies report that patients with CKD are more likely to have CFI [8–10] and that the combination of CKD and CFI reduced physical function, lowered the quality of life and introduced a higher risk of death [11].

Previous studies have found numerous factors to be associated with cognitive functional impairment (CFI). Mild CFI, for instance, has been correlated with decreased haemoglobin and higher serum creatine [12]. In contrast, severe CFI has shown associations with stroke, extended periods of education, and an equilibrated Kt/V greater than 1.2 [10]. Many variables have been linked to CFI as well, including low glomerular filtration rate, low serum albumin, proteinuria, high uric acid, elevated C-reactive protein, and higher levels of homocysteine [9,13–15]. Due to structural and functional abnormalities in the brain, patients with CKD are prone to CFI [16–18]. Meanwhile, CKD is an independent risk factor for cognitive impairment, with cognitive impairment symptoms manifesting in the early stages of the disease in some CKD patients, especially in elderly patients, but these symptoms usually appear atypical and are easily disregarded [14,19–22].

Although the pathogenesis of CFI in patients with CKD also may involve a variety of factors as mentioned above, currently no simple and effective risk prediction tool exists to aid clinicians in identifying the onset of CFI early, and thus implement preventive or mitigating strategies. Thus, we analysed data from the National Health and Nutrition Examination Survey (NHANES) to establish a reliable model for the detection and evaluation of CFI in non-hospitalized individuals and construct a nomogram for use in clinical settings. Notably, CFI may also affect adherence and decision-making, which could affect longevity and prognosis. Therefore, in this study, the constructed nomogram was used to stratify the predicted probability of developing CFI for each individual and derive survival curves from the follow-up data to determine whether this risk stratification was associated with the actual outcome.

2. Methods

2.1. Study population and design

The NHANES is a nationally representative, cross-sectional survey conducted by the Centres for Disease Control and Prevention (CDC) that uses a complex, multi-stage sampling design, wherein when the sample weights are considered, the results are representative of the non-institutionalised resident population of the United States [23]. NHANES data are released in 2-year cycles, and data from two cycles between 2011 and 2014 were used for model development and internal validation, whereas data from 1999 to 2002 were used for external validation. The screening of 3,632 participants provided the cohort data for model development. Finally, a total of 545 study participants were included after excluding data with an estimated glomerular filtration rate (eGFR) of 60 mL/min/1.73 m^2 or higher (n = 2,855), no cognitive function tests (n = 143) and missing variables (n = 89). Furthermore, 131 participants were included in the external validation cohort after excluding data with eGFR ≥60 ml/min/1.73 m^2 (n = 3,519), no cognitive function tests (n = 48) and other missing variables (n = 8). All NHANES procedures were approved by the Ethics Committee of the National Centre for Health Statistics, and all survey participants provided written informed consent. Fig. 1 presents the flow of participant screening.

2.2. Candidate predictors

General demographic data (age, sex, race, education, annual family income and body mass index (BMI)), previous history (smoking history, drinking status, hypertension, diabetes, congestive heart failure (CHF), congenital heart disease (CHD), stroke, sleep disorders and dialysis) and laboratory data (albuminuria, eGFR, serum albumin, blood urea, blood calcium/phosphorus, blood cholesterol, blood bicarbonate, blood uric acid (UA), serum iron and haemoglobin) were collated as potential predictors.

The eGFR in our study was calculated using the CKD-EPI algorithm [24]. Age was divided into three categories: 60–69 years, 70–79

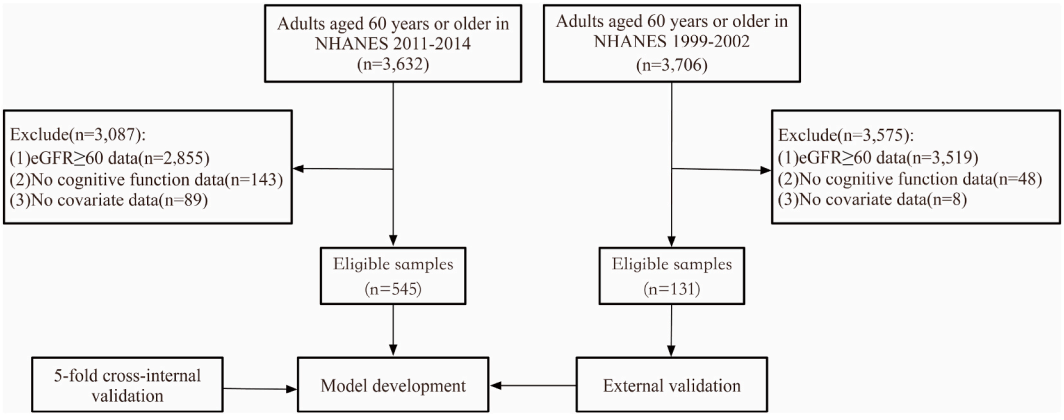

Fig. 1. Participants flow diagram.

Table 1
Characteristic of participants in the model development cohort.

Characteristic	Overall, n = 545	No CFI, n = 399	CFI, n = 146	*P*-value
Weighted, n	10,166,832	8,269,303	1,897,529	
Demographics				
Age[a]	74.0 (68.0, 80.0)	73.0 (67.0, 80.0)	80.0 (71.0, 80.0)	**<0.001**
Age group, n (%) [b]				**<0.001**
60–69 years	137 (28)	117 (32)	20 (12)	
70–79 years	181 (34)	133 (35)	48 (28)	
80+ years	227 (38)	149 (33)	78 (60)	
Sex, n (%) [b]				0.6
Female	285 (57)	215 (56)	70 (59)	
Male	260 (43)	184 (44)	76 (41)	
Race, n (%) [b]				**<0.001**
Mexican American	26 (2.1)	11 (1.1)	15 (6.5)	
Non-Hispanic White	347 (85)	280 (88)	67 (69)	
Non-Hispanic Black	101 (7.0)	60 (5.3)	41 (15)	
Other Hispanic	37 (2.4)	21 (1.5)	16 (6.3)	
Asian	24 (1.7)	18 (1.6)	6 (2.6)	
Other	10 (1.8)	9 (2.1)	1 (0.6)	
Education, n (%) [b]				**<0.001**
Less than high school	146 (20)	73 (15)	73 (42)	
High school graduate or GED	123 (21)	87 (19)	36 (30)	
Some college or above	276 (59)	239 (66)	37 (29)	
Annual family income, n (%) [b]				**<0.001**
<$ 20,000	152 (20)	90 (17)	62 (37)	
≥$ 20,000	393 (80)	309 (83)	84 (63)	
BMI[a], kg/m^2	28 (25,33)	29 (26,33)	26 (24,32)	0.076
BMI, kg/m^2, n (%) [b]				**0.013**
<18.5	6 (0.9)	3 (0.5)	3 (2.9)	
18.5–25	122 (22)	83 (19)	39 (34)	
25-30	195 (36)	150 (37)	45 (32)	
≥30	222 (41)	163 (43)	59 (32)	
Previous History, n (%) [b]				
Smoking history	351 (67)	264 (70)	87 (54)	**0.007**
Drinking status	286 (51)	216 (52)	70 (49)	0.6
Hypertension	417 (76)	298 (74)	119 (82)	0.13
Diabetes	177 (28)	121 (27)	56 (33)	0.3
CHF	87 (15)	54 (12)	33 (25)	**<0.001**
CHD	87 (16)	61 (15)	26 (17)	0.7
Stroke	52 (9.4)	35 (9.0)	19 (16)	0.6
Sleep disorder	70 (13)	55 (14)	15 (9.8)	0.2
Dialysis	7 (1.0)	5 (0.5)	2 (3.6)	**0.026**
Laboratory tests[b]				
eGFR, ml/min/1.73m^2	50 (42, 55)	51 (44, 56)	47 (38, 54)	**<0.001**
Albuminuria, mg/L	13 (6, 34)	12 (5, 31)	18 (10, 84)	**<0.001**
Albumin, g/L	42 (40, 43)	42 (40, 44)	41 (39, 43)	**0.004**
BUN, mmol/L	7.5 (6.1, 8.9)	7.5 (6.1, 8.9)	7.1 (5.9, 8.9)	0.8
Calcium, mmol/L	2.35 (2.30, 2.42)	2.35 (2.30, 2.42)	2.35 (2.28, 2.42)	0.093
Phosphorus, mmol/L	1.23 (1.13, 1.36)	1.23 (1.13, 1.36)	1.23 (1.10, 1.36)	>0.9
Cholesterol, mmol/L	42 (40, 43)	42.00 (40, 44)	41 (39, 43)	**0.004**
Bicarbonate, mmol/L	25 (23, 27)	25 (23, 27)	25 (24, 27)	0.7
UA, umol/L	381 (321, 440)	381 (327, 440)	376 (309, 429)	**0.041**
Iron, umol/L	13.6 (10.6, 17.6)	14.0 (10.7, 17.7)	12.4 (10.0, 15.9)	**0.035**
Haemoglobin, g/dL	14 (13, 15)	14 (13, 15)	12.60 (12, 14)	**0.001**
Cognitive function tests[b]				
CERAD Immediate	19 (16, 22)	20 (17, 23)	14 (10, 16)	**<0.001**
CERAD Delayed	6 (4, 7)	7 (5, 8)	3 (2, 5)	**<0.001**
Animal Fluency Test	16 (13, 20)	17 (15, 21)	11 (9, 13)	**<0.001**
DSST	48 (35, 59)	51 (42, 61)	23 (20, 30)	**<0.001**
Total scores	88 (71, 104)	94 (82, 107)	56 (45, 61)	**<0.001**
Follow up, months				
Follow-up time[a]	76 (63,92)	77 (65,92)	71 (37,83)	**0.001**
Death[b]	188 (32)	120 (27)	68 (53)	**<0.001**

[a] Median (IQR).
[b] Unweighted frequency counts and weighted percentages, General educational development; BMI, Body mass index; CHF, Congestive heart failure; CHD, Congenital heart disease; eGFR, Estimated glomerular filtration rate; BUN, Blood urea nitrogen; UA, Uric acid; CERAD, The consortium to establish a registry for Alzheimer's Disease; DSST, The digit symbol substitution test. CFI, Cognitive function impairment.

years and 80 years and older. Education was categorised as less than high school, high school graduate or GED and some college or higher education. According to the WHO International Classification, BMI was divided into four categories: 18.5, 18.5–25, 25–30 and 30. Annual family income was categorised as below $20,000 and above $20,000.

The following questions were used to collect information on smoking history and drinking status: 'Have you smoked at least 100 cigarettes in your lifetime?' and 'Have you had at least one drink in a year?' (yes/no). Participants were asked, 'Have you ever been told by a doctor or health professional that you have _?' to determine diagnoses of hypertension, diabetes, CHF, CHD, stroke and sleep disorders. Dialysis receipt was determined by the question 'Have you received dialysis in the past 12 months?' All blood and urine samples were collected using a standardised procedure. The detailed methodology can be obtained from the NHANES website (https://www.cdc.gov/nchs/nhanes).

2.3. Outcome variable

Participants in this study who were 60 years of age or older received two cycles of cognitive testing from 2011 to 2014 were administered. Memory function was assessed using the Consortium to Establish a Registry for Alzheimer's Disease Vocabulary Learning subtest, while the executive function was assessed using the Animal Fluency Test. Processing speed, sustained attention and working memory were measured using the Digit Symbol Substitution Test. The results of these tests were scored by trained interviewers during the interview. Based on previous studies [25,26], CFI was defined as at or below the lower quartile range of the total score of these three tests in the 2011 to 2014 cycles. Additionally, CFI was also defined as a score below the lower quartile range of the DSST in the two cycles from 1999 to 2002 [25].

2.4. Statistical analysis

R software version 4.2.2 was used for all statistical analyses in this study. The Wilcoxon rank-sum test for complex survey samples was used for continuous variables and the chi-squared test was used for categorical variables. The log-rank test was used for survival curves in this study. All P values < 0.05 were considered statistically significant.

The mobile examination center exam weight was the weight variable used for analysis herein. The 'svydesign' function of the survey package of the R software processed all weight variables before they were extracted and used for data analysis, including group comparisons, model building and survival curve plotting.

2.5. Model development and validation

In the model development phase, a stepwise regression model was initially used to screen for statistically significant variables. The predictor variables were then included in a logistic regression model. In the model validation phase, we performed internal validation using a 5-fold cross-validation approach and external validation of the model in the model external validation set. Furthermore, we developed a simple nomogram to visualise the model results, ensuring easy applicability in clinical practice.

2.6. Evaluation of model performance

The C statistic and the Brier Score (BS), which range from 0 to 1, were used to evaluate the performance of the model. The C statistic ranged from 0.9 to 1, indicating excellent model discrimination performance, 0.8 to 0.9, good performance, 0.7 to 0.8, moderate performance and less than 0.7, poor or worthless performance. The BS is a comprehensive criterion for model discrimination and calibration, with values ranging from 0 to 1. The closer the value is to 0, the better the overall performance of the model and the closer the model's prediction probability is to the actual value; however, if it exceeds 0.25, the model's performance is considered to be worse than the actual value.

The estimated predicted probabilities (EPP) of the model were used to stratify the participants into low-risk (EPP < 0.1), intermediate-risk ($0.1 \leq$ EPP < 0.3), and high-risk ($0.3 \leq$ EPP) groups. Risk stratification and participant death prognosis were examined by plotting Kaplan–Meier survival curves on the model development cohort death follow-up data. Participant death follow-up data are available at https://ftp.cdc.gov/pub/Health_Statistics/NCHS/datalinkage/linked_mortality/.

3. Results

A total of 545 (weighted = 10,166,832) participants were included in the model development cohort. Of these, 57 % were female and the median age was 74 years. The median glomerular filtration rate was 50 ml/min/1.73 m^2 and the median cognitive test score was 88. CFI was diagnosed in 146 (19 %) participants. Moreover, 188 participants died during a median follow-up of 76 months (Table 1). CFI was associated significantly with age, race, education, annual family income, BMI, drinking status, CHF, dialysis, eGFR, albuminuria, serum albumin, blood cholesterol, blood uric acid, serum iron and haemoglobin ($P < 0.05$). Other variable characteristics are listed in Table 1.

3.1. Model development and nomogram construction

Predictors of the final model included age, race, education, annual family income, BMI, CHF, eGFR, serum albumin and UA. In the

final logistic regression model, age (80+ years, odds ratio (OR): 5.44, 95 % confidence interval (CI): 2.50–12.90, P < 0.001), education (less than high school, OR: 4.28, 95 % CI: 2.23–8.29, P < 0.001; high school graduate or GED, OR: 2.98, 95 % CI: 1.57–5.71, P < 0.001), annual family income (<$20,000, OR: 2.12, 95%CI: 1.17–3.85, P = 0.013) and CHF (OR:2.10, 95 % CI: 1.07–4.13, P = 0.031) were risk factors for CFI. Race (non-Hispanic white, OR: 0.12, 95 % CI: 0.02–0.53, P = 0.007; other, OR: 0.05, 95 % CI: 0.00–0.62, P = 0.047), high eGFR (OR: 0.97, 95 % CI: 0.95–1.00, P = 0.017) and high UA (OR: 0.99, 95 % CI: 0.99–1.00, P < 0.001) were protective factors for CFI (Table 2). Finally, a simplified nomogram (Fig. 2) was established to guide clinical practice.

3.2. Model performance and validation

The C-statistic of the final model was 0.808 (95 % CI: 0.769–0.847) and the BS was 0.149. In the 5-fold cross-internal validation, the mean C-statistic of the model was 0.764 (IQR: 0.763–0.807) and BS was 0.154 (Table 3).

Compared with the model development cohort, the external validation cohort had a higher proportion of participants aged 80+ years (52 % vs 42 %; P = 0.03), a lower education level (less than high school: 43 % vs 27 %; P < 0.001), lower annual family income (<$20,000: 56 % vs 28 %; P < 0.001), and significant differences in race, BMI and serum albumin (Supplementary Table 1). After external validation, the C-statistic of the model was 0.752 (95 % CI: 0.654–0.850) and BS was 0.182 (Table 3).

3.3. Kaplan–Meier survival estimates for risk groups

The Kaplan–Meier survival curve plot in the model development cohort is shown in Fig. 3. Compared to the low-risk group, those in the intermediate to the high-risk group had shorter survival times (log-rank test, P = 0.00042).

4. Discussion

This study provides the first diagnostic prediction model for CFI in non-institutionalised patients with CKD. To represent a nationally sampled population, we weighted a complex NHANES data sample for modelling. Primary care physicians can use the established simplified nomogram, which includes nine clinically accessible variables, that demonstrated robust model discrimination and calibration using internal and external validation. Additionally, we risk-stratified the nomogram-calculated participant CFI risk. After calculating survival curves with the mortality follow-up data, we found that patients with CKD in the intermediate and high-risk groups for CFI had shorter survival times and higher mortality rates than those in the low-risk group.

Table 2
Multivariate logistic regression model.

Characteristic	Beta	OR[a]	95 % CI[a]	*P*-value
Intercept	4.26			
Age				
60–69 years	ref			
70–79 years	0.842	2.32	1.02, 5.63	0.052
80+ years	1.69	5.44		**<0.001**
Race				
Mexican American	ref		2.50, 12.9	
Non-Hispanic White	−2.14	0.12	0.02, 0.53	**0.007**
Non-Hispanic Black	−0.57	0.57	0.10, 2.96	0.5
Other Hispanic	−0.01	0.99	0.13, 7.29	>0.9
Asian	−1.04	0.35	0.03, 2.98	0.4
Other	−3.03	0.05	0.00, 0.62	**0.047**
Education				
Less than high school	1.45	4.28	2.23, 8.29	**<0.001**
High school graduate or GED	1.09	2.98	1.57,5.71	**<0.001**
Some college or above	ref			
Annual family income				
<$ 20,000	0.75	2.12	1.17, 3.85	**0.013**
≥$ 20,000	ref			
BMI				
<18.5	0.74	2.11	0.28, 18.9	0.5
18.5–25	ref			
25–30	−0.47	0.62	0.32, 1.20	0.2
>30	−0.84	0.43	0.21,0.86	**0.018**
CHF				
No	ref			
Yes	0.74	2.10	1.07, 4.13	**0.031**
eGFR	−0.03	0.97	0.95, 1.00	**0.017**
Albumin	−0.07	0.93	0.86, 1.01	0.10
UA	−0.01	0.99	0.99, 1.00	**<0.001**

[a] OR = Odds Ratio, CI = Confidence Interval; GED, General educational development; BMI, Body mass index; CHF, Congestive heart failure; eGFR, Estimate glomerular filtration rate; UA, Uric acid.

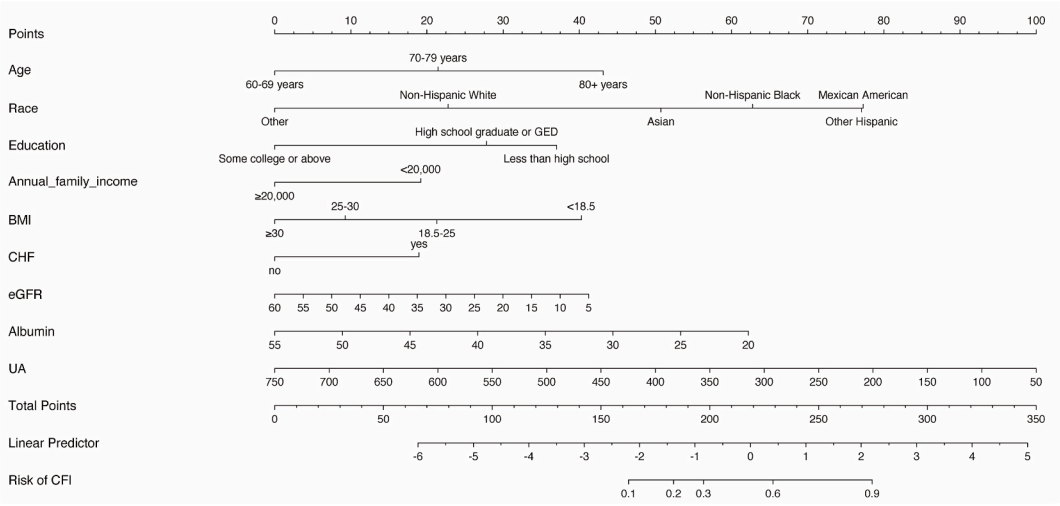

Fig. 2. The nomogram is a tool for estimating CFI in CKD patients, utilizing a multivariate logistic regression model. It works by assigning points to each independent variable (for instance, an eGFR of 5 ml/min/1.73 m^2 receives 40 points). These points are then added together to form a total score. The CFI score is determined by correlating this total with the risk of bias scale.

Table 3
Model performance.

	Development cohort	Internal validation cohort	External validation cohort
C-statistic	0.808 (0.769, 0.847)	0.764 (0.763, 0.807)	0.752 (0.654, 0.850)
Brier score	0.149	0.154	0.182

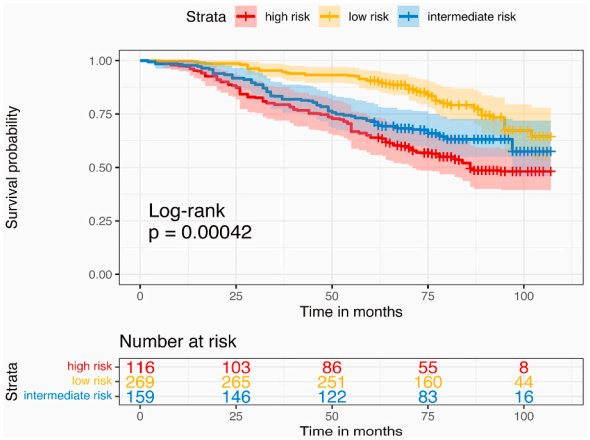

Fig. 3. Kaplan–Meier survival curves were created in NHANES 2011–2014 dataset. The participants were stratified into low-risk (EPP $<$ 0.1), intermediate-risk (0.1 $\leq$ EPP $<$ 0.3), and high-risk (0.3 $\leq$ EPP) groups based on the score of the nomogram. The Kaplan–Meier survival curves demonstrated that intermediate-to-high-risk participants had shorter overall survival time than low-risk participants (log-rank test: p = 0.00042). EPP, estimated predicted probabilities.

In the current study, the rate of CFI in patients with CKD of the two cycles of NHANES (2011–2014) was 19 %, which is higher than the rate of CFI in American patients with CKD, as reported by Tamura et al. [6,9], but lower than the rate of CFI with CKD patients reported by Puy et al. [13], which could be related to the study population, cognitive function assessment techniques and the stage of CKD of the patients at the time of evaluation.

Studies have found that the proportion of dialysis in CKD patients with cognitive impairment is higher, compared with CKD patients without cognitive impairment, suggesting that dialysis could be a risk factor for CFI in CKD patients. The dialysis process has been previously implicated in the development of brain lesions, such as cerebral ischemia and cerebral edema, which may potentially elevate the susceptibility to cognitive impairment [10,11,27–30]. However, dialysis was not included as a predictor in our final model.

The reason could be attributed to the fact that NHANES primarily focuses on surveying non-hospitalized residents in the United States, resulting in a relatively low representation of dialysis patients within the included sample, accounting for only 1 % of individuals with CKD (after appropriate weighting is applied). Furthermore, our results support the findings of most other studies [9,14,31] indicating that eGFR is a risk factor for CFI in patients with CKD. Additionally, the incidence of CFI increased with decreasing eGFR, which is consistent with previous findings [32].

Our research indicate that, older age (≥80 years), low annual family income (<$20,000) and low educational level (less than high school, high school graduate or GED) are the risk factors for CFI. Generally, the risk of cognitive deterioration and dementia increases with age [33], and this may be particularly evident in patients with CKD. Older age and cognitive decline are significantly correlated in patients with CKD [34]. The prevalence of CKD has been found to vary among different ethnic groups in previous studies [35–37]. However, racial differences in CFI risk in patients with CKD have rarely been studied. In this study, Mexican Americans are at a higher risk for CFI, while non-Hispanic whites and other races are protective factors of CFI among patients with CKD. Future studies should investigate the risk of CFI in patients with CKD of different races and other risk factors.

CHF is considered a risk predictor of CFI in patients with CKD. Research has shown that, CHF and CKD are bidirectionally associated. Patients with CHF have a significantly increased risk of rapid CKD progression, and the prevalence of CHF increases with CKD progression [38–40]. Furthermore, many patients with CHF have cognitive deficits. Declining cardiac function can result in reduced cerebral perfusion, oxidative stress, and inflammation, these factors may contribute to cognitive decline in patients with CHF [41]. Meanwhile, cognitive decline also can be promoted by these factors in CKD. Furthermore, serum albumin levels of older individuals were observed to be independently associated with a higher risk of CFI [42,43], dialysis patients showed similar results [44]. Our findings also imply that as albumin levels decrease in patients with CKD, the risk of CFI gradually rises. We also observed that patients with CKD with lower UA levels have a higher risk of CFI. However, contradictory evidence has been reported regarding the relationship between UA and CFI. Studies have shown that higher UA levels are associated with decreased working memory, processing speed, verbal fluency and verbal memory [45]. However, another study found significantly lower UA levels in patients with cognitive impairment and dementia [46]. Low UA levels are a risk factor for mild cognitive impairment (MCI), and appropriate increases in UA may slow the onset and progression of MCI [47]. A prospective study showed that higher baseline UA levels were associated with better cognitive function (overall cognitive function, executive function and memory function) later in life and a lower risk of dementia [48], which could be attributed to the anti-inflammatory effects of UA.

Moreover, consistent with the findings of earlier research, a BMI of 30 or above was considered a protective factor in preventing the occurrence of CFI in patients with CKD. As the studies shown that, BMI was positively associated with cognitive function in patients with CKD [32], and the risk of dementia decreased with BMI increased, whereas the risk increased with underweight [49]. These previous studies, despite some conflicting results, indicate that CFI in patients with CKD is caused by multiple influencing factors that interact and may enhance or weaken certain mechanisms of cognitive impairment. Thus, a combined approach is required to improve cognitive performance in patients with CKD to prevent further decline.

According to the follow-up data we incorporated into the queue, approximately 53 % of CKD patients with CFI experienced a fatal outcome within a median follow-up period of 71 months. Our constructed predictive model enables stratification of CKD patients based on the risk of developing CFI. We observed that compared to CKD patients with a low risk of CFI, those with moderate to high risk had shorter survival times and earlier fatal outcomes, indicating a significant impact of CFI on the survival of CKD patients. Therefore, we recommend early diagnosis and intervention of CFI in CKD patients, as it may help extend their survival and improve the quality of life.

The main shortcomings of the study are as follows: Firstly, while the study is cross-sectional, it is less significant than longitudinal cohort studies and does not reveal the causal relationships between the variables. Secondly, the external validation cohort had a small sample size. Moreover, the results require further validation as the R software has no procedure for external validation weighting, so the NHANES survey weights were not used in the external validation process. Thirdly, our study was conducted in a non-clinical population, thus model performance must be validated in a clinical population and with a larger external study cohort.

5. Conclusions

This study developed a diagnostic prediction model for CFI in patients with CKD and constructed nomogram plots, offering practical application tools for clinicians. The quality of life and prognosis of patients with CKD have the potential to improve with the acceptance of this prediction model as a screening and risk assessment tool for CFI in patients with CKD. Therefore, the use of this model in clinical practice could contribute to the management and treatment of patients with CKD.

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Availability of data

You can get the data used for this study at https://www.cdc.gov/nchs/nhanes.

Ethical approval and consent to participate

All NHANES procedures were approved by the Ethics Committee of the National Centre for Health Statistics, and all survey participants provided written informed consent.

CRediT authorship contribution statement

Tong Zhou: Writing – review & editing, Writing – original draft, Validation, Project administration, Methodology, Investigation, Formal analysis, Data curation, Conceptualization. **Heping Zhang:** Writing – review & editing, Visualization, Validation, Supervision, Software, Resources, Project administration, Funding acquisition, Conceptualization. **Jiayu Zhao:** Writing – review & editing, Writing – original draft, Methodology, Investigation. **Zhouting Ren:** Writing – original draft, Investigation, Data curation. **Yimei Ma:** Writing – original draft, Investigation, Data curation. **Linqian He:** Writing – original draft, Investigation, Data curation. **Jiali Liu:** Writing – original draft, Methodology, Investigation, Data curation. **Jincheng Tang:** Writing – original draft, Investigation, Formal analysis, Data curation. **Jiaming Luo:** Writing – review & editing, Validation, Supervision, Software, Resources, Project administration, Funding acquisition, Conceptualization.

Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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Appendix A. Supplementary data

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