Lipids in Health and Disease

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Identification and optimization of relevant factors for chronic kidney disease in abdominal obesity patients by machine learning methods: insights from NHANES 2005–2018

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Abstract

Background The intake of dietary antioxidants and glycolipid metabolism are closely related to chronic kidney disease (CKD), particularly among individuals with abdominal obesity. Nevertheless, the cumulative effect of multiple comorbid risk factors on the progression and complications of CKD remains inadequately characterized.

Methods This study analyzed data from the National Health and Nutrition Examination Survey (NHANES) dat abase (2005–2018), to examine potential factors related to CKD, including glycolipid metabolism, dietary antioxidant intake, and pertinent medical history. To explore the associations between these variables and CKD, the present study used a multivariable-adjusted least absolute shrinkage and selection operator (LASSO) regression model, along with a restricted cubic spline (RCS) model. Furthermore, an optimal predictive model was developed for CKD using ten machine learning algorithms and enhanced model interpretability with the Shapley Additive Explanations (SHAP) method.

Results A cohort comprising 8,764 eligible individuals (52% male, including 1,839 CKD patients) with abdominal obesity aged 20–85 years were included. The findings revealed significant positive correlations in patients with abdominal obesity between the presence of CKD and age, a history of heart failure, hypertension, diabetes, elevated lipid accumulation product (LAP) and triglyceride glucose-waist circumference (TyG-WC) levels. Conversely, negative correlations were identified between CKD and variables such as sex, high-density lipoprotein cholesterol (HDL-C) levels, and the composite dietary antioxidant index (CDAI). In parallel, RCS regression analysis revealed significant nonlinear associations between the CDAI, HDL-C, TyG-WC, and CKD among patients with abdominal obesity aged 60–80 years. The development of predictive models demonstrated that the CatBoost model surpassed other models, achieving an accuracy of 86.74% on the validation set. The model's area under the receiver operator curve (AUC) and F1 score were 0.938 and 0.889, respectively. The SHAP values revealed that age was the most significant predictor, followed by diabetes history, hypertension, HDL-C levels, CDAI index, TyG-WC, and LAP.

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Conclusion CatBoost models, along with glycolipid metabolism indexes and dietary antioxidant intake, are effective for early CKD detection in patients with abdominal obesity.

Keywords Chronic kidney disease, Machine learning, Triglyceride glucose-waist circumference, Composite dietary antioxidant index, Abdominal obesity

Introduction

Chronic kidney disease (CKD) has garnered significant attention as the sole non-communicable disease with a consistently rising age-adjusted mortality rate, increasing from 813,000 in 2000 to 1.3 million in 2019 [1]. The consequences of CKD include premature mortality, disability, diminished quality of life, and various psychosocial impacts, resulting in substantial financial burdens on governments, healthcare systems, and families [2]. As a result, efforts to identify and mitigate avoidable risk factors for the onset and progression of CKD have been ongoing.

A high body mass index (BMI) is a well-established and significant risk factor for kidney impairment in the general population [3, 4]. However, individuals with CKD constitute a unique group for studying the interplay between obesity and kidney function. Merely defining obesity based on BMI alone no longer accurately captures the connection between obesity and CKD [5]. Recent studies have highlighted that abdominal obesity in middle-aged and young adults is recognized as one of the most influential risk factors for CKD, with evidence suggesting that it may also lead to higher mortality rates among CKD patients [6, 7]. Additionally, anthropometric indices that assess obesity and metabolism have been developed to help predict CKD, such as the visceral adiposity index (VAI), lipid accumulation product (LAP), and triglyceride glucose (TyG) [8–10]. These indices provide a more precise assessment of an unhealthy metabolic phenotype. Whereas, existing research on the TyG index in relation to CKD has primarily focused on the general population rather than specifically on individuals with abdominal obesity.

However, obesity can directly predispose individuals to CKD through its association with obesity-related glomerulopathy, as well as indirectly through its well-established complications including hypertension, and type 2 diabetes. Additionally, measures of abdominal fat, like waist circumference, are often associated with higher morbidity and mortality in people on maintenance hemodialysis [3]. Thus, addressing obesity is vital for preventing and slowing the progression of CKD.

Studies have demonstrated that modifications in lifestyle habits, such as poor dietary choices and sedentary behaviors, have varying impacts on improvements in obesity, blood pressure, and renal function in individuals with CKD [11–14]. Previous studies have shown that an antioxidant-rich diet is beneficial for various health outcomes, including lung function, cardiovascular function, and mental health [15–17], and can offer protection against both CKD and obesity, with the composite dietary antioxidant index (CDAI) demonstrating a positive correlation with a reduced prevalence of CKD in the adult population in the United States [18]. In other words, reducing lifestyle-related risk factors can assist in the prevention of CKD and its complications.

Multiple contributory factors require the consideration of comprehensive models, which is why machine learning (ML) for predicting chronic diseases has received widespread attention in recent years [19, 20]. Hence, this study was to find the factors linked to CKD in individuals with abdominal obesity through the application of ML techniques, considering obesity indices, overall pro- and antioxidant exposure status, and other relevant factors. Such a comprehensive model is relatively uncommon in existing literature, highlighting the distinctiveness of this study. This study aimed to improve understanding of CKD risk factors in individuals with abdominal obesity by developing predictive ML models using cross-sectional data from the National Health and Nutrition Examination Survey (NHANES) database.

Methods

Study design and subjects

The study analyzed data from the NHANES database spanning 2005–2018 (https://www.cdc.gov/nchs/nhanes/). All methods adhered to relevant guidelines and regulations, ensuring no use of personally identifiable information. Publicly accessible interview questionnaires and test response rates are documented in sources [21, 22]. The exclusion criteria include: (1) subjects < 18 years of age, with missing waist circumference and BMI data; (2) incomplete renal function indexes, triglyceride-glucose index data, CDAI information, and medical history details (such as asthma, hypertension, diabetes, cancer, stroke, heart failure, etc.); and (3) incomplete information on covariates (race, marital status, education, etc.), and body weight data. A total of 8,764 patients with abdominal obesity were included. The study procedure is illustrated in Fig. 1. All participants provided written informed consent in accordance with the NHANES study protocol, which received approval from the Research

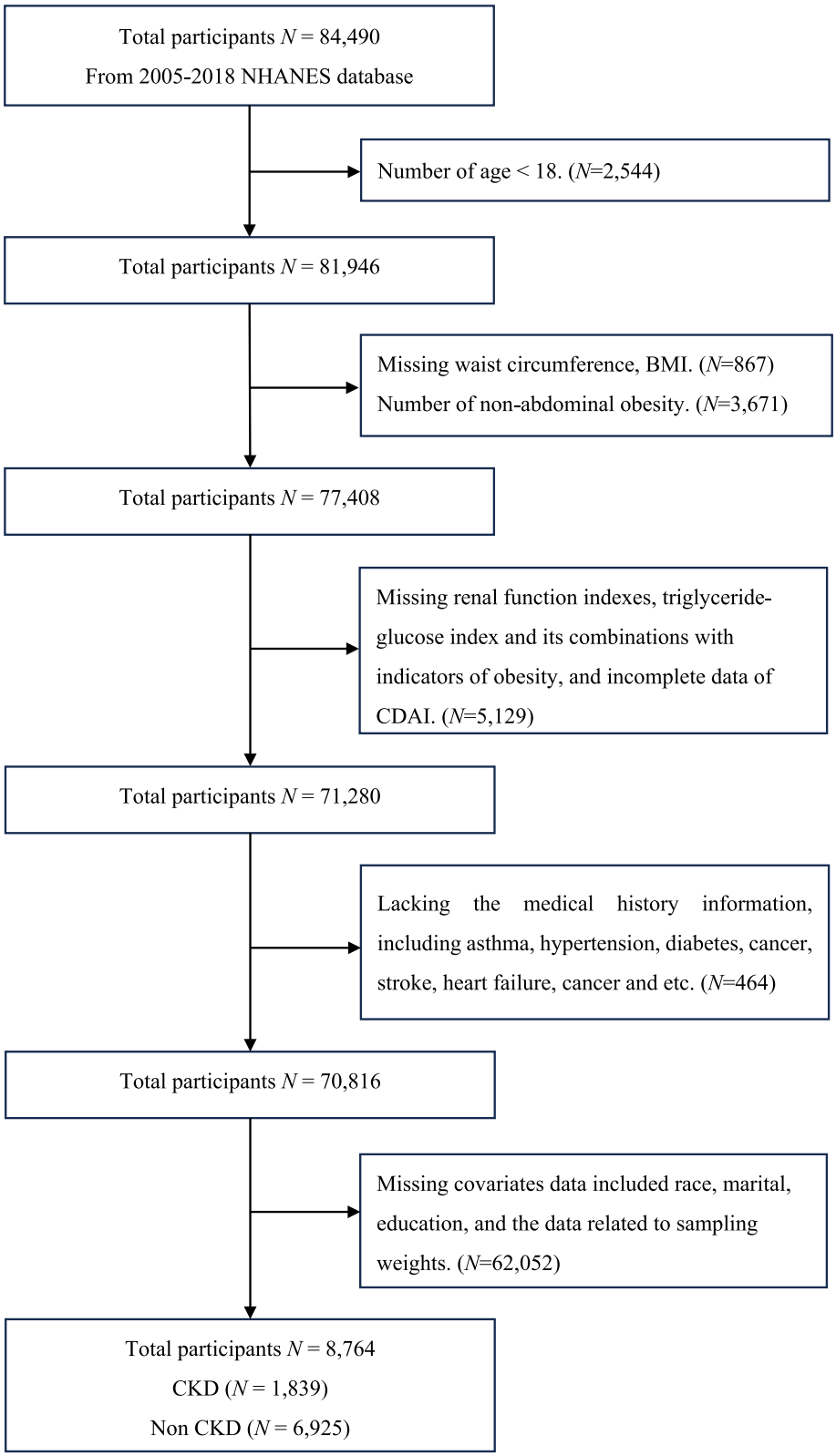


Fig. 1 Eelection flowchart of study participants in NHANES, 2005–2018

Ethics Review Board of the National Center for Health Statistics (NCHS).

Definitions of CKD, Abdominal Obesity

The maximum urinary albumin-to-creatinine ratio (ACR) of 30 mg/g was classified as albuminuria. The estimated glomerular filtration rate (eGFR) was determined using the 2021 Chronic Kidney Disease Epidemiology Collaboration equation based on standardized serum creatinine levels across survey cycles [23]. A decreased eGFR was considered to be $\leq$60 mL/min/1.73 m^2 [24]. CKD was defined as the presence of albuminuria or decreased eGFR. Abdominal obesity was defined by an absolute waist circumference (WC) of more than 102 cm in males and 88 cm in females, respectively [25].

Measurement of TyG, TyG-WC, VAI, LAP, CDAI

The TyG index is a measure of insulin resistance. Serum fasting plasma glucose (FPG), high-density lipoprotein cholesterol (HDL-C) levels, and triglyceride (TG) were assessed at baseline. TyG = ln [TG(mg/dl) × FPG(mg/dl)/2], TyG-WC = TyG × WC [26], VAI (male) = WC/(39.68 + 1.88 × BMI) × TG/1.03 × 1.31/HDL-C, VAI (female) = WC/ (36.58 + 1.89 × BMI) × TG/0.81 × 1.52/HDL-C [27], LAP (male) = (WC-65) × TG, LAP (female) = (WC-58) × TG [28].

To measure dietary antioxidant intake, the present study utilized a modified CDAI based on the method of Wright et al. [29]. In this approach, each individual's antioxidant intake was normalized by determining how far it differed from the average intake of different antioxidants, including β-carotene, vitamin A, selenium, vitamin C, vitamin E, zinc, copper, and iron. These standardized values were then combined to create the CDAI score [30]. Importantly, antioxidants from supplements, medications, or water alone were not considered in this calculation. The formula is presented below:

$$CDAI = \sum_{i=1}^{n=8} \frac{(IndividualIntake - Mean)}{SD}$$

Covariates

At the commencement of the study, standardized questionnaires were employed to gather demographic data (including age, sex, race/ethnicity, educational attainment, and marital status), lifestyle factors, and self-reported medical history (encompassing conditions such as stroke, heart failure, hypertension, diabetes, insulin usage, liver disorders, and cancer). The baseline laboratory test results included glycosylated hemoglobin (HbAlc), total cholesterol (TC), TG, HDL-C, serum creatinine (Scr), and urine creatinine levels. Further details on the variable assessment and categories can be found in Supplementary Table 1.

Statistical analysis

Continuous variables are presented as medians (Q1–Q3) and were compared using either the independent samples t-test or the Kruskal–Wallis rank sum test. Categorical variables are represented as percentages, and intergroup differences were evaluated using the chi-square test or Fisher's exact test. All values were adjusted using the sampling weights provided by the NHANES to ensure representativeness for the general U.S. population. A two-sided *P*-value of less than 0.05 was deemed statistically significant. To address potential issues related to high dimensionality impacting ML algorithms, a multivariable-adjusted least absolute shrinkage and selection operator (LASSO) regression model [31] was utilized to identify potential factors that were most strongly associated with CKD risk. Moreover, a subgroup analysis based on multiple logistic regression was used to explore the impact of other variables on different stages of CKD. GFR categories (G1–G5) and albuminuria categories (A1–A3) are used to classify CKD [32], as shown in Supplementary Table 2. A restricted cubic spline (RCS) model was conducted to find the dose–response relationship between CKD risk and other variables. The synthetic minority over-sampling technique (SMOTE) is an oversampling method that is widely used in medicine to handle class imbalances in datasets [33, 34]. In the present study, the SMOTE combined with the edited nearest neighbors (SMOTE-ENN) technique was utilized to increase the sensitivity of classifiers to minority groups.

Construction of the ML model

Ten ML algorithms were then applied to predict CKD in patients with abdominal obesity using the training set: AdaBoost, CatBoost, decision tree (DT), gradient boosting decision tree (GBDT), light gradient boosting machine (LightGBM), logistic regression (LR), naive bayes (NB), random forest (RF), support vector machine (SVM), and eXtreme gradient boosting (XG). These models were implemented using the DecisionLinnc1.0 software [35]. Furthermore, Supplementary Table 3 provides details on hyperparameter configurations.

Evaluation of ML model

Different evaluation metrics were used to evaluate the model, including accuracy, receiver operating characteristic (ROC) curves, AUC, sensitivity/recall, specificity, the Matthews correlation coefficient (MCC) and the F1 score. To evaluate the robustness of the predictive model's performance, a sensitivity analysis utilizing k-fold cross-validation was performed.

SHAP method

To enhance the comprehensibility of our models, the SHAP method was used to assess the relative significance of predictor variables [36]. In the model, a predicted value is produced for each anticipated sample. The SHAP Summary plot graphically displays the SHAP value associated with each feature across all samples, with purple and yellow dots indicating higher or lower feature values separately.

Results

Baseline characteristics

In this study, 8,764 individuals with abdominal obesity aged 20–85 years were included, comprising 4,532 (52%) males and 4,232 (48%) females. The patients were divided into CKD patients ($n=1839$) and non-CKD patients ($n=6925$) as shown in Table 1. Table 1 shows a significant association between abdominal obesity in patients with CKD and older age in females compared with those of the non-CKD group, as evidenced by higher values of various anthropometric and metabolic markers such as WC, HbAlc, FPG, TG, Scr, TyG, TyG-WC, VAI, and LAP ($P<0.05$). Additionally, a greater proportion of participants in the CKD group reported a history of smoking, stroke, heart failure, hypertension, liver conditions, diabetes, and insulin use ($P<0.05$).

Association of candidate predictive variables with CKD in patients with abdominal obesity.

Supplementary Fig. 1A illustrates the link between cross-validation error variability and the log-transformed penalty parameter (λ) in LASSO regression for identifying key variables related to CKD in patients with abdominal obesity. Within the optimal LASSO regression model, a total of nine variables (age, sex, TyG-WC, HDL-C, LAP, CDAI, and a history of heart failure, hypertension, and diabetes) with non-zero coefficients were determined to be more significant in relation to CKD, as illustrated in Supplementary Fig. 1B. The study revealed significant positive correlations between age (Coeff se = 0.0435), history of heart failure (Coeff se = 0.8327), hypertension (Coeff se = 0.3326), diabetes (Coeff se = 0.2440), LAP (Coeff se = 0.0001), and TyG-WC (Coeff se = 0.0008) with CKD in patients with abdominal obesity. Conversely, a negative correlation was observed between sex (Coeff se = −0.3341), HDL-C level (Coeff se = −0.0157), and CDAI score (Coeff se = −0.0012) and CKD in the study participants.

Subgroup analysis of the influence of various variables on different stages of CKD

The subgroup analysis revealed that age significantly influences CKD risk levels. Specifically, individuals aged > 60 years are markedly more likely to develop CKD than those aged 40–60. For individuals over 60 years, the odds ratio (OR) for a moderately increased risk of CKD was 3.83 (95% CI: 3.16–4.64, $P<0.001$), whereas in the 40–60 years age group, the OR was 1.55 (95% CI: 1.28–1.87, $P<0.001$). Furthermore, the OR for a high risk of CKD was 5.59 (95% CI: 3.59–8.70, $P<0.001$) versus 1.49 (95% CI: 0.93–2.36, $P=0.094$), and for a very high risk of CKD, the OR was 13.30 (95% CI: 5.66–31.29, $P<0.001$) compared with 1.57 (95% CI: 0.62–3.97, $P=0.341$) in the younger cohort. The severity of abdominal obesity is closely associated with the risk level of CKD. Similarly, a history of diabetes, hypertension, and heart failure significantly elevates the risk of CKD. For comprehensive results, please consult Supplementary Table 2.

Association of the CDAI, LAP, TyG-WC and HDL-C with CKD in patients with abdominal obesity

RCS regression analysis was employed to investigate the relationship between CDAI, LAP, TyG-WC, and HDL-C and CKD in patients with abdominal obesity. A significant nonlinear association was observed between the CDAI score, HDL-C level, and CKD in obese abdominal patients of both genders (Fig. 2 A-D). However, sex differences had been found in the nonlinear relationships between LAP, TyG-WC and CKD, with females exhibiting a significant association with LAP and males with TyG-WC (Fig. 2 E–H). The present study investigated potential age-related variations in the impact of various factors on CKD in individuals with abdominal obesity. These findings indicate a nonlinear correlation between LAP and CKD in the 18–40 years age group (Fig. 2 I-L). Interestingly, no correlations were detected between each of the four factors and CKD in obese abdominal patients aged 40–60 years (Fig. 2 M-P). However, significant nonlinear correlations between the CDAI, HDL-C, and TyG-WC and CKD in patients with abdominal obesity were maintained in the 60–80 years age group (Fig. 2 Q-T).

Evaluation of the model

Figure 3 illustrates the ROC curves for the ten models analyzed in this study. Specifically, AdaBoost (Fig. 3 A), CatBoost (Fig. 3 B), DT (Fig. 3 C), GBDT (Fig. 3 D), LightGBM (Fig. 3 E), LR (Fig. 3 F), NB (Fig. 3 G), RF (Fig. 3 H), SVM (Fig. 3 I) and XG (Fig. 3 J) models achieved AUC values of 0.838, 0.938, 0.863, 0.869, 0.783, 0.886, 0.823, 0.891, 0.882, and 0.923, respectively. The SMOTE-ENN technique balanced the classes in the training dataset to avoid model bias, as shown in Table 2 with a detailed summary of the performance metrics for each model. Notably, the CatBoost algorithm achieved superior prediction performance, with an accuracy of 86.74% and an F1 score of 0.889. Therefore, the CatBoost-based

Table 1 Baseline characteristics of the abdominal obesity participants grouped by CKD status

Characteristic	Overall *N* = (8,764)	Non-CKD (*N* = 6,925)	CKD (*N* = 1,839)	*P*
Age (years)	51.0 (37.0—64.0)	47.0 (35.0—60.0)	65.0 (51.5—75.0)	< 0.001
Gender				
Female	4,232 (48.0%)	3,212 (46.0%)	1,020 (55.0%)	< 0.001
Male	4,532 (52.0%)	3,713 (54.0%)	819 (45.0%)	
Race				
Mexican American	1,559 (17.8%)	1,304 (18.8%)	255 (13.9%)	< 0.001
Other Hispanic	928 (10.6%)	780 (11.3%)	148 (8.1%)	
Non-Hispanic White	3,735 (42.6%)	2,918 (42.1%)	817 (44.4%)	
Non-Hispanic Black	1,848 (21.1%)	1,343 (19.4%)	505 (27.5%)	
Other Race—Including Multi-Racial	694 (7.9%)	580 (8.4%)	114 (6.2%)	
Education				
Less Than 9th Grade	959 (10.9%)	712 (10.3%)	247 (13.4%)	< 0.001
9-11th Grade	1,252 (14.3%)	966 (14.0%)	286 (15.6%)	
High School Grad/GED or Equivalent	2,026 (23.1%)	1,548 (22.3%)	478 (26.0%)	
Some College or AA degree	2,606 (29.7%)	2,107 (30.4%)	499 (27.1%)	
College Graduate or above	1,921 (21.9%)	1,592 (23.0%)	329 (17.9%)	
Marital				
Married	4,793 (54.7%)	3,820 (55.2%)	973 (53.0%)	< 0.001
Widowed	665 (7.6%)	368 (5.3%)	297 (16.2%)	
Divorced	989 (11.3%)	774 (11.2%)	215 (11.7%)	
Separated	302 (3.5%)	238 (3.4%)	64 (3.5%)	
Never married	1,289 (14.7%)	1,108 (16.0%)	181 (9.8%)	
Living with partner	726 (8.3%)	617 (8.9%)	109 (5.9%)	
Smoke history				
No	4,764 (54.0%)	3,860 (56.0%)	904 (49.0%)	0.011
Yes	4,000 (46.0%)	3,065 (44.0%)	935 (51.0%)	
Alcohol				
No	7,806 (89.1%)	6,147 (88.8%)	1,659 (90.2%)	0.14
Yes	958 (10.9%)	778 (11.2%)	180 (9.8%)	
BMI	29.8 (26.9—33.6)	29.8 (26.9—33.6)	29.9 (26.8—34.0)	0.265
WC	102.3 (95.2—111.2)	101.7 (94.8—110.4)	104.9 (96.7—114.1)	< 0.001
HbAlc (%)	5.6 (5.3—6.0)	5.5 (5.2—5.8)	5.8 (5.4—6.5)	< 0.001
Fasting Glucose (mmol/L)	5.66 (5.3—6.3)	5.6 (5.2—6.1)	6.0 (5.5—7.2)	< 0.001
TC (mmol/L)	5.0 (4.3—5.7)	5.0 (4.3—5.7)	4.8 (4.1—5.6)	< 0.001
TG (mmol/L)	1.3 (0.9—1.9)	1.3 (0.9—1.9)	1.4 (1.0—2.0)	< 0.001
HDL-C (mmol/L)	1.3 (1.1—1.6)	1.3 (1.1—1.6)	1.2 (1.06—1.5)	0.0024
Scr (umol/L)	0.9 (0.7—1.0)	0.8 (0.7—1.0)	1.1 (0.8—1.3)	< 0.001
Urine creatinine (umol/L)	7.0 (4.5—14.0)	6.2 (4.3—9.9)	38.6 (9.1—88.6)	< 0.001
Serum creatinine (umol/L)	75.1 (62.8—88.4)	72.5 (61.0—84.0)	93.7 (71.6—113.2)	< 0.001
eGFR	91.3 (73.4—107.9)	95.1 (80.6—109.9)	59.1 (50.1—89.3)	< 0.001
TyG	8.7 (8.3—9.1)	8.7 (8.3—9.1)	8.9 (8.4—9.3)	< 0.001
TyG_WC	894.7 (812.8—995.5)	887.8 (806.7—984.4)	927.4 (840.8—1,039.5)	< 0.001
VAI	1.6 (1.0—2.7)	1.6 (1.0—2.6)	1.9 (1.2—3.1)	< 0.001
LAP	53.2 (34.6—83.1)	51.2 (33.2—80.4)	59.9 (39.4—95.2)	< 0.001
CDAI	−0.9 (−4.0—2.9)	−0.7 (−3.9—3.0)	−1.7 (−4.8—2.0)	< 0.001
Vitamin A (mg/d)	495.5 (271.0—708.5)	461.0 (273.5—709.5)	453.5 (260.3—701.8)	0.547
Vitamin E (mg/d)	6.7 (4.6—9.4)	6.8 (4.8—9.5)	6.1 (4.1—8.8)	< 0.001
Beta-carotene (mcg)	1,050.0 (439.0—2,620.6)	1,052.5 (443.0—2,642.5)	1,030.0 (424.3—2,595.0)	0.84
Vitamin C (mg/d)	62.3 (29.8—112.4)	63.2 (30.4—113.6)	58.3 (27.6—106.8)	0.004

Table 1 (continued)

Characteristic	Overall *N* = (8,764)	Non-CKD (*N* = 6,925)	CKD (*N* = 1,839)	*P*
Zinc (mg/d)	10.0 (7.3—13.4)	10.2 (7.5—13.6)	9.2 (6.6—12.7)	< 0.001
Selenium (mcg/d)	103.1 (75.5—134.8)	105.0 (77.8—136.6)	96.5 (69.1—126.3)	< 0.001
Copper	1.1 (0.8—1.4)	1.1 (0.9—1.5)	1.0 (0.8—1.4)	< 0.001
iron	13.2 (9.7—17.7)	13.4 (9.9—17.9)	12.5 (9.0—16.9)	< 0.001
Stroke				
No	8,448 (96.4%)	6,765 (97.7%)	1,683 (91.5%)	< 0.001
Yes	316 (3.6%)	160 (2.3%)	156 (8.5%)	
Heart failure				
No	8,487 (96.8%)	6,821 (98.5%)	1,666 (90.6%)	< 0.001
Yes	277 (3.2%)	104 (1.5%)	173 (9.4%)	
Hypertension				
No	5,312 (60.6%)	4,607 (66.5%)	705 (38.3%)	< 0.001
Yes	3,452 (39.4%)	2,318 (33.5%)	1,134 (61.7%)	
Insulin				
No	8,420 (96.1%)	6,767 (97.7%)	1,653 (89.9%)	< 0.001
Yes	344 (3.9%)	158 (2.3%)	186 (10.1%)	
Liver condition				
No	8,390 (95.7%)	6,651 (96.0%)	1,739 (94.6%)	0.002
Yes	374 (4.3%)	274 (4.0%)	100 (5.4%)	
Diabetes				
No	7,306 (83.4%)	6,057 (87.5%)	1,249 (67.9%)	< 0.001
Borderline	212 (2.4%)	160 (2.3%)	52 (2.8%)	
Yes	1,246 (14.2%)	708 (10.2%)	538 (29.3%)	

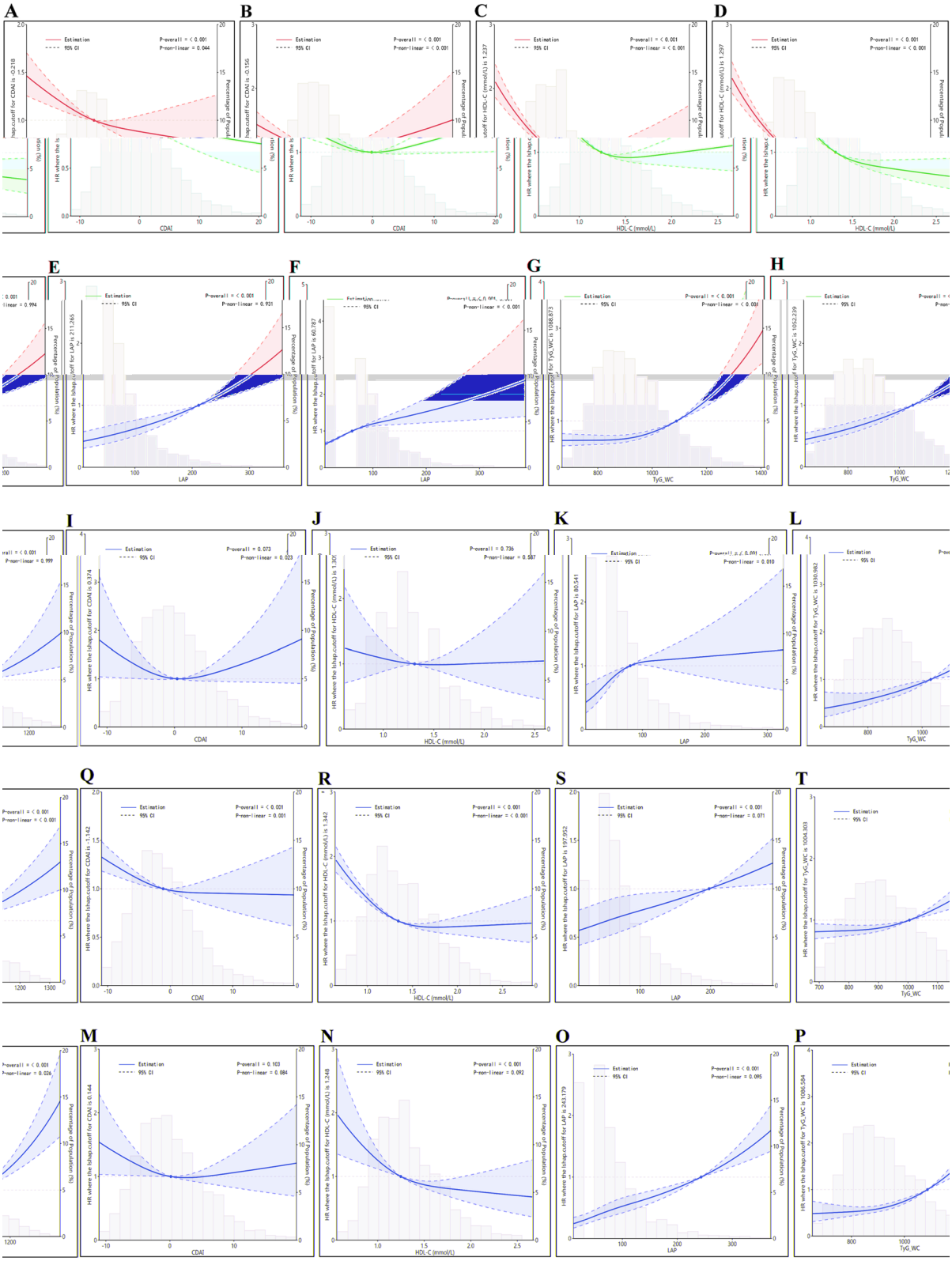
Abbreviations: Data are presented as frequencies (percentages) or median (Q1-Q3); *GED* General Educational Development, *AA degree* Associate of Arts Degree, *BMI* body-mass index, *WC* circumference waist, *HbAlc* glycosylated hemoglobin, *TG* triglycerides, *TC* total cholesterol, *HDL-C* high density lipoproteincholesterol, *Scr* serum creatinine, *eGFR* estimated glomerular filtration, *VAI* visceral adiposity index, *LAP* lipid accumulation product, *TyG* triglyceride-glucose index, *TyG-WC*Triglyceride Glucose-Waist Circumference, *CDAI* composite dietary antioxidant index, *CKD* chronic kidney disease

prediction model was ultimately selected for the subsequent analysis.

The outcomes of the tenfold cross-validation indicated that the CatBoost-based prediction model exhibited robust performance, achieving an average AUC of 0.938, a mean accuracy of 86.41%, and an F1 score of 0.885. The ROC curve corresponding to the cross-validation is presented in Supplementary Fig. 2.

(See figure on next page.)

Fig. 2 The study examines the Restricted Cubic Spline (RCS) modeling of the relationship between composite dietary antioxidant index (CDAI), HDL-C (mmol/L), Lipid accumulation product (LAP), triglyceride glucose-waist circumference (TyG-WC) and the prevalence of CKD in abdominal obesity patients. **A** The RCS curve of the association between CDAI (male) and CKD with abdominal obesity. **B** The RCS curve of the association between CDAI (female) and CKD with abdominal obesity. **C** The RCS curve of the association between HDL-C (male) and CKD with abdominal obesity. **D** The RCS curve of the association between HDL-C (female) and CKD with abdominal obesity. **E** The RCS curve of the association between LAP (male) and CKD with abdominal obesity. **F** The RCS curve of the association between LAP (female) and CKD with abdominal obesity. **G** The RCS curve of the association between TyG-WC (male) and CKD with abdominal obesity. **H** The RCS curve of the association between TyG-WC (female) and CKD with abdominal obesity. **I** The RCS curve of the association between CDAI and CKD with abdominal obesity at age 18–40. **J** The RCS curve of the association between HDL-C and CKD with abdominal obesity at age 18–40. **K** The RCS curve of the association between LAP and CKD with abdominal obesity at age 18–40. **L** The RCS curve of the association between TyG-WC and CKD with abdominal obesity at age 18–40. **M** The RCS curve of the association between CDAI and CKD with abdominal obesity at age 40–60. **N** The RCS curve of the association between HDL-C and CKD with abdominal obesity at age 40–60. **O** The RCS curve of the association between LAP and CKD with abdominal obesity at age 40–60. **P** The RCS curve of the association between TyG-WC and CKD with abdominal obesity at age 40–60. **Q** The RCS curve of the association between CDAI and CKD with abdominal obesity at age 60–80. **R** The RCS curve of the association between HDL-C and CKD with abdominal obesity at age 60–80. **S** The RCS curve of the association between LAP and CKD with abdominal obesity at age 60–80. **T** The RCS curve of the association between TyG-WC and CKD with abdominal obesity at age 60–80



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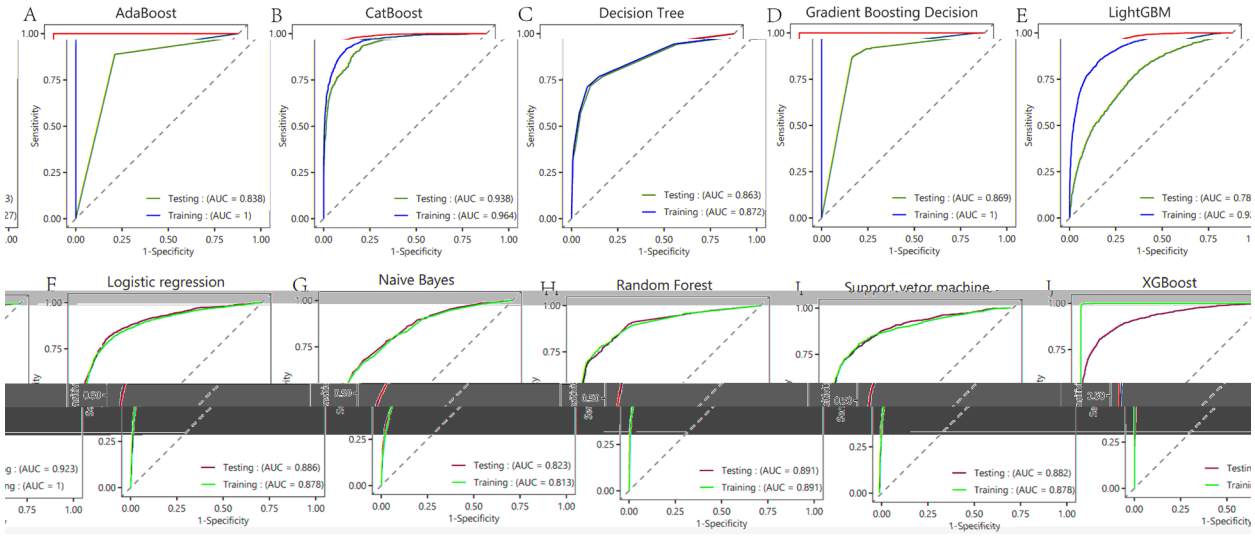

Fig. 3 The ROC of the ten machine learning models. Machine learning model was utilized to fit the prediction between composite dietary antioxidant index (CDAI), HDL-C (mmol/L), Lipid accumulation product (LAP), triglyceride glucose-waist circumference (TyG-WC) and the prevalence of CKD in abdominal obesity patients. The plots show the ROC of (**A**) AdaBoost; (**B**) CatBoost; (**C**) Decision Tree; (**D**) Gradient Boosting Decision Tree; (**E**) LightGBM; (**F**) Logistic regression; (**G**) Naive Bayes; (**H**) Random Forest; (**I**) Support Vector machine; (**J**) XGBoost Machine. These curves depict the performance of the models on both the training set and the testing set

Table 2 Comparison of discrimination characteristics among ten machine learning models

Model name	Training	Recall	Accuracy	F1-Score	MCC	AUC
AdaBoost	Imbalance	0.376325	0.729278	0.374341	0.201620	1.000
CatBoost	Imbalance	0.298587	0.806844	0.399527	0.326193	0.875
Decision Tree	Imbalance	0.287986	0.806844	0.390887	0.322116	0.754
Gradient Boosting Decision Tree	Imbalance	0.362191	0.730798	0.366726	0.195853	1.000
LightGBM	Imbalance	0.280919	0.800000	0.376777	0.298428	0.944
Logistic regression	Imbalance	0.266784	0.806844	0.37284	0.314087	0.768
Naive Bayes	Imbalance	0.613074	0.660456	0.437303	0.241620	0.714
Random forest	Imbalance	0.286219	0.805703	0.388024	0.317875	0.780
Support vetor machine	Imbalance	0.000000	0.784791	NA	NA	0.646
XGBoost	Imbalance	0.323322	0.801521	0.412162	0.320935	0.990
AdaBoost	Balance	0.887827	0.848218	0.875550	0.681574	0.838
CatBoost	Balance	0.886552	0.867382	0.889386	0.723863	0.938
Decision Tree	Balance	0.768005	0.793024	0.816949	0.587348	0.863
Gradient Boosting Decision Tree	Balance	0.891013	0.853200	0.879522	0.692142	0.869
LightGBM	Balance	0.852135	0.728248	0.790423	0.418553	0.783
Logistic regression	Balance	0.873168	0.811422	0.847772	0.602344	0.886
Naive Bayes	Balance	0.698534	0.666922	0.716106	0.314429	0.823
Random Forest	Balance	0.787126	0.803756	0.828303	0.605274	0.891
Support vetor machine	Balance	0.876992	0.804140	0.843396	0.586093	0.882
XGBoost	Balance	0.825420	0.846691	0.843834	0.694092	0.923

Abbreviations: *MCC* Matthews correlation coefficient, *AUC* Area under the receiver operator curve

Model decision of SHAP

The SHAP plot of the CatBoost model, as depicted in Fig. 4, illustrates the profiles of patients at varying levels of risk for developing CKD. This visualization offers insight into individualized care planning strategies informed by the predictive capabilities of the CatBoost model. In Fig. 4, the impact of each feature on the model output is quantified by calculating the average of the

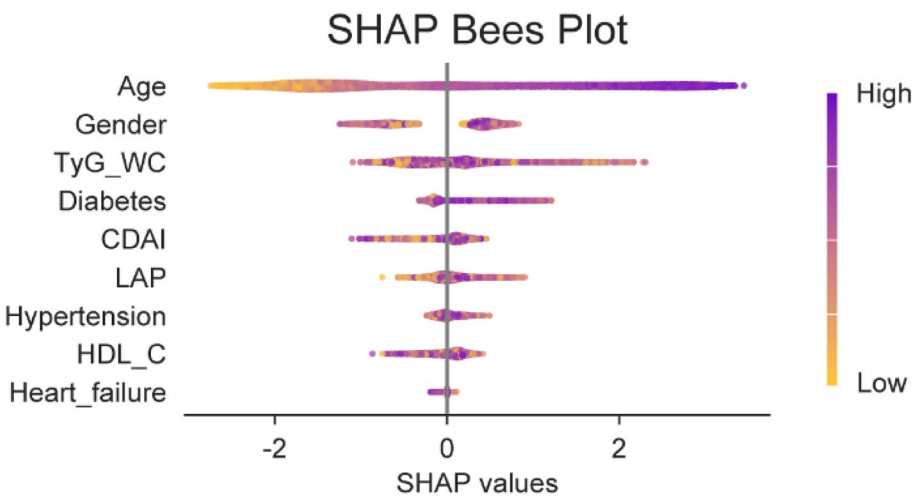

Fig. 4 Importance ranking of SHAP variables based on the CatBoost model. The SHAP summary plot illustrates that the color gradient of the horizontal bars corresponds to the variable magnitude, ranging from purple (high) to yellow (low). The width of the horizontal bars indicates the impact on model predictions, with wider bars suggesting a greater effect

absolute SHAP values across all samples. Age emerged as the most influential predictor, followed by a history of diabetes, hypertension, sex, HDL-C levels, CDAI, TyG-WC, and LAP. The SHAP summary plot illustrates that the color gradient of the horizontal bars corresponds to the variable magnitude, ranging from purple (high) to yellow (low). The width of the horizontal bars indicates the impact on model predictions, with wider bars suggesting a greater effect.

Discussion

This study provides a comprehensive analysis of ML models related to various factors in abdominal obesity individuals combined CKD. ML, a widely used statistical method, utilizes clinical data to create predictive models and evaluate their accuracy. The current research utilizes ML in combination with the NHANES database to explore potential factors related to CKD in individuals with abdominal obesity and their predictive capabilities. In particular, this study examined LAP and TyG-WC as significant risk factors, as well as HDL-C and CDAI as protective factors associated with CKD.

The clinical diagnosis of CKD relies predominantly on the assessment of GFR or albuminuria, necessitating repeated measurements over a period exceeding three months [37]. Diagnosing CKD can be challenging, particularly in its initial stages, as certain renal functions may be compromised prior to the onset of symptoms [38]. Timely identification of CKD is imperative for predicting its prognosis. Diabetes, hypertension, and obesity continue to be significant contributors to the prevalence of CKD risk factors [2, 39].

The concept of LAP as a predictor of cardiovascular risk was introduced by Kahn et al. as a mathematical model incorporating WC and TG levels [40]. LAP has since been recognized as a valuable non-imaging indicator of visceral obesity and metabolic syndrome [41–43]. According to this study, LAP may also lend insight into the presence of abdominal obesity in patients with CKD.

The relationship between LAP and CKD in individuals with abdominal obesity may be attributed to the interaction between adipokines and CKD, suggesting a potential mechanism of action. Initially, dysregulation of adipokine levels has the potential to disrupt the glomerular filtration barrier, resulting in a decline in GFR [44]. Subsequently, adipokines may contribute to renal injury through the mediation of endothelial dysfunction, promotion of oxidative stress, development of atherosclerosis, and induction of inflammation [45–47].

The equilibrium between glucose metabolism and lipid metabolism plays a critical role in preserving optimal kidney function. In instances of pathology, disruptions in glucose-lipid metabolism can result in compromised cellular structure and inflammatory reactions within the kidneys [48–50]. The excessive buildup of visceral fat can hinder insulin clearance and signaling pathways within the pancreatic islets, thereby impacting renal energy provision and potentially causing kidney damage [51, 52]. Both HDL-C and TyG-WC are acknowledged as emerging and reliable markers for evaluating insulin resistance [53, 54]. Triglyceride glucose-waist circumference has garnered increased attention as a potential predictor of fatty liver disease and cardiovascular disease in recent research [55–57]. This study suggests that HDL-C and

TyG-WC may serve as equally effective markers in individuals with CKD and abdominal obesity.

Insulin resistance, a common feature in disorders of fat metabolism, can also contribute to kidney injury by inducing hyperglycemia, metabolic acidosis, and the overproduction of endothelin-1 and aldosterone, ultimately leading to renal tubulointerstitial injury. A further consequence of insulin resistance is the excessive production of reactive oxidative stress, which results in fibrosis of the renal tissue [35]. Additionally, hyperinsulinemia may promote renal vasodilation, causing glomerular hyperfiltration and compromised endothelial function, thereby exacerbating proteinuria excretion [58–60].

The CDAI score is derived by normalizing and combining the intake levels of various antioxidants, including beta-carotene, selenium, vitamins A, C, and E, as well as zinc, copper, and iron [61]. In this study, a nonlinear relationship was found between the CDAI and CKD in patients with abdominal obesity, which is consistent with the findings of Wang et al. [18]. The underlying mechanism between the CDAI and CKD is unclear, but it may be that oxidative stress and inflammation pathways cause kidney injury, which further contributes to the development of CKD. The CDAI score is strongly correlated with the level of oxidative stress. Oxidative stress leads to the infiltration of neutrophils, an increase in protease secretion, and the generation of substantial quantities of oxidative intermediates, contributing to inflammatory processes. First, the overproduction of reactive oxygen species (ROS) impairs cellular structure and function, leading to tissue damage. CKD is accompanied by significant protein, carbohydrate, and lipid oxidation, and these processes lead to lipid peroxidation and the accumulation of advanced glycosylation end products, further exacerbating tissue damage. Oxidative stress can affect a variety of renal functions, including glomerular filtration rate, fluid electrolyte disturbances, elevated blood pressure, and renal sympathetic nerve activity. Second, the activation of the renin–angiotensin–aldosterone system (RAAS) is linked to oxidative stress. RAAS blockers may be renoprotective through mechanisms that attenuate the inflammatory response and resist renal fibrosis [62]. The CDAI functions as an extrinsic modulator of plasma redox balance, inhibiting the generation of ROS and reactive nitrogen species. This inhibition consequently mitigates oxidative stress and decelerates the onset and progression of CKD [63, 64].

Diets with a high dietary antioxidant index tend to be rich in these antioxidants and are therefore able to reduce the level of oxidative stress in the body, which in turn reduces the release of inflammatory factors. Pathogenesis of CKD is heavily influenced by inflammatory factors. When the kidneys are damaged, inflammatory cells infiltrate renal tissues and release inflammatory factors, leading to renal tubular atrophy, tubulointerstitial fibrosis and glomerulosclerosis. These pathological changes gradually destroy the normal structure and function of the kidneys and eventually lead to renal failure. In addition, inflammatory factors are involved in the pathological processes of renal arteriolar sclerosis and ischemia, glomerular hyperperfusion and increased intracapsular pressure, which further exacerbate renal injury [29, 65].

According to the findings of the RCS study and subgroup analysis, the predictive signature had a pronounced impact on the demographics of individuals aged 60–80 years. This predictive signature holds promise in assisting healthcare professionals in tailoring personalized care plans for individuals with chronic kidney disease and abdominal obesity, particularly among elderly patients, by considering dietary and endocrine factors.

The current research underscores the advantages of combining ML methodologies with SHAP techniques to analyze the diverse effects of glucose and lipid metabolism factors on CKD outcomes in individuals with abdominal obesity. Machine learning techniques offer a significant advantage in managing intricate, multidimensional data and identifying nonlinear connections among various variables. This capability is particularly advantageous in the context of CKD, where the progression of the disease is influenced by a multitude of factors, such as age, comorbidities, metabolic disorders, and environmental influences. Conventional risk prediction models frequently encounter difficulties in accommodating these complex interactions, resulting in suboptimal performance [66]. Moreover, the predictive model established in this research may offer effective management strategies for CKD in elderly patients with abdominal obesity.

Study strengths and limitations

This study represents a highly representative and innovative cross-sectional analysis encompassing the entire United States, focusing on the prevalence and severity of CKD in individuals with abdominal obesity. The research utilizes a sophisticated predictive model that incorporates various factors and applies ten different ML algorithms. The ultimate CatBoost model indicates that for males > 60 years of age with diabetes, hypertension, and a history of cardiovascular disease, it is advisable to promote a reduction in waist circumference, decrease fat intake, and enhance the intake of antioxidant-rich diets, alongside effective management of blood glucose levels. This strategy is advantageous for preventing CKD and decelerating its progression. Nevertheless, this study is subject to certain limitations. First, the predictive model utilized in this research was developed using cross-sectional data, implying that the model's predictions do not

establish causal relationships and necessitate further investigation through prospective studies or external validation. Second, all data utilized in this study were sourced from the NHANES database, which is representative of the U.S. population and may not be generalizable to populations in other countries.

Conclusion

This nationally representative study investigated the association of TyG-WC, LAP, HDL-C, and CADI with CKD risk in patients with abdominal obesity, particularly those over the age of 60 years. The study utilized CatBoost models to develop a predictive tool for identifying CKD risk in this population. These findings may facilitate early detection of CKD in patients with abdominal obesity and support informed decision-making by healthcare providers.

Abbreviations

CKD	Chronic kidney disease
NHANES	National Health and Nutrition Examination Survey
TyG-WC	Triglyceride glucose-waist circumference
CDAI	Composite dietary antioxidant index
LAP	Lipid accumulation product
HDL-C	High-density lipoprotein cholesterol
BMI	Body mass index
VAI	Visceral adiposity index
GFR	Glomerular filtration rate
ACR	Albumin-to-creatinine ratio
FPG	Fasting plasm glucose
TG	Triglycerides
TC	Total cholesterol
LASSO	Least absolute shrinkage and selection operator
RCS	Restricted cubic spline
SHAP	Shapley additive explanations
WC	Waist circumference
ROC	Receiver operating curve
AUC	Area under curve
MCC	Matthews correlation coefficient
OR	Odds ratio
CI	Confidential interval
ML	Machine learning
SMOTE	Synthetic minority over-sampling technique
DT	Decision tree
GBDT	Gradient boosting decision tree
LightGBM	Light gradient-boosting machine
LR	Logistic regression
NB	Naive bayes
RF	Random forest
SVM	Support vector machine
XG	EXtreme gradient boosting

Supplementary Information

The online version contains supplementary material available at https://doi.org/10.1186/s12944-024-02384-7.

Supplementary Material 1: Table 1. The resource and definition of Baseline characteristics. Table 2. Subgroup analysis of CKD at different risk levels based on various influencing factors. Table 3. Details of hyperparameter for ML models. Figure 1. Lasso regression of CKD with abdominal obesity related variables. Figure 2. The ROC curve corresponding to the cross-validation.

Supplementary Material 2.

Supplementary Material 3.

Acknowledgements

We are grateful to the National Health and Nutrition Examination Survey for the data provided and to all participants for their selfless dedication.

Authors' contributions

XL.D, LF.M, P.L and MY.H wrote the main manuscript text. RY.J, YD.T and HL.C prepared figures. HY.G, WQ.Z, and K.L prepared Tables. XY.C prepared Supplementary Materials. WC.L and HX.Z designed the manuscript. All authors reviewed the manuscript.

Funding

This research did not receive any specific grant from funding agencies in the public, commercial, or not-for-profit sectors.

Data availability

No datasets were generated or analysed during the current study.

Declarations

Ethics approval and consent to participate

Not applicable.

Consent for publication

All contributing authors were consent for publication.

Competing interests

The authors declare no competing interests.

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Received: 17 September 2024 Accepted: 19 November 2024
Published online: 26 November 2024

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