



Original Article

Risk Factors Associated with Renal Mass Compared to Renal Cyst: A Case-Control Study

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ABSTRACT

Background: Renal mass is a significant global health issue; studying its risk factors relative to benign renal cysts is essential for prevention and risk modification.

Objective: This study aimed to identify renal mass risk factors compared to renal cysts in the Iraqi Kurdistan population.

Patients and Methods: This unmatched case-control study was conducted from October 2025 to February 2026 at two hospitals in Sulaymaniyah, Iraq. The cases comprised 308 adults with renal masses, while the control group comprised 307 adults with renal cysts. Structured interviews, questionnaires, and medical record reviews were used to collect information on sociodemographic, clinical, and lifestyle aspects. Multivariable logistic regression discerned independent risk variables.

Results: Male gender (AOR=0.42 for females, 95% CI: 0.27–0.64, $p<0.001$), obesity (AOR=1.59, 95% CI: 1.20–2.10, $p=0.001$), hypertension (AOR=3.03, 95% CI: 1.79–5.12, $p<0.001$), urinary tract infection (AOR=1.63, 95% CI: 1.02–2.62, $p=0.042$), smoking (AOR=1.66, 95% CI: 1.07–2.58, $p=0.025$), family history of cancer (AOR=4.89, 95% CI: 2.11–11.34, $p<0.001$), inadequate sleep (AOR=2.05, 95% CI: 1.20–3.51, $p=0.008$), and high perceived stress (AOR=2.97, 95% CI: 2.09–4.23, $p<0.001$) were identified as significant risk factors. Healthier dietary patterns were protective (AOR=0.54, 95% CI: 0.35–0.81, $p=0.003$).

Conclusion: Multiple modifiable risk factors were identified, including male gender, obesity, hypertension, urinary tract infection, smoking, inadequate sleep, and high perceived stress. The positive family history for cancer was the highest non-modifiable risk; additionally, healthier dietary patterns were protective. These findings indicate the need for a prevention strategy, particularly to minimize modifiable risk factors in Iraqi Kurdistan.

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Keywords: Renal mass, renal cyst, risk factors, case-control, Iraqi Kurdistan.

1 Introduction

Kidney malignancies constitute a major health concern worldwide, with 400,000 new cases and 175,000 deaths annually [1]. Renal cell carcinoma (RCC) accounts for almost 90 percent of all kidney cancers, making it the ninth most frequent cancer among males and the 14th most frequent cancer among females worldwide [2]. The incidence of kidney cancer is increasing steadily, with predictions for further increases in the future, particularly in developing countries that increasingly follow the Western way of life [3]. The highest incidence rates have been reported in North America and Europe, with developing countries experiencing a high proportion of advanced cases and increased mortality rates [4, 5].

Sexual dimorphism is a notable feature in the epidemiology of kidney cancers worldwide, with males being 1.7–2.0 times more susceptible to kidney cancers than females [6, 7]. The etiology of renal cell carcinoma is multifactorial in origin, with well-established modifiable risk factors including

[8, 9]. Obesity is dose-response related and increases RCC risk by over 2-fold in individuals with a body mass index of 30 kg/m² or greater [10]. Hypertension increases RCC risk by 1.6-fold; evidence shows that for every 10 mmHg increase in blood pressure, RCC risk increases by 5–7% [11, 12]. Cigarette smoking increases RCC risk by 40%, and smoking cessation leads to a gradual decrease in RCC risk [13, 14].

Moreover, the role of heredity is also significant, as it is responsible for 4–8% of the cases that are related to well-characterized syndromes [15, 16]. In addition, family history is responsible for doubling the risk of the condition [17, 18]. Recent studies have shown that dietary habits, especially the intake of fruits and vegetables, are responsible for reducing the risk, while the intake of red meat increases the risk of the condition [8, 17, 19–22].

Differentiating benign renal cysts from malignant masses is an essential issue in medicine. Simple renal cysts occur in 30% of the population over the age of 40 [23]. Renal cysts are mostly benign in nature. On the contrary, complex cystic masses are malignant in nature, ranging from 0% in Bosniak I–II to 90% in Bosniak IV masses. Preoperative differentiation of malignant masses is essential to avoid unnecessary surgeries while providing proper treatment for malignancy [24].

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overweight/obesity, high blood pressure, and cigarette smoking

In the region of Iraqi Kurdistan, cancer ranks third in male cancer incidence. The rates are three to four times higher in men than in women ^[25]. Aggressive characteristics of kidney cancer in the region are documented, with approximately 40% of patients showing disease progression and mortality rates exceeding 70% within five years ^[26]. These unfavorable outcomes are partly due to the significantly lower incidental detection rates of kidney cancer in the region (16%) compared to developed countries ^[27].

Despite significant advances in the epidemiology of RCC in developed countries, knowledge gaps persist regarding the patterns of risk factors in the Middle East. The distinctive genetic characteristics, high rates of consanguineous marriages, eating habits, and possible environmental factors in the region are important issues that are not adequately addressed in the international literature. The present case-control study was designed to systematically investigate the patterns of risk factors for the occurrence of renal masses in comparison to renal cysts in the region of Iraqi Kurdistan. Through a comprehensive assessment of related factors, this study aimed to identify the independent predictors of differentiation between malignant masses and benign cysts.

2 Patients and Methods

2.1 Study Design, Setting, and Ethical Approval

The design used was an unmatched case-control design, conducted between October 2025 and February 2026 at two hospitals in Sulaymaniyah city, Iraq. The case-control design was utilized because of its efficiency in assessing multiple risk factors simultaneously. The study proposal was approved by the Ethics Committee at the University of Sulaimani, College of Nursing (Approval ID: 028/9, May 2025). All steps performed in studies involving human participants were in accordance with the ethical standards of the national research committee and the 1964 Declaration of Helsinki and its later amendments ^[28]. Written informed consent was obtained from all participants before their inclusion in the study, and they were informed of their right to withdraw at any time.

2.2 Study Population

The study population comprised 308 adult patients with radiologically confirmed renal masses diagnosed through ultrasonography, computed tomography, or magnetic resonance imaging, confirmed as malignancy (RCC) histopathologically, and considered as a case group. To minimize misclassification bias, only patients with documented histopathological confirmation of RCC in their medical records were included in the case group. Clinical, radiological, and pathology reports were reviewed during data collection to verify the diagnosis and eligibility of all enrolled cases. The control group consisted of 307 adult individuals with uncomplicated benign renal cysts (Bosniak class I–II), which demonstrate minimal malignancy potential ^[4], and benign renal cysts (Bosniak class III–IV) were excluded. The use of individuals with simple renal cysts as controls may have introduced some degree of selection bias, as renal cysts and renal masses may share certain demographic or metabolic risk

factors. Nevertheless, controls were selected from the same clinical setting as cases to ensure comparability and minimize referral bias. Patients with chronic kidney diseases, congenital anomalies, metastatic lesions, a history of prior kidney cancer, polycystic kidney disease, and insufficient data were excluded. OpenEpi version 3.01 was used to calculate the sample size, which had a 95% confidence level and 80% statistical power. The estimated prevalence of the primary exposure among controls was 10% ^[29], with an anticipated odds ratio of 2.0. The minimum sample size needed was 279 cases and 279 controls (1:1 case-to-control ratio). A 10% contingency was added to account for possible missing data, bringing the total to 620 participants (310 cases and 310 controls). Of 620 individuals, 615 participated and completed the survey (308 renal masses and 307 renal cysts).

2.3 Data Collection and Definitions of Variables

The data were collected from medical records and through structured interviews. The outcome measure of interest was renal mass or renal cyst. The sociodemographic information collected included age, sex, educational level, marital status, occupation, financial status, and place of residence. The clinical information collected included body mass index, categorized according to the World Health Organization criteria ^[30]; hypertension, according to the Joint National Committee-8 criteria ^[31]; diabetes mellitus, according to the American Diabetes Association criteria; renal calculi; urinary tract infection; and family history of cancer. The lifestyle information collected included smoking habits, dietary pattern (using a 10-item scale with 5-point Likert-type scales ranging from 0 [never] to 4 [always]); water consumption; tea/coffee consumption; sweet drink consumption; physical activity level (Quick Physical Activity Rating Scale) ^[32]; sleep duration; perceived stress level (4-item Perceived Stress Scale with 0–16 scores) ^[33]; and analgesic use. Analgesic use was defined as regular use (≥ 3 days a week for ≥ 3 months) of non-steroidal anti-inflammatory drugs or acetaminophen. Dietary intake was assessed using a simplified food frequency questionnaire (FFQ) developed for the present study to ensure ease of administration and comprehension among both patients and healthcare providers. The tool was designed based on established FFQ frameworks and adapted to the local dietary context. It consisted of 10 items covering major food groups and dietary behaviors, including consumption of fruits, vegetables, whole grains, legumes, dairy products, red and processed meats, fried and fast foods, sugary beverages, sweets, and salt intake. Each item was scored on a four-point frequency scale ranging from 0 (never/rarely) to 3 (daily or almost daily), with reverse scoring applied for unhealthy dietary items so that higher total scores consistently indicated healthier dietary practices. The total dietary score ranged from 0 to 30 and was categorized into three levels of dietary risk based on score distribution: low/no to mild risk (<10), moderate risk (10–20), and high risk (>20). Content validity of the questionnaire was established through review by a panel of three experts in nutrition and public health, and necessary modifications were made based on their feedback. The instrument was further pretested in a pilot study to assess clarity, feasibility, and consistency before its application in the main study. Internal consistency reliability

was evaluated during the pilot phase to ensure the appropriateness of the tool for use in the final analysis. Lifestyle-related variables, including dietary habits, physical activity, sleep characteristics, perceived stress, smoking status, and other behavioral factors, were assessed retrospectively through structured participant interviews. Participants in both groups were specifically asked to report their usual lifestyle practices during the 6–12 months preceding their RCC diagnosis. This approach was adopted to better capture pre-diagnostic exposures and reduce the likelihood that lifestyle changes resulting from cancer diagnosis, disease progression, or treatment would influence the reported data.

2.4 Statistical Analysis

The data were analyzed using IBM SPSS Statistics version 26.0. Chi-square was used to compare categorical variables, while the independent t-test was used for continuous variables. A statistically significant level was established at $p \leq 0.05$. Candidate predictors were selected using univariable logistic regression analysis (p values < 0.25), of the 18 variables assessed in univariable analysis, 13 met the $p < 0.25$ threshold and were entered into the multivariable logistic regression model: sex, financial status, BMI, hypertension, diabetes mellitus, urinary tract infection, smoking, family history of cancer, dietary pattern, physical activity, sleep quality, perceived stress, and tea/coffee consumption. Backward elimination was used to derive the final, most parsimonious model, which was further subjected to multiple logistic regression analysis using backward elimination. For assessing multicollinearity among the predictor variables, variance inflation factor values were computed. Adjusted odds ratios

along with 95% confidence intervals were computed. Model fit of the logistic regression model was checked using Hosmer–Lemeshow goodness-of-fit tests, area under the ROC curve, sensitivity, and specificity.

Biologically plausible interaction terms (e.g., sex×smoking, BMI category×hypertension, smoking×hypertension) were tested and found to be statistically non-significant (all $p > 0.05$), supporting a main-effects-only model.

3 Results & Discussion

3.1 The Results

This unmatched case-control study was conducted on 615 participants (307 with simple renal cysts and 308 with renal masses). The findings are presented in four categories: participants' sociodemographic characteristics, clinical data, lifestyle factors, and multivariable analysis.

Sociodemographic Characteristics

The age distribution was comparable between the groups, with 91.9% and 93.2% of the patients with renal cysts and renal masses, respectively, aged 40 years and older ($p = 0.710$). However, the sex distribution was significantly different ($p < 0.001$); the proportion of males was higher in the renal mass (66.9%) compared to the renal cyst (48.2%) group. Financial status was significantly associated ($p = 0.013$) and higher in the renal mass (50.3%) compared to the renal cyst (42.7%) groups. Education, marital status, occupation, and residency were not significantly different (Table 1).

Table 1: Sociodemographic characteristics of renal mass and renal cyst groups.

Variable	Class	Renal Cyst N=307 F (%)	Renal Mass N=308 F (%)
Age (Years)			
	< 40	25 (8.1)	21 (6.8)
	40–60	140 (45.6)	149 (48.4)
	> 60	142 (46.3)	138 (44.8)
Sex*			
	Male	148 (48.2)	206 (66.9)
	Female	159 (51.8)	102 (33.1)
Education			
	No formal	86 (28.0)	101 (32.8)
	Primary	99 (32.2)	89 (28.9)
	Secondary	72 (23.5)	77 (25.0)
	Higher	50 (16.3)	41 (13.3)
Marital Status			
	Married	284 (92.5)	279 (90.6)
	Unmarried	23 (7.5)	29 (9.4)
Financial Status*			
	Low	131 (42.7)	155 (50.3)

Variable	Class	Renal Cyst	Renal Mass
	Medium	136 (44.3)	133 (43.2)
	High	40 (13.0)	20 (6.5)

* $p < 0.05$ (Chi-square test)

Clinical Characteristics

The BMI distribution was significantly different ($p < 0.001$); the proportion of obese patients was higher in the renal mass (46.4%) compared to the renal cyst (28.7%) groups. Hypertension was significantly associated ($p < 0.001$); the proportion of patients with hypertension was higher in the renal mass (54.2%) compared to the renal cyst (35.8%) groups. The proportion of patients with urinary tract infections was higher in the renal mass (78.9%) compared to the renal cyst (70.4%)

groups ($p = 0.015$). The family history of cancer was significantly different ($p < 0.001$); the proportion of patients with a family history of cancer was higher in the renal mass (11.4%) compared to the renal cyst (3.6%) groups. Stress levels were significantly different ($p < 0.001$); the proportion of patients with high stress levels was higher in the renal mass (23.1%) compared to the renal cyst (9.8%) groups. Diabetes mellitus and renal stones were not significantly different (Table 2).

Table 2: Clinical characteristics of renal mass and renal cyst groups.

Variable	Class	Renal Cyst	Renal Mass
		N=307 F (%)	N=308 F (%)
BMI*			
	Normal	53 (17.3)	57 (18.5)
	Overweight	166 (54.1)	108 (35.1)
	Obese	88 (28.7)	143 (46.4)
Hypertension*			
	No	197 (64.2)	141 (45.8)
	Yes	110 (35.8)	167 (54.2)
Diabetes Mellitus			
	No	210 (68.4)	196 (63.6)
	Yes	97 (31.6)	112 (36.4)
Urinary Tract Infection*			
	No	91 (29.6)	65 (21.1)
	Yes	216 (70.4)	243 (78.9)
Family History of Cancer*			
	No	296 (96.4)	273 (88.6)
	Yes	11 (3.6)	35 (11.4)
Stress Level*			
	Low	107 (34.9)	63 (20.5)
	Moderate	170 (55.4)	174 (56.5)
	High	30 (9.8)	71 (23.1)

* $p < 0.05$ (Chi-square test); BMI = Body Mass Index

Lifestyle and Behavioral Factors

Smoking showed a strong, significant relationship ($p < 0.001$); smoking history was high in the renal mass group (48.7%) compared with the renal cyst group (28.0%). Differences in dietary patterns were significant ($p < 0.001$); more patients in the renal mass group had high-risk dietary patterns (52.6%) than in

the renal cyst group (37.8%). Differences in tea and coffee consumption were significant ($p < 0.001$). The proportion of poor sleep quality was significantly higher in the renal mass group than the renal cysts group ($p = 0.0067$). Plain water consumption and sweet drink consumption did not show significant differences (Table 3).

Table 3: Lifestyle and behavioral factors of renal mass and renal cyst groups.

Variable	Class	Renal Cyst	Renal Mass
		N=307 F (%)	N=308 F (%)

Variable	Class	Renal Cyst	Renal Mass
Smoking*			
	Never Smoke	221 (72.0)	157 (51.3)
	Ever Smoke	86 (28.0)	150 (48.7)
Diet Risk*			
	High (> 20)	116 (37.8)	162 (52.6)
	Moderate (10-20)	191 (62.2)	146 (47.4)
Tea/Coffee Consumption*			
	1–2 Cups/day	119 (38.8)	152 (49.4)
	3–4 Cups/day	179 (58.3)	132 (42.9)
	≥5 Cups/day	9 (2.9)	24 (7.8)
Sweet Beverages			
	0 Glass/day	114 (37.1)	103 (33.4)
	1–2 Glass/day	178 (58.0)	184 (59.8)
	≥3 Glass/day	15 (4.9)	21 (6.8)
Water Intake			
	< 8 glasses/day	119 (38.8)	126 (40.9)
	≥ 8 glasses/day	179 (58.2)	182 (59.1)
Physical Activity*			
	Inactive	194 (63.2)	223 (72.4)
	Moderately Active	113 (36.8)	85 (27.6)
Sleep Quality*			
	Poor (< 6 hours)	151(49.2)	185 (60.1)
	Normal (≥ 6 hours)	156 (50.8)	123 (39.9)

* $p < 0.05$ (Chi-square test)

Multivariable Logistic Regression Analysis

The model demonstrated acceptable discrimination, with an area under the ROC curve of 0.747 (95% CI: 0.707–0.784). At a predicted probability cutoff of 0.50, the model correctly classified 68.5% of renal mass cases (sensitivity) and 69.1% of renal cyst controls (specificity). The final multivariable model was statistically significant ($\chi^2 = 154.80$, $df = 20$, $p < 0.001$). Nagelkerke $R^2 = 0.297$ explained about 29.7% of the variance. The overall classification accuracy of the model was 70.2%. Variance inflation factor (VIF) values for all predictors ranged from 1.07 to 1.34 (tolerance 0.74–0.93), indicating no evidence of problematic multicollinearity. The Hosmer–Lemeshow goodness-of-fit test indicated an adequate model fit ($\chi^2 = 14.03$, $df = 8$, $p = 0.081$), confirming no significant difference between observed and predicted classifications.

Female gender was associated with significantly lower odds (AOR=0.42, 95% CI: 0.27–0.64, $p < 0.001$); males were 2.4

times more likely to have a renal mass than renal cysts. BMI category showed significance (AOR=1.59, 95% CI: 1.20–2.10, $p = 0.001$). Hypertension increased the risk threefold (AOR=3.03, 95% CI: 1.79–5.12, $p < 0.001$). Urinary tract infection increased the risk by 63% (AOR=1.63, 95% CI: 1.02–2.62, $p = 0.042$). Smoking elevated the risk by 66% (AOR=1.66, 95% CI: 1.07–2.58, $p = 0.025$). Family history showed the strongest association, increasing the risk nearly five times (AOR=4.89, 95% CI: 2.11–11.34, $p < 0.001$). Healthy dietary habits were found to be protective (AOR=0.54, 95% CI: 0.35–0.81, $p = 0.003$). Sleep duration of less than 6 hours increased the risk twofold (AOR=2.05, 95% CI: 1.20–3.51, $p = 0.008$). Perceived stress levels showed strong significance (AOR=2.97, 95% CI: 2.09–4.23, $p < 0.001$). Education, marital status, financial status, diabetes, water intake, sweet beverage, physical activity, and coffee consumption did not maintain significance (Table 4).

Table 4: Multivariable logistic regression analysis of risk factors for renal mass.

Variable	B	SE	p-value	AOR	95% CI	VIF (Tolerance)
Gender (female vs male) *	−0.879	0.224	<0.001	0.42	0.27–0.64	1.25 (0.80)
BMI category*	0.463	0.143	0.001	1.59	1.20–2.10	1.07 (0.93)
Hypertension*	1.107	0.269	<0.001	3.03	1.79–5.12	1.09 (0.92)

Variable	B	SE	p-value	AOR	95% CI	VIF (Tolerance)
Urinary tract infection*	0.491	0.241	0.042	1.63	1.02–2.62	1.24 (0.81)
Smoking*	0.506	0.225	0.025	1.66	1.07–2.58	1.34 (0.74)
Family history of cancer*	1.587	0.429	<0.001	4.89	2.11–11.34	1.12 (0.89)
Diet pattern*	−0.624	0.211	0.003	0.54	0.35–0.81	1.19 (0.84)
Sleep duration*	0.719	0.273	0.008	2.05	1.20–3.51	1.30 (0.77)
Perceived stress*	1.089	0.180	<0.001	2.97	2.09–4.23	1.23 (0.81)

* $p < 0.05$; AOR = Adjusted Odds Ratio; CI = Confidence Interval; VIF = Variance Inflation Factors; SE = Standard Error; BMI = Body Mass Index

Model statistics: $\chi^2 = 154.80$, $df = 20$, $p < 0.001$; Cox & Snell $R^2 = 0.223$; Nagelkerke $R^2 = 0.297$; Overall classification accuracy = 70.2%

3.2 Discussion

This unmatched case-control study has identified several independent risk factors for differentiating renal masses from renal cysts in the Iraqi Kurdistan region. The data suggest consistency with the international literature, but also some unique characteristics that should be considered for the implementation of preventive strategies.

Being male has been identified as an independent risk factor, consistent with global epidemiological data [6, 7]. This could be due to hormonal, occupational, or behavioral factors. Obesity has also been identified as an independent risk factor, consistent with extensive literature regarding its association with RCC through various mechanisms [9, 10]. A significant dose-response relationship has also been identified between body mass index and RCC, which emphasizes the importance of controlling obesity for preventing RCC in the Iraqi Kurdistan region.

Hypertension has also been identified as the most significant risk factor for RCC, consistent with the international literature [11, 12]. Although the exact mechanism for the association of hypertension and RCC is unknown, it may be due to renal damage, oxidative stress, or genetic factors. It is interesting to note that the prevalence of hypertension among cases (45.2%) is the highest; therefore, controlling hypertension is an important measure for preventing RCC. Tobacco has also been identified as an independent risk factor for RCC, consistent with extensive literature regarding the carcinogenic effects of smoking [13–15]. Cigarette smoke contains many carcinogenic components that accumulate in the kidneys, thereby increasing the risk of RCC. Smoking cessation has been found to reverse the risk for RCC.

Familial history of cancer showed the strongest association with a fivefold increase in risk, consistent with reports of familial RCC syndromes and evidence of familial aggregation of RCC [16–18]. The relatively high proportion of familial cancer among patients (8.8%) compared with Western populations may also be due to founder effects, consanguineous marriages, and unique environmental factors prevalent in this region. Urinary tract infections showed a moderate association with renal masses. The literature is controversial on this topic, but chronic inflammation and recurrent infections may contribute to carcinogenesis through oxidative stress and DNA damage. However, it is also possible that urinary tract infections may result from or co-exist with renal masses.

Significant findings were also obtained for sleep duration and perceived stress levels in this study population. Inadequate sleep resulted in a twofold increase in risk for renal masses. The

reasons for this association may include disruptions in circadian rhythms and associated effects on immune function and metabolism [34]. Sleep disorders and obstructive sleep apnea have shown a link with RCC [35]. Higher levels of perceived stress resulted in a near threefold increase in risk for renal masses, possibly due to the effects of chronic activation of the stress system and associated lifestyle factors such as diet and exercise [36]. Furthermore, a protective association for dietary quality is consistent with an emerging body of evidence for plant-based dietary patterns and RCC risk reduction [8, 17, 19–22]. The traditional diet, rich in plant-based foods and low in processed and fast food, may provide a protective benefit for RCC risk reduction.

This present study has several strengths in terms of sample size, response rate, assessment of risk factors, and statistical methods used. However, it is important to acknowledge several limitations. First, the case-control design makes it impossible to establish causality, and there is a potential for recall bias. There is potential for selection bias in this study, despite every attempt to select consecutive patients. Although lifestyle exposures were assessed for the period preceding RCC diagnosis, the retrospective nature of data collection may still be subject to recall bias, and some degree of reverse causality cannot be completely excluded. Data on occupational exposures, environmental toxins, alcohol consumption, and detailed family history of RCC were not systematically collected during the study period. Although most participants reported no alcohol intake during clinical interviews, information on occupational and environmental exposures was not available from either patient reports or medical records. Similarly, family history of RCC was inconsistently documented. Therefore, these variables could not be included in the analysis, which may introduce unmeasured confounding. Adjustments for multiple variables cannot completely avoid residual confounding. There is also a limitation in establishing a temporal relationship in this cross-sectional assessment of lifestyle factors. The generalizability of the findings is limited due to the hospital-based design conducted in Sulaymaniyah. The study population was drawn from patients attending a tertiary care facility, which may not fully represent the broader community or populations in other regions. Therefore, caution is warranted when extrapolating these findings to the general population. Future multi-center and population-based studies are recommended to enhance external validity and confirm these results in more diverse settings.

Conclusion

In conclusion, this investigation identified multiple modifiable and non-modifiable risk factors distinguishing renal masses from renal cysts. The significant impact of modifiable factors, including obesity, hypertension, smoking, sleep deprivation, stress, and inadequate dietary quality, suggests a substantial opportunity for primary prevention. These results substantiate the necessity for comprehensive prevention strategies aimed at lifestyle modification, chronic disease management, and improved surveillance for high-risk individuals in the Iraqi Kurdistan population. Subsequent research should concentrate on prospective cohort studies, genetic susceptibility analyses, and preventive intervention trials to corroborate and expand these findings.

Authors Contribution

Both authors made equal and substantial contributions to all aspects of this work, including conceptualization, study design, data collection, data analysis and interpretation, manuscript preparation, and final approval of the submitted version.

Conflict of Interests

The authors declare no conflicts of interest. The funders had no role in the design, data collection, analysis, manuscript writing, or decision to publish.

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References

- [1] Cirillo, L., Innocenti, S. & Becherucci, F. Global epidemiology of kidney cancer. *Nephrol. Dial. Transplant.* **39**, 920–928, doi: 10.1093/ndt/gfae036 (2024).
- [2] Padala SA, Barsouk A, Thandra KC, Saginala K, Mohammed A, Vakiti A, Rawla P, Barsouk A.. Epidemiology of renal cell carcinoma. *World J Oncol* **11**, 79–87, doi: 10.14740/wjon1279 (2020).
- [3] Bukavina, L., Bensalah, K., Bray, F., Carlo, M., Challacombe, B., Karam, J. A., Kassouf, W., Mitchell, T., Montironi, R., O'Brien, T., Panebianco, V., Scelo, G., Shuch, B., van Poppel, H., Blosser, C. D. & Psutka, S.. Epidemiology of renal cell carcinoma: 2022 update. *Eur Urol* **82**, 529–542, doi: 10.1016/j.eururo.2022.08.019 (2022).
- [4] Ljungberg B, Albiges L, Abu-Ghanem Y, Bedke J, Capitanio U, Dabestani S, Fernández-Pello S, Giles RH, Hofmann F, Hora M, Klatte T, Kuusk T, Lam TB, Marconi L, Powles T, Tabbaz R, Volpe A, Bex A. European Association of Urology Guidelines on Renal Cell Carcinoma: The 2022 Update. *Eur Urol* **82**, 399–410, doi: 10.1016/j.eururo.2022.03.006 (2022).
- [5] Young M, Jackson-Spence F, Beltran L, Day E, Suarez C, Bex A, Powles T, Szabados B.. Renal cell carcinoma. *Lancet* **404**, 476–491, doi: 10.1016/S0140-6736(24)01240-0 (2024).
- [6] Ning, K., Peng, Y., Jiang, Y., Li, Z., Luo, X., Lin, L., Deng, M., Wu, Y., Huang, T., Huang, Y., Xie, Y., Yang, X., Zhang, M., Xiong, L., Zou, X., Zhou, Z., Zhou, F., Dong, P., Yu, C. & Zhang, Z. Sex differences in renal cell carcinoma: a single-cell analysis reveals exhausted CD8⁺ T-cells highly infiltrated in males. *Biol Sex Differ* **14**, 58, doi: 10.1186/s13293-023-00540-9 (2023).
- [7] Ladurner, M., Lindner, A. K., Rehder, P., Tulchiner, G. The influence of sex hormones on renal cell carcinoma. *Ther Adv Med Oncol* **16**, doi: 10.1177/17588359241269664 (2024).
- [8] Campi, R., Stewart, G. D., Staehler, M., Dabestani, S., Kuczyk, M. A., Shuch, B. M., Finelli, A., Bex, A., Ljungberg, B. & Capitanio, U. Novel liquid biomarkers and innovative imaging for kidney cancer diagnosis: what can be implemented in our practice today? A systematic review of the literature. *Eur Urol Oncol* **6**, 22–36, doi: 10.1016/j.euo.2022.09.004 (2023).
- [9] Venkatesh, N., Martini, A., McQuade, J. L., Msaouel, P., Hahn, A. W. Obesity and renal cell carcinoma: Biological mechanisms and perspectives. *Semin Cancer Biol* **94**, 21–33, doi: 10.1016/j.semcancer.2023.05.006 (2023).
- [10] Graff, R. E., Wilson, K. M., Sanchez, A., Chang, S. L., McDermott, D. F., Choueiri, T. K., Cho, E., Signoretti, S., Giovannucci, E. L. & Preston, M. A. Obesity in relation to renal cell carcinoma incidence and survival in three prospective studies. *Eur Urol* **82**, 247–251, doi: 10.1016/j.eururo.2022.04.032 (2022).
- [11] Kim, C. S., Han, K. D., Choi, H. S., Bae, E. H., Ma, S. K., Kim, S. W. Association of hypertension and blood pressure with kidney cancer risk: a nationwide population-based cohort study. *Hypertension* **75**, 1439–1446, doi: 10.1161/HYPERTENSIONAHA.120.14820 (2020).
- [12] Wang, L., Du, H., Sheng, C., Dai, H. & Chen, K. Association between metabolic syndrome and kidney cancer risk: a prospective cohort study. *Lipids Health Dis.* **23**, 142, doi: 10.1186/s12944-024-02138-5 (2024).
- [13] Kime, S., White, N., Pelucchi, C., Boffetta, P., Brennan, P., Zaridze, D. Cigarette smoking and risk of renal cell carcinoma: a pooled analysis of population-based studies. *Cancer Epidemiol Biomarkers Prev* **29**, 1885–1893, doi: 10.1158/1055-9965.EPI-20-0326 (2020).
- [14] Hung, R. J., Christiani, D. C., Risch, A., Popanda, O., Haugen, A., Zienolddiny, S. International Lung Cancer Consortium: pooled analysis of sequence variants in DNA repair and cell cycle pathways. *Cancer Epidemiol Biomarkers Prev* **30**, 618–632, doi: 10.1158/1055-9965.EPI-20-0719 (2021).
- [15] Han, S., Zhao, S., Zhong, R., Li, P., Pang, Y., He, S., Duan, J., Gong, H., Shi, J., Liu, L., Yan, Y. Global burden, trends, and disparities in kidney cancer attributable to smoking from 1990 to 2021. *Front Public Health* **12**, 1506542, doi: 10.3389/fpubh.2024.1506542 (2024).
- [16] Garutti M, Foffano L, Mazzeo R, Michelotti A, Da Ros L, Viel A, Miolo G, Zambelli A, Puglisi F. Hereditary Cancer Syndromes: A Comprehensive Review with a Visual Tool. *Genes (Basel)* **14**, 1025, doi: 10.3390/genes14051025 (2023).
- [17] Jakobsson, R. G., Nasic, S., Bratt, O., Johansson, M. E., Grenabo Bergdahl, A. Family History and Risk of Renal Cell Carcinoma: A National Multiregister Case-Control Study. *J Urol* **211**, 71–79, doi: 10.1097/JU.0000000000003740 (2024).
- [18] Yngvadottir B, Andreou A, Bassaganyas L, Larionov A, Cornish AJ, Chubb D, Saunders CN, Smith PS, Zhang H, Cole Y, Research Consortium GE, Larkin J, Bowning L, Turajlic S, Litchfield K, Houlston RS, Maher ER. Frequency of pathogenic germline variants in cancer susceptibility genes in 1336 renal cell carcinoma cases. *Hum Mol Genet* **31**, 3001–3011, doi: 10.1093/hmg/ddac094 (2022).
- [19] Liao Z, Fang Z, Gou S, Luo Y, Liu Y, He Z, Li X, Peng Y, Fu Z, Li D, Chen H, Luo Z. The role of diet in renal cell carcinoma incidence: An umbrella review of meta-analyses of observational studies. *BMC Med* **20**, 39, doi: 10.1186/s12916-022-02229-w (2022).
- [20] Heravi, G., Yazdanpanah, O., Podgorski, I., Matherly, L. H., Liu, W. Lipid metabolism reprogramming in renal cell carcinoma. *Cancer Metastasis Rev* **41**, 17–31, doi: 10.1007/s10555-021-10006-w (2022).
- [21] Zhang, S., Jia, Z., Yan, Z., Yang, J. Consumption of fruits and vegetables and risk of renal cell carcinoma: a meta-analysis of observational studies. *Oncotarget* **8**, 27892–27903, doi: 10.18632/oncotarget.15425 (2017).
- [22] HeHe X, Hou J, Liu L, Chen X, Zhang L, Pang C, Tong Y, Li H, Chen F, Peng R, Shi Z. Dietary fiber consumption and outcomes of different cancers: An umbrella review. *Food Nutr Res* **69**, 11034, doi: 10.29219/fnr.v69.11034 (2025).
- [23] Goksu, S. Y., Vadakekut, E. S. & Khattar, D. Renal cystic disease. In StatPearls [Internet]. (StatPearls Publishing, Treasure Island (FL),

updated 23 October 2023).
<https://www.ncbi.nlm.nih.gov/books/NBK554504/> (2023)

- [24] Satariano, M., Ghose, S., Raina, R. The Pathophysiology of Inherited Renal Cystic Diseases. *Genes (Basel)* **15**, 91, doi: 10.3390/genes15010091 (2024).
- [25] M-Amen K, Abdullah OS, Amin AMS, Mohamed ZA, Hasan B, Shekha M, Najmuldeen HH, Rahman FM, Housein Z, Salih AM, Mohammed AS, Sulaiman LR, Barzingi BT, Mahmood D, Othman HE, Mohammad DK, Salih FM, Ali SAK, Mohamad TS, Mahmood K, Othman GO, Aali MH, Qader G, Hussien BM, Awla FA, Kareem SW, Qadir FA, Taher DM, Salihi A. Cancer Incidence in the Kurdistan Region of Iraq: Results of a Seven-Year Cancer Registration in Erbil and Duhok Governorates. *Asian Pac J Cancer Prev* **23**, 601–615, doi: 10.31557/APJCP.2022.23.2.601 (2022).
- [26] Ali, S., Hama, H. T., Hassan, N. A., Karim, A. J., Jalal, P. J., Amin, K.. Treatment outcomes and survival patterns of renal cell carcinoma in Iraqi Kurdistan. *Cancer Rep* **6**, e1789, doi: 10.1002/cnr2.1789 (2023).
- [27] Salih FM, Omar SS, Hamza HT, Namiq KS, Ameen HRM, Rasul KI, Ismael SA. Long-term outcomes and survival rates of renal cell carcinoma patients in Erbil, Iraq: A follow-up study. *BMC Cancer* **25**, 384, doi: 10.1186/s12885-025-13506-7 (2025).
- [28] World Medical Association. World Medical Association Declaration of Helsinki: ethical principles for medical research involving human subjects. *JAMA* **310**, 2191–2194, doi: 10.1001/jama.2013.281053 (2013).
- [29] Larcher A, Campi R, Bex A, Bray F, Bukavina L, Jonasch E, Jemal A, Marston Linehan W, Marandino L, Mir MC, Mollica V, Shuch B, Stewart GD, Sung H, Tran M, Kutikov A. Epidemiology of renal cancer: incidence, mortality, survival, genetic predisposition, and risk factors. *Eur Urol* **88**, 341–358, doi: 10.1016/j.eururo.2025.06.005 (2025).
- [30] World Health Organization. Obesity: preventing and managing the global epidemic. Report of a WHO consultation. *World Health Organ Tech Rep Ser* **894**, i–xii, 1–253 (2000).
- [31] James, P. A., Oparil, S., Carter, B. L., Cushman, W. C., Dennison-Himmelfarb, C., Handler, J., Lackland, D. T., LeFevre, M. L., MacKenzie, T. D., Ogedegbe, O., Smith, S. C. Jr, Svetkey, L. P., Taler, S. J., Townsend, R. R., Wright, J. T. Jr, Narva, A. S. & Ortiz, E. 2014 evidence-based guideline for the management of high blood pressure in adults: report from the panel members appointed to the Eighth Joint National Committee (JNC 8). *JAMA* **311**, 507–520, doi: 10.1001/jama.2013.284427 (2014).
- [32] Galvin, J. E., Tolea, M. I., Rosenfeld, A., Chrisphonte, S. The Quick Physical Activity Rating (QPAR) scale: a brief assessment of physical activity in older adults with and without cognitive impairment. *PLoS One* **15**, e0241641, doi: 10.1371/journal.pone.0241641 (2020).
- [33] Cohen, S., Kamarck, T., Mermelstein, R. A global measure of perceived stress. *J Health Soc Behav* **24**, 385–396 (1983).
- [34] Park, H. K., Choi, W. S., Cho, J. H. The incidence of renal cell carcinoma is increased in patients with obstructive sleep apnea. *Sleep Med Res* **13**, 81–84, doi: 10.17241/smr.2022.01281 (2022).
- [35] Blask, D. E. Melatonin, sleep disturbance, and cancer risk. *Sleep Med Rev* **13**, 257–264, doi: 10.1016/j.smr.2008.07.007 (2009).
- [36] Wang L, Liao WC, Tsai CJ, Wang LR, Mao IF, Chen CC, Kao PF, Yao CC. The effects of perceived stress and lifestyle leading to breast cancer. *Women Health* **53**, 20–40, doi: 10.1080/03630242.2012.732680 (2013).