

## Original article

## Effect of obesity on histological type of renal tumor: The Uroccr study 69

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## ABSTRACT

Renal cell carcinomas (RCC) are increasing worldwide and obesity is one of its recognized causal factor. Obesity thus could be differently associated with the different histologic types of renal tumors and also impact prognosis and patient care.

**Purpose:** The primary objective was to evaluate an association between obesity and histological type of RCC. The secondary objective was to assess if there was an effect of obesity on tumor characteristics.

**Methods:** A multicenter comparative descriptive study was carried out in nearly 30 French hospitals. Histological features of operated RCC of patients included between 2007 and 2020 were compared according to their BMI. Obesity was defined as a BMI  $\geq 30$  kg/m<sup>2</sup>, with a  $P < 0.05$ .

**Results:** In total, 6749 RCC were analyzed, including 1687 (25%) from obese patients. This population was more frequently diabetic (61% versus 41%,  $P < 0.001$ ) and hypertensive (27% versus 11%,  $P < 0.001$ ). Regarding

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histological evaluation, the obese group presented clearer cell carcinoma. Other types presented odd ratios below 1 on multivariate analysis,  $P < 0.01$  except for type II papillary RCC. Tumor size (pT) and nuclear ISUP grade were both lower when BMI was above  $30 \text{ kg/m}^2$ : 72.4% of size pT 1–2 versus 68.5% ( $P = 0.026$ ) and 50% of nuclear ISUP grade 1–2 versus 43.3% for the non-obese group ( $P < 0.0001$ ). There was no difference in survival due to an insufficient number of events.

**Conclusion:** In this large cohort study, we report novel data on a positive association between obesity and histological type of RCC, in particular clear cell RCC.

**Level of evidence:** 3.

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## R É S U M É

Les tumeurs malignes du rein ont une incidence croissante et sont favorisées par l'obésité. Cette association serait variable selon le type histologique. Aussi, l'impact de l'obésité sur la survie du carcinome rénal fait débat.

**Objectifs:** Caractériser la relation entre l'obésité et le type histologique de tumeur rénale et évaluer l'influence de l'obésité sur les caractéristiques tumorales pronostiques.

**Méthodes:** Étude descriptive comparative multicentrique réalisée dans près de 30 hôpitaux français. Les caractéristiques histologiques des CCR opérés des patients inclus entre 2007 et 2020 ont été comparées en fonction de leur IMC. L'obésité était définie comme un  $\text{IMC} \geq 30 \text{ kg/m}^2$ , avec un  $p < 0,05$ .

**Résultats:** Au total, 6749 patients opérés de tumeurs rénales ont été inclus, 25 % des sujets étaient obèses. Ces derniers étaient plus souvent diabétiques (61,2 % versus 41,2 %,  $p < 0,001$ ) et hypertendus (27,2 % versus 11,4 %,  $p < 0,001$ ) que les sujets normopondérés. Concernant l'évaluation histologique, le groupe obèse présentait plus de carcinomes rénaux à cellules claires avec des *odd ratios* inférieurs à 1 en analyse multivariée pour les carcinomes chromophobe, papillaire de type 1, angiomyolipome et,  $p < 0,01$ . La taille de la tumeur (pT) et le grade ISUP étaient tous deux inférieurs lorsque l'IMC était supérieur à  $30 \text{ kg/m}^2$  : 72,4 % de la taille pT 1–2 contre 68,5 % ( $p = 0,026$ ) et 50,8 % du grade nucléaire ISUP 1–2 contre 43,3 % pour le groupe non obèse ( $p < 0,0001$ ). Il n'y avait pas eu de différence retrouvée sur la survie du fait d'un nombre insuffisant d'événements.

**Conclusion:** L'obésité semble prédisposer à l'histologie cellules claires mais serait associée à des caractéristiques tumorales plus favorables.

**Niveau de preuve:** 3.

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## 1. Introduction

The diagnosis of malignant kidney tumors has been rising since the 1980s, with kidney cancer becoming the 6th most common cancer in France by 2018, with 15,323 new cases [1]. Meanwhile, obesity, defined by a body mass index (BMI)  $\geq 30 \text{ kg/m}^2$ , leads to various complications, including cancer development [2]. In 2020, 17% of adults in France were obese, with prevalence increasing with age [3].

The rise in renal cancer diagnoses can be largely attributed to the increased use of abdominal imaging, particularly computed tomography (CT), and to the growing prevalence of obesity, an established risk factor for kidney cancer. According to a meta-analysis by Liu et al., the relative risk of renal cancer increases by 1.06 for each additional BMI point ( $\text{kg/m}^2$ ), with a relative risk of 1.76 for obese patients compared to those with a normal weight (1.61–1.91) [4].

There are several histological types of renal cell carcinoma (RCC), each distinguished by genetic characteristics and prognosis [5]. The majority of these cancers are clear cell RCC (80–90%), with 10–15% being papillary RCC (subdivided into two subtypes). Less than 5% are chromophobe RCC, and the remainder consists of several rarer histological types [6]. The relationship between obesity and RCC is assumed to vary according to histological type: many studies suggest a stronger association for clear cell RCC [7–11], with a few also supporting a link between obesity and chromophobe RCC [10,11]. However, most published studies focus on the three main RCC variants or even just clear cell RCC.

Thus, the impact of obesity on the prognosis and survival of renal carcinoma remains unclear [12–20]. Many articles have noted improved survival rates for obese patients, whether in terms of specific, overall, or recurrence-free survival. These results contribute to the concept of the “obesity paradox” [12–18], in which obesity is considered a protective factor for survival. Other studies suggest that obesity leads to poorer

prognosis [19]. Similarly, studies on the relationship between BMI and renal tumor characteristics, such as size and nuclear grade, are heterogeneous and contradictory [14,16,17,20].

The primary objective of this study was to evaluate in a multicenter cohort whether there is an association between obesity and histological type of RCC. Secondary objectives included assessing prognostic characteristics of these tumors and exploring the relationship between obesity and survival.

## 2. Methods

### 2.1. Study population and characteristics

Patients treated surgically for RCC were recruited from centers in the French kidney cancer network, UroCCR, between January 2007 and November 2020. We also analyzed data from patients operated on since 1983 and retrospectively included in the UroCCR network. All patients were included after written consent and anonymization in the prospective national clinical-biological database on kidney cancer, UroCCR (CNIL DR 2013-206 authorization; NCT03293563). Non-operated patients and those lacking demographic or follow-up data were excluded.

### 2.2. Data collection

Clinicopathological, pre-, peri-, and postoperative data were analyzed retrospectively. Age, sex, and personal history of renal cancer were collected preoperatively, along with the mode of diagnosis (incidental, local or general symptoms). We included personal histories of smoking, type 2 diabetes, high blood pressure, and Chronic Kidney Disease (CKD) as these are recognized risk factors for renal cancer. BMI was calculated based on the patient's weight and height at the time of the pre-anesthetic visit, either self-reported by the patient or measured by the anesthetist.

Obesity was defined as a BMI  $\geq 30$  kg/m<sup>2</sup> in accordance with World Health Organization (WHO) criteria. The American Society of Anesthesiologists (ASA) score was also recorded [21], as it defines the overall health status of the patient. RCC data collected included tumor size, stage (according to the TNM 2017 classification) [22], and histological types according to the 2004 WHO classification. These were compared among clear cell RCC, type 1 papillary RCC, type 2 papillary RCC (which were the valid subtypes at the time of the study, now referred to as high-grade and low-grade papillary RCC), chromophobe RCC, oncocytoma, angiomyolipoma, and other RCC types (described in Appendix 1). Nuclear grade (International Society of Urological Pathologists, ISUP) was specified, as well as the presence of a sarcomatoid component and its percentage when applicable. Follow-up was defined from the date of surgery, with monitoring according to national health recommendations. Deaths and time to recurrence (local or distant) were recorded and defined by the time in months between surgery and recurrence during follow-up.

### 2.3. Statistical analysis

Data were integrated into a spreadsheet (Microsoft Excel®). Quantitative variables were described by median (with interquartile range, IQR) and/or mean (with extreme values), and qualitative variables by absolute and percentage values. Statistical analyses were performed using MedCalc Statistical Software version 18.9 (MedCalc Software bvba, Ostend, Belgium; <http://www.medcalc.org>; 2018), and multivariate analysis was performed with SAS Enterprise Guide v8.1. Variables chosen for univariate analysis were based on risk factors identified in the literature review. Significant variables from univariate analyses were then retained for multivariate analysis. As clear cell RCC is the most common RCC, the multivariate analysis calculated odds ratios (OR) comparing the risk of different histologies versus clear cell RCC based on obesity status. BMI groups below and above 30 kg/m<sup>2</sup> (non-obese and obese) were compared using the Chi-square test, with statistical significance set at 0.05.

## 3. Results

### 3.1. Demographic characteristics

The UroCCR database listed 7041 patients. After excluding non-operated patients and those with missing data, 6749 patients were finally included in the analysis. Seventy-four patients were retrospectively included and had been operated on since 1983 (1.1%), while the remainder were prospectively included and operated on between January 2007 and November 2020. The median follow-up was 18 months, with follow-up ranging from 0 to 353 months.

The mean age of the total population was 61 years (IQR: 13.0, ranging from 16 to 96 years). There were 4492 men (66.6%), 20.9% smokers, 46.2% hypertensive, 15.3% diabetics, and 7.7% had chronic kidney disease, including 20.7% hemodialysis patients. A total of 5062 patients had a BMI below 30 kg/m<sup>2</sup> (75.0%), and 1687 were obese.

There were significantly more hypertensive and diabetic patients in the obese population (61.2% vs. 41.2% and 27.5% vs. 15.3%,  $P < 0.001$  for

both cases). Similarly, obese patients were more likely to have incidental diagnoses (72.7% vs. 66.5% in non-obese patients,  $P < 0.0001$ ).

Patient demographics and diagnostic presentation are described in Table 1.

### 3.2. Histological analysis

A significant difference was observed between histological types based on obesity status: patients with a BMI over 30 kg/m<sup>2</sup> had a higher proportion of clear cell renal carcinomas (70.1% versus 60.1%,  $P < 0.0001$ ), with lower rates for all other histological types compared to non-obese patients (all results significant with  $P < 0.0001$ ). No significant difference was found for the sarcomatoid component.

A distribution diagram comparing the two groups compared is shown in Fig. 1.

Men and women had different histology distributions (64.9% of clear cell carcinoma in men versus 58.2% in women,  $P < 0.0001$ ). Since the BMI groups were significantly different for diabetes and hypertension, multivariate analysis considering gender, age, diabetes, hypertension, and obesity was performed.

Obesity was more strongly associated with clear cell RCC, with all other histological types showing a lower Odds Ratio (OR); only type II papillary renal carcinoma did not show a significantly lower OR. This analysis also revealed significant favorable relationships between clear cell RCC and diabetes, as well as between the female gender and angiomyolipoma.

These results are shown in Table 2.

### 3.3. Tumor characteristics

The obese group had less extensive tumors with 72.4% of tumors classified as pT1-pT2 versus 61.5% in non-obese patients ( $P = 0.0063$ ). Obesity was also significantly associated with a lower ISUP nuclear grade, with higher proportions of ISUP 1 and ISUP 2 grades: 6.2% and 44.6%, respectively, versus 3.6% and 39.7% for non-obese patients ( $P < 0.0001$ ).

The distribution of ISUP nuclear grades is shown in Fig. 2.

No significant differences were found in lymph node status or metastatic status with equivalent pN and pM stages for both groups.

Results regarding size and grade are presented in Table 3.

### 3.4. Survival data

Among the 6749 patients analyzed, 1222 experienced tumor progression (18.1%) including 536 local recurrences and 945 metastatic evolutions (259 patients had both local and distant progression). The median time to recurrence was 103 months (ranging from 0 to 308 months). In all, 307 of obese patients have a tumor recurrence, which was equivalent to the group of 915 non-obese patients with progression (18.2% versus 18.1%,  $P = 0.86$ ).

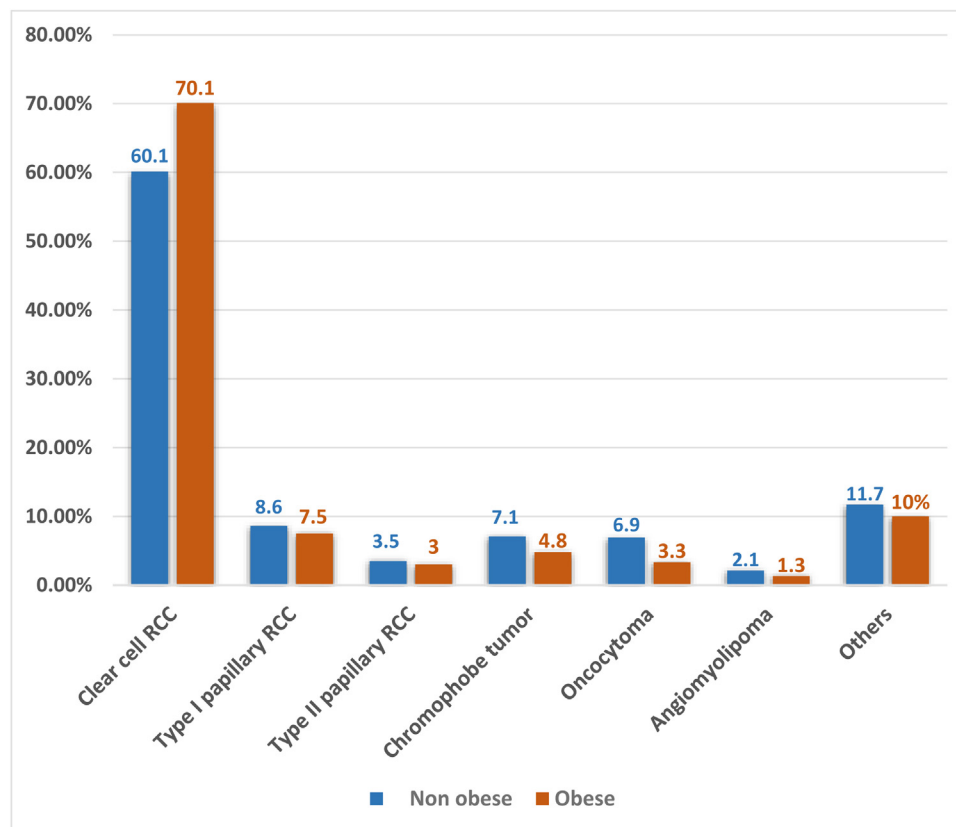
During follow-up, 48 patients died, including 11 obese patients. Comparative statistics were not calculated due to the expected low statistical power given the small number of deaths.

**Table 1**

Demographic data of the operated subjects.

	Population (6749)	Non-obese (5062)	Obese (1687)	P-value
Mean age	61 (IQR: 13.1)	61 (IQR: 13.2)	61 (IQR: 12.6)	NS
Median ASA score	2	2	2	NS
Women	2257 (33.4%)	1681 (33.2%)	576 (34.1%)	NS
Men	4492 (66.6%)	3381 (66.8%)	1111 (65.9%)	
Smoker	1412 (20.9%)	1040 (20.5%)	373 (22.1%)	NS
High blood pressure	3116 (46.2%)	2085 (41.2%)	1031 (61.2%)	< 0.001
Diabetic	1036 (15.3%)	577 (11.4%)	459 (27.2%)	< 0.001
CKD	521 (108 dialysis)	394 (85 dialysis)	127 (22 dialysis)	NS
Asymptomatic at diagnosis	4592 (68.8%)	3366 (66.5%)	1226 (72.7%)	< 0.001

IQR: interquartile range; NS: non significant.



**Fig. 1.** Proportions (in percentage) of histological types according to BMI. It represents the distribution of different histologies of renal cancer among obese population (indicated by orange bars) and non-obese population (indicated by blue bars).

**Table 2**

Multivariate analysis comparing histological subtypes to clear cell RCC in case of obesity.

Histology	Odd ratio	95% confidence interval	P-value
Type I papillary RCC	0.732	0.589–0.909	0.0048
Type II papillary RCC	0.783	0.563–1.089	NS
Chromophobe tumor	0.631	0.488–0.816	0.0004
Oncocytoma	0.438	0.325–0.591	< 0.001
Angiomyolipoma	0.538	0.332–0.872	0.0118
Others	0.739	0.611–0.895	0.0019

Multivariate analysis was controlled for age, gender, diabetic status and hypertensive status. NS: non significant.

#### 4. Discussion

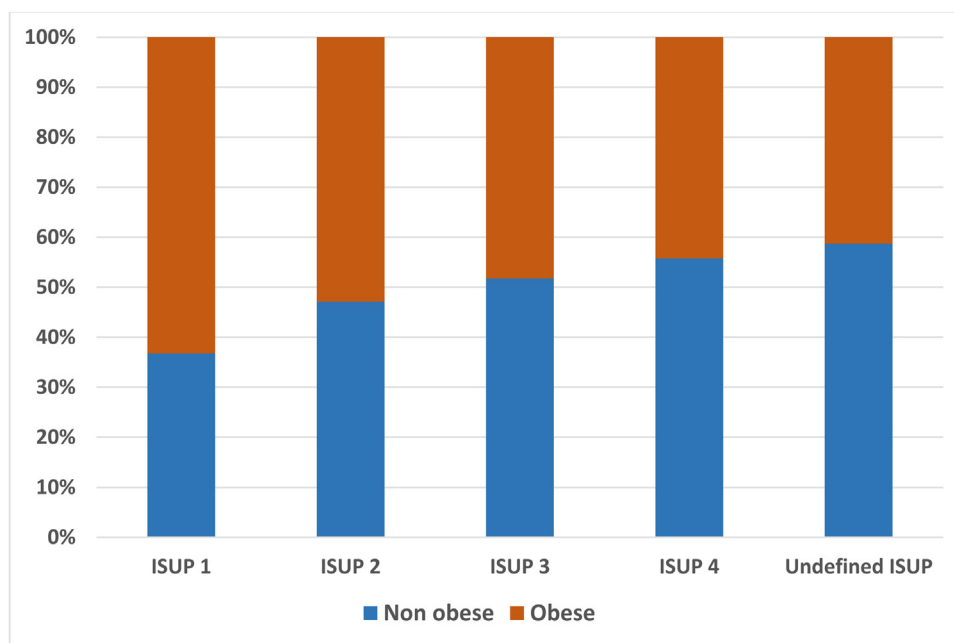
The present study reports a significant association between obesity and clear cell RCC in a french population, with all other histological types showing lower odd ratios in multivariate analysis. To our knowledge, we report here the results of the largest cohort on this association, and they tend to confirm those recently reported in the literature. In the present study, there was no positive association between obesity and chromophobe RCC. Therefore, this association remains rather imprecise since previous studies suggested a positive trend: Purdue et al. multinational cohort showed obesity to be significantly associated with chromophobe RCC with an OR of 1.2 (95% CI 1.1–1.2) for a gain of 5 points in BMI, with a unilinear increase through the BMI categories and loss of significance when BMI exceeded 35 kg/m<sup>2</sup> [10]. In Wang et al. Chinese cohort, median BMI for the chromophobe carcinoma group corresponded to overweight category [9].

The histological types of kidney cancer are different conditions with distinct cellular origins within the renal parenchyma and which occur via different biological pathways, as demonstrated by the numerous somatic mutations identified to date [23]. In sporadic clear cell RCC, almost 90% of cases have altered VHL gene expression [24], either by direct mutation or hypermethylation, leading to increased hypoxia-inducible factor (HIF) activity.

Several interactions with obesity are possible: firstly, obesity could easily be associated with repression of the VHL gene, since an increase of the methylation rate has been shown in obese patients [25] and obesity-induced chronic inflammation is accompanied by an increased amount of reactive oxygenated genotoxic derivatives [26]; On the other hand, excess adiposity alters the renal parenchyma, leading to chronic hypoxia, with an increase in HIF-1 alpha transcriptional activity in this adipose tissue [27]. This amplifies the expression of oncogenes, which may ultimately lead to clear cell carcinoma development [28].

Our results indicate that obese patients presents less aggressive tumor characteristics, with lower pT size and lower nuclear ISUP grade. This is consistent with previous findings: Parker et al. study, based on 970 patients operated on for clear cell RCC, associated obesity with more favorable tumor characteristics: size less than 5 centimeters (40.8% in obese people versus 33.2% in case of normal BMI), more pTNM stage I (57.4% versus 46.1%) and more Fuhrman grades 1 and 2 (8.6% and 47.9% respectively versus 7.9% and 35.3%), as well as significantly lower pN and pM stages [16].

The present study did not show any effect on prognosis, however this may be related to limited follow-up. In Kim's meta-analysis, obesity was associated with better survival: Hazard Ratio (HR) of 0.68 (95% CI 0.59–0.78) for progression-free survival, HR of 0.85 (95% CI 0.79–0.93) for cancer specific survival and HR of 0.66 (95% CI 0.55–0.78) overall survival, considering patients with BMI greater than 25 versus patients with a normal BMI [18]. This article seems to confirm the obesity



**Fig. 2.** Proportions (%) of ISUP grades according to BMI. It represents the distribution of different histologies of renal cancer among obese population (indicated by orange bars) and non-obese population (indicated by blue bars).

**Table 3**

Distribution of pT sizes and nuclear ISUP grades according to BMI.

pT size	Population	Non-obese	Obese	P-value
pT1 + pT2	4054 (69.5%)	2951 (68.5%)	1103 (72.4%)	0.0063
pT3 + pT4	1743 (29.9%)	1329 (30.8%)	414 (27.1%)	
pTx	37 (0.6%)	30 (0.7%)	7 (0.5%)	
Nuclear grade				
ISUP 1	249 (4.3%)	156 (3.6%)	93 (6.2%)	< 0.0001
ISUP 2	2363 (41%)	1691 (39.7%)	672 (44.6%)	
ISUP 3	1941 (33.7%)	1459 (34.3%)	482 (32.0%)	
ISUP 4	812 (14.1%)	634 (14.9%)	178 (11.8%)	
Undefined ISUP	394 (6.8%)	315 (7.4%)	79 (5.2%)	

paradox, already validated for other cancers and which could be partly explained by the fact that obese patients are diagnosed and managed early due to their greater propensity to undergo health examinations including imaging. In the present analysis, the number of incidental diagnoses was significantly higher in obese patients, supporting this argument. However, the obesity paradox could also be partly biased due the comparison to sarcopenic patients with poor survival included in non-obese groups.

These results should be interpreted with keeping some limitations in mind:

- the retrospective nature of the study introduces specific biases, particularly due to missing information on lymph node and metastatic status. A large proportion of patients lacked regional and distant assessments (70.0% for pNx stage and 68.4% for pMx stage), and additional patients had no details on these statuses (1026 for lymph nodes and 1359 for metastasis). This missing data limits the reliability of our interpretations and could have reinforced our other prognostic findings;
- our study did not encounter enough recurrences to establish survival data, probably due to insufficient follow-up to reach a significant

number of events: among the 6749 patients analyzed, 74.5% were operated from 2015 onwards (5025 patients) and so have a follow-up of less than 5 years;

- the choice of BMI at the time of diagnosis to define obesity is detrimental for several reasons. Firstly, other markers could be best guarantors of excess body fat, as BMI encounter both muscle and fat: in our opinion, the visceral fat area (VFA) represents the ideal measure. It has been shown that adipocytes with peri-visceral distribution are metabolically more active than those under the skin, through the production of various cytokines (PAI1, TNF alpha, IL6, adipokines, VEGF) involved in angiogenesis and inflammation, but also with a lower rate of adiponectin, which is involved in insulin resistance [29]. This indicator was chosen by Wang and al whose results showed a greater effect of VFA than that attributed to BMI [9]. This indicator was not included in our data analysis because it is not calculated by radiologists in current practice, and because of the unavailability of CT scans. Secondly, the majority of BMIs were calculated on the basis of self-reported height and weight, which represents a reporting bias, especially as it has been shown that some overweight or obese patients tend to report a lower weight than actual weight [30]. Nevertheless, BMI remains the most accessible and the widely accepted measure of overweight and obesity among scientific societies.

It would therefore be interesting to repeat this type of analysis in the future, combining BMI with another anthropometric measurement more representative of excess body fat; in fact, the MAP score which specifically evaluates perinephric fat could be a relevant index. To the extent that our study was unable to deduce credible presumptions on survival, it would be necessary to re-study our cohort in a few years to be able to supplement our results with survival analyzes in order to be able to better study the influence of obesity on RCC prognosis.

## 5. Conclusion

In this study, we reported a significant positive association between obesity and clear cell renal cell carcinoma. We also found less aggressive tumor characteristics in patients with BMI > 30 kg/m<sup>2</sup>. The incidental detection and early treatment of renal tumor in obese patients may



explain the obesity paradox. Further research is needed to confirm the impact of BMI on RCC, particularly regarding its prognosis.

### Disclosure of interest

The authors declare that they have no competing interest.

### Appendix A. Supplementary data

Supplementary data associated with this article can be found, in the online version, at <https://doi.org/10.1016/j.fjurol.2025.102884>.

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