


Higher Body Mass Index is Associated with Improved Overall Survival in Kidney Cancer Patients: A Systematic Review and Meta-Analysis

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ABSTRACT

Introduction: Obesity is linked to better oncological outcomes for renal cell carcinoma (RCC), despite being a well-known risk factor for many diseases. This article assesses the relationship between kidney cancer outcomes (overall survival/OS) and body mass index (BMI). **Methods:** PubMed, Medline, Embase, CINAHL, and Central were used to retrieve studies. Research published up to December 2024 is included in this review. The review was carried out using the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) 2020. The quality of the study was assessed with the Newcastle-Ottawa Scale (NOS). RevMan version 5.4.1 was used to analyze and evaluate data. **Results:** Seven retrospective studies (HR 0.80, 95% CI 0.70–0.93, $p = 0.03$) showed that the group with normal BMI had a significantly lower overall survival rate than the group with obese or overweight BMI. Obesity may improve kidney cancer treatment outcomes, but the pathophysiology is not yet clear. Some studies suggest that overweight or obese individuals often have higher cancer biology, as they have lower levels of metabolic genes and fatty acids, which are important factors in tumor development. Healthy nutrition can also protect against treatment-related stress or make treatment work better. **Conclusion:** Obese or overweight individuals with kidney cancer had better results. Conversely, underweight group had more negative consequences.

Keywords: Body mass index, obesity, overall survival, renal cell carcinoma.

ABSTRAK

Pendahuluan: Obesitas dikaitkan dengan hasil onkologis yang lebih baik pada karsinoma sel ginjal (*renal cell carcinoma*/RCC), meskipun obesitas merupakan faktor risiko berbagai penyakit. Artikel ini menilai hubungan antara luaran kanker ginjal (kelangsungan hidup keseluruhan/OS) dan indeks massa tubuh (IMT). **Metode:** PubMed, Medline, Embase, CINAHL, dan Central digunakan untuk memilah studi. Penelitian yang diterbitkan hingga Desember 2024 dimasukkan dalam tinjauan ini. Tinjauan menggunakan Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) 2020. Kualitas penelitian dinilai dengan Newcastle-Ottawa Scale (NOS). RevMan versi 5.4.1 digunakan untuk analisis dan evaluasi data. **Hasil:** Tujuh studi retrospektif (HR 0,80, 95% CI 0,70–0,93, $p = 0,03$) menunjukkan bahwa kelompok IMT normal memiliki tingkat kelangsungan hidup keseluruhan yang secara signifikan lebih rendah dibandingkan dengan kelompok IMT obesitas atau kelebihan berat badan. Obesitas mungkin dapat meningkatkan hasil pengobatan kanker ginjal, tetapi patofisiologinya belum jelas. Beberapa studi menjelaskan bahwa individu dengan kelebihan berat badan atau obesitas sering memiliki biologi kanker yang lebih tinggi, karena memiliki tingkat gen metabolik dan asam lemak yang lebih rendah, yang merupakan faktor penting dalam perkembangan tumor. Nutrisi yang sehat juga dapat melindungi dari stres terkait pengobatan atau membuat pengobatan bekerja lebih baik. **Simpulan:** Individu dengan IMT di atas normal memiliki hasil pengobatan yang lebih baik untuk kanker ginjal. Sebaliknya, kelompok yang memiliki berat badan kurang memiliki konsekuensi yang lebih negatif. **Abdul Munawwir, Zalsabila Tiara Adhani, Aristo. Indeks Massa Tubuh yang Lebih Tinggi Berkaitan dengan Peningkatan Kelangsungan Hidup Secara Keseluruhan pada Pasien Kanker Ginjal: Tinjauan Sistematik dan Meta-analisis.**

Kata Kunci: Indeks massa tubuh, obesitas, kelangsungan hidup keseluruhan, karsinoma sel ginjal.

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INTRODUCTION

Obesity is a significant health concern in many affluent countries. It contributes to the development and progression of chronic kidney disease, metabolic disorders, and cardiovascular illnesses. Obesity also increases the risk of various cancers, including kidney cancer.[1–3] In surgical contexts, especially in urological oncology and general surgery, obesity is associated with a higher likelihood of perioperative complications.[1–3]

The "obesity paradox" refers to the substantial relationship that recent research has shown between obesity and better outcomes from kidney cancer. According to a few studies, individuals with higher body mass indices had better pathological outcomes than those with normal body mass index (BMI), which may account for their improved prognosis.[4–6] More recent research has shown a strong correlation between cancer outcomes and the adiposity index, which measures visceral and subcutaneous abdominal adiposity tissue.[7,8]

BMI below 18.5 kg/m² is considered underweight; between 18.5 and 24.99 kg/m² is normal weight, between 25 and 29.99 kg/m² is overweight, and above 30 kg/m² is obese.[9] The BMI-based definition of obesity may need to be clarified due to the many possible confounders. However, BMI has a wider clinical use. This study aims to examine the relationship between BMI and overall survival in kidney cancer patients.

METHODS

The research sources are PubMed, Medline, Embase, CINAHL, and Central. For this review, studies published up to December 2024 were considered. Randomized controlled trials, case-control studies, and cohort studies were all accepted. The eligibility criteria are: original data from case-control or prospective cohort studies, publications after December 2024, and assessment of the correlation between overall survival (OS), exposure factors, and BMI. Articles in languages other than English are not included in this study.

Data collected from each study include: name of the first author, study period, study design, type of control group, location, age, gender, body mass index (BMI) (measured or self-reported), nephrectomy status (yes or no), disease classification (local or metastatic), follow-up duration, hazard ratio or relative risk estimate, and 95% confidence interval.

Newcastle-Ottawa Scale's selection, similarity, and outcome components were used to evaluate the quality of the studies included in the systematic review.[10] The following variables' information was collected from each study: number of cases and deaths per BMI group; multivariate hazard/risk ratios and confidence intervals for oncological outcomes per BMI group; mean age, gender, follow-up duration, author name, decade of publication, and confounding variables controlled in the multivariate approach. Three authors, AM, ZTA, and A, independently carried out a quality assessment. This meta-analysis used Review Manager 5.4.1. Standard chi-square test and a visual inspection of forest plots were employed to detect heterogeneity. Due to the test's low power, the I^2 statistic was also employed. Evaluating research anomalies is a well-known method for determining how heterogeneity affects meta-analysis. Both fixed- and random-effects models were applied based on the I^2 heterogeneity index. Fixed effect models were used to analyze I^2 values of 50% or less, and random effects models were used to analyze I^2 values greater than 50%. A statistically significant two-tailed *p*-value was defined as being less than 0.05.

RESULTS

The initial search included 450 articles: 235 articles from PubMed, 79 articles from Medline, 46 articles from Embase, 50 articles from CINAHL, and 40 articles from Central. After reviewing the abstracts and titles, 410 articles were deemed duplicates, and 30 others were excluded based on inclusion and exclusion criteria **(Figure 1).** Of the remaining ten articles, only seven met the criteria for comprehensive analysis after thorough evaluation **(Table).** Compared to the group with normal BMI, OS was higher in overweight or obese individuals. Seven retrospective studies (HR 0.80, 95% CI 0.70–0.93, $p = 0.03$) showed that the group with normal BMI had a significantly lower overall survival rate than the group with obese or overweight BMI **(Figure 2)**; none of the studies showed a higher overall survival rate in the normal BMI group as compared with the obese or overweight BMI group.

DISCUSSION

This study found that the percentage of obese people ranged from 12.6% to 44%, with an overall downward trend in obesity rates, particularly in Asian countries. Furthermore, obesity has been linked to an increased risk of prostate and bladder cancer.[11,12] However, there is a negative correlation between obesity and heart failure.[13,14] It is estimated that individuals with a higher BMI have more muscle and fat, which indicates better nutritional health and a higher chance of survival. This has been demonstrated by waist-to-hip ratio and other measures of adiposity.[15] Similar trends have been observed in other chronic diseases, such as chronic kidney disease and chronic obstructive pulmonary disease. This 'fat paradox' has also been observed in patients with various types of cancer, including breast, thyroid, colon, lymphoma, leukemia and metastatic lung cancers.[16]

Obesity may improve kidney cancer treatment outcomes, but the mechanism is not yet clear. Being overweight or obese makes small kidney tumors more likely to be found early, as they may be checked more often for other health problems.[17] Several studies indicate that overweight and obese individuals may exhibit a higher risk of cancer. This is partly attributed to lower levels of certain metabolic genes and fatty acids, both of which play crucial roles in tumor development and progression.[17]

Another theory suggests that too much perirenal fat could stop cancer from growing.[17] However, this idea has been rejected recently.[17] A sufficient nutritional status may protect against treatment-induced stress or enhance the therapeutic response.[17] Adipose tissues are essential for immune regulation at the molecular biology level, as they produce various inflammatory substances and signals, such as leptin, adiponectin, and cytokines. The downregulation of adiponectin signaling or fatty acid synthase (FASN) and the overexpression of leptin and insulin-like growth factor are significantly correlated with obesity and may serve as advantageous prognostic markers.[17] FASN appears to promote cancer by enabling tumor cells to

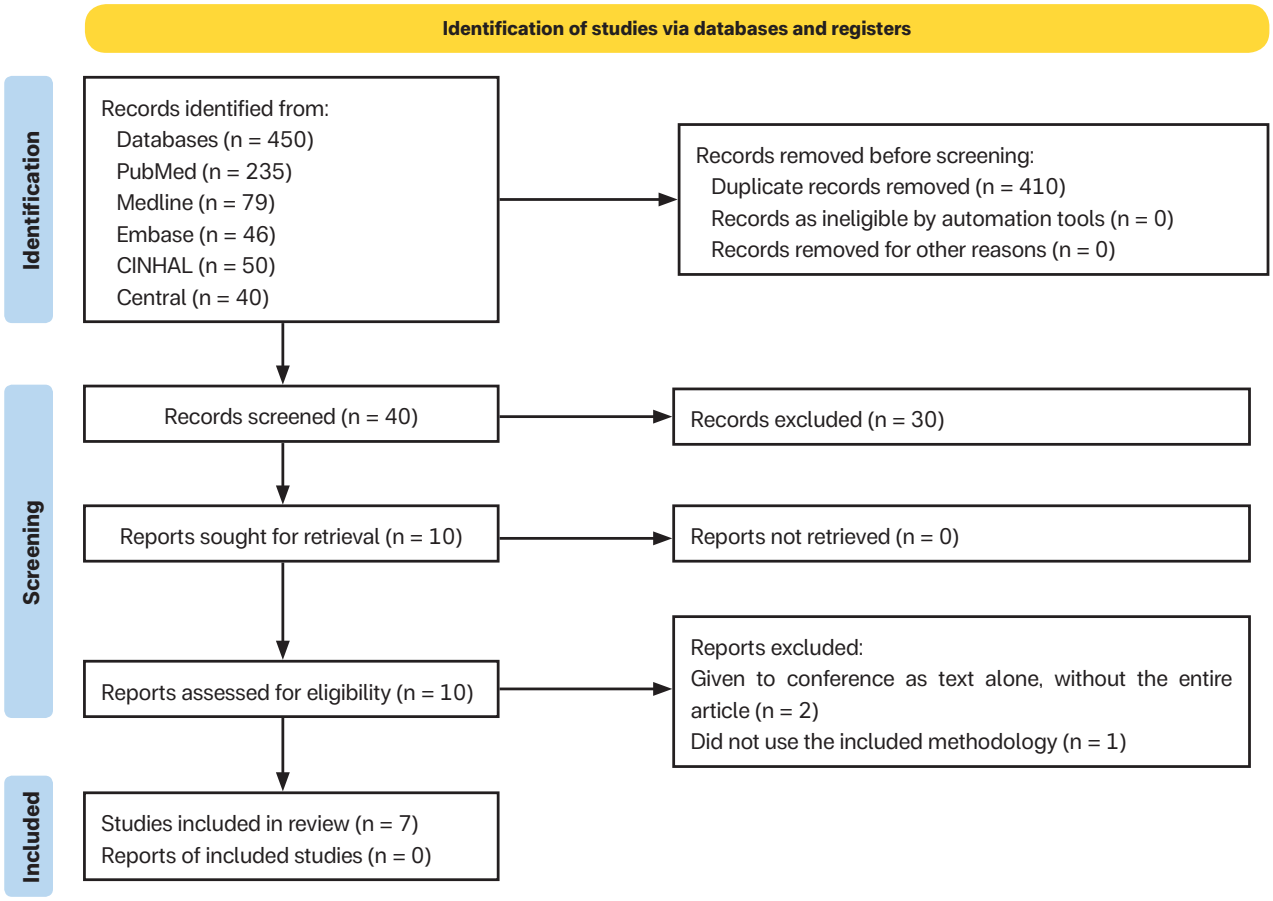

Figure 1. PRISMA 2020 flow diagram for systematic review.

Table. The Newcastle-Ottawa Scale for all studies in quantitative synthesis.

Domain	Item	Ahmedov, 2018.[5]	Byun, 2018.[20]	Eskelinen, 2017.[32]	Hakimi, 2013.[22]	Ito, 2017.[33]	Lee, 2015.[30]	Park, 2013.[34]
Selection (maximum 4 stars)	Representativeness of the exposed	-	*	*	-	*	*	*
	Selection of the non-exposed cohort	*	*	*	-	*	*	-
	Ascertainment of exposure	*	*	*	*	-	*	*
	Outcome of interest was not present at the start of the study	*	*	*	*	-	*	*
Comparability (maximum of 2 stars)	Comparability of cohorts on the basis	*	*	*	*	*	-	-
	Design analysis	*	*	*	*	*	*	*
Outcomes (maximum of 3 stars)	Assessment of outcome	*	*	*	*	*	*	*
	Follow-up long enough for outcomes	*	-	-	-	*	*	*
	Adequacy of follow-up	*	*	*	*	*	-	*
Score		8 (Good)	8 (Good)	8 (Good)	6 (Fair)	6 (Fair)	7 (Good)	7 (Good)

Study or Subgroup	Year	log[Hazard Ratio]	SE	Weight %	Hazard Ratio IV, Random, 95% CI
Ahmedov	2018	-0.1912	0.0708	34.8	0.83 [0.72, 0.95]
Byun	2018	-0.4323	0.1603	14.9	0.65 [0.47, 0.89]
Eskelimen	2017	0.0198	0.1435	17.3	1.02 [0.77, 1.35]
Hakimi	2013	-0.3079	0.1315	19.4	0.73 [0.57, 0.95]
Ito	2017	-1.8773	1.0381	0.5	0.15 [0.02, 1.17]
Lee H	2015	-0.2797	0.213	9.6	0.76 [0.50, 1.15]
Park	2013	0.1319	0.3855	3.4	1.14 [0.54, 2.43]
Total (95% CI)				**100.0**	**0.80 [0.70, 0.93]**

Heterogeneity. Tau² = 0.01; Chi²= 8.59, df = 6 (*p* = 0.20); I² = 30%

Test for overall effect: Z= 2.99 (*p* = 0.003)

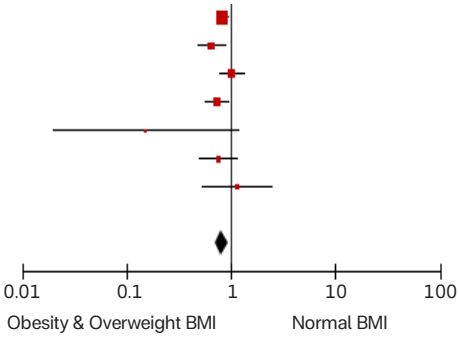

Figure 2. Forest plot of meta-analysis of overall survival based on BMI comparing overweight & obese group vs normal BMI control group.

produce lipids.[18] Leptin is known for its anti-cancer effects because it makes natural killer cells grow and work better. It also plays a big role in "crosstalk" with humoral and cell-mediated immunity by making pro-inflammatory T helper 1 immune responses stronger, which may help slow the growth of kidney cancer.[17,18] C-reactive protein acts as a possible prognostic marker in patients with kidney cancer.[17,18]

This correlation between kidney cancer and BMI is unique to histologic subtypes, as kidney cancer is a complex and multifaceted disease.[19] Higher BMI is associated with a better prognosis for patients with clear cell renal cancer.[20] It is associated with poor outcomes in the chromophobe variant, but not in the papillary variant.[21] VHL gene mutations, which are found in 75% of clear cell subtype patients, may be associated with downregulation of FASN and inability to control IGF.[21,22] When OS was the primary objective in comparing different BMI groups, there was the strongest predictive relationship between higher obesity and kidney cancer survival. This was also true when comparing the obese group by itself with the normal BMI group. Studies have found that nutritional deficiencies lead to higher postoperative mortality and recurrence. Obesity can serve as a direct indicator of overall health and physical reserve capacity. BMI has a significant influence on overall survival (OS), even after underweight individuals are excluded from the normal BMI group.[18,23]

Obesity is a known risk factor for metabolic disease, chronic heart disease, and other perioperative problems. Studies have shown that the negative effects on underweight patients are similar.[2] In addition, a number of studies have shown that lower BMI is associated with higher risk of cancer.[24] This is usually thought to be the result of cachexia, or unintentional weight loss caused by sarcopenia and shrinkage of adiposity in patients with advanced cancer.[24] Brookman-May et al. showed that unplanned weight loss of more than 10 kg had a significant effect on survival, whereas BMI did not. This suggests that preoperative weight loss screening is important.[25] The causal relationship between cachexia and kidney cancer survival, however, remains unclear. Instead, an inverse causal relationship, meaning that underweight people are more likely to have worse outcomes because they progress faster-may be the case. Since patients with localized kidney cancer are thought to have a much lower prevalence of cachexia and a similar association between BMI and outcome across all domains, cachexia does not seem to be the main cause of their poor outcome.[25] This may imply that tumor-induced cachexia does not necessarily lead to decreased life span for individuals with lower BMI. The aforementioned results are still relevant even after testing a lower weight group of individuals diagnosed with localized kidney cancer. Due to its favorable prognosis, it is not clinically significant, but further research is clearly needed.[17,26,27]

Studies have shown that distinguishing between underweight individuals before the onset of cachexia is difficult, as it can be indistinguishable from individuals who developed cachexia due to more severe kidney cancer. Cachexia and underweight BMI tend to interact, so they are not good predictors of final outcome.[27,28] But there are some limitations that should be noted. For predicting kidney cancer prognosis, BMI is severely limited by many factors. These include the retrospective design of most studies, selection bias, collider stratification bias, heterogeneity of the study population across series, BMI classification, inconsistent use of covariate analysis, and little long-term follow-up, including BMI trajectory over the study period. Sarcopenia, weight changes after kidney cancer diagnosis and treatment, and associated lifestyle changes such as smoking may undermine validity. As a number of trials did not use BMI calculation methods, the accuracy of self-reported height, weight, and time to diagnosis is also doubtful. A study showed that alternative adiposity indices, such as sarcopenia, waist-to-hip ratio, or visceral or subcutaneous fat area, are more reliable for indicating oncologic outcomes than BMI.[5,7,27]

Women and men have different adiposity distributions. Men have more visceral fat than women. The association between obesity and cancer outcomes that is more pronounced in men than women may be explained by the fact that a higher visceral fat content seems to be associated with a better prognosis.[29]

A population-based prospective study, however, refuted this result and found no difference.[29–31] To find the most accurate prognostic indicators for men and women, more thorough prospective studies are needed. It is not always evident that weight loss to achieve a normal BMI results in better outcomes. However, given the significant limitations and biases of these studies, as well as other adverse health effects, it would be prudent to advise people against becoming overweight or obese as a way to reduce cancer risk. To overcome the biases and limitations mentioned above, basic scientific research and prospective clinical trials are essential. This will allow us to draw additional conclusions about the effect of BMI on kidney cancer and assist us in finding the right BMI that negatively impacts tumor development.

CONCLUSION

According to this study, individuals with BMIs over the normal range had better results for kidney cancer. Conversely, the group that was underweight had more negative consequences. The relationship between BMI and kidney cancer outcomes is intricate. Although they are currently not widely used in many locations, other methods of assessing adiposity may be more representative than BMI. This finding needs to be clarified by multicenter, large-scale, high-quality prospective studies and additional research on basic biological mechanisms.

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After obtaining the relevant sources, AM and A were assigned responsibility for methodically investigating and evaluating the literature individually. AM wrote the research article, including the abstract, introduction, research methods, and citations. ZTA and A were responsible for organizing the study results, discussion, and conclusion as well as for extracting pertinent data.

REFERENCES

1. Chow WH, Dong LM, Devesa SS. Epidemiology and risk factors for kidney cancer. Nat Rev Urol. 2010;7(5):245–57. doi: 10.1038/nrurol.2010.46.
2. Tjeertes EEKM, Hoeks SSE, Beks SSBJC, Valentijn TTM, Hoofwijk AAGM, Stolker RJRJ. Obesity – a risk factor for postoperative complications in general surgery? BMC Anesthesiol. 2015;15(1):112. doi: 10.1186/s12871-015-0096-7.
3. Albiges L, Hakimi AA, Xie W, McKay RR, Simantov R, Lin X, et al. Body mass index and metastatic renal cell carcinoma: clinical and biological correlations. J Clin Oncol. 2016;34(30):3655–63. doi: 10.1200/JCO.2016.66.7311.
4. Abufaraj M, Iwata T, Kimura S, Haddad A, Al-Ani H, Abusubaih L, et al. Differential impact on gonadotropin-releasing hormone antagonist versus agonist on clinical safety and oncologic outcomes on patients with metastatic prostate cancer: a meta-analysis of randomized controlled trials. Eur Urol. 2021;79(1):44–53. doi: 10.1016/j.eururo.2020.06.002.
5. Ahmedov V, Kizilay F, Cureklibatir I. Prognostic significance of body mass index and other tumor and patient characteristics in non-metastatic renal cell carcinoma. Urol J. 2018;15(3):96–103. https://doi.org/10.22037/uj.v15i3.4067.
6. Jeon HG, Jeong IG, Lee JH, Lee CJ, Kwak C, Kim HH, et al. Prognostic value of body mass index in Korean patients with renal cell carcinoma. J Urol. 2010;183(2):448–54. doi: 10.1016/j.juro.2009.10.004.
7. Ebadi M, Martin L, Ghosh S, Field CJ, Lehner R, Baracos VE, et al. Subcutaneous adiposity is an independent predictor of mortality in cancer patients. Br J Cancer. 2017;117(1):148–55. doi: 10.1038/bjc.2017.149.
8. Cho HJ, Kim SJ, Ha US, Hong SH, Kim JC, Choi YJ, et al. Prognostic value of capsular invasion for localized clear-cell renal cell carcinoma. Eur Urol. 2009;56(6):1006–12. doi: 10.1016/j.eururo.2008.11.031.
9. World Health Organization. What is overwight and obesity [Internet]. Geneva: World Health Organization; 2019. Available from: https://www.who.int/dietphysicalactivity/childhood_what/en/.
10. Norris JM, Simpson BS, Ball R, Freeman A, Kirkham A, Parry MA, et al. A modified Newcastle-Ottawa Scale for assessment of study quality in genetic urological research. Eur Urol. 2021;79(3):325–6. doi: 10.1016/j.eururo.2020.12.017.
11. McGuire BB, Fitzpatrick JM. BMI and the risk of renal cell carcinoma. Curr Opin Urol. 2011;21(5):356–61. doi: 10.1097/MOU.0b013e32834962d5.
12. Vucenik I, Stains JP. Obesity and cancer risk: evidence, mechanisms, and recommendations. Ann N Y Acad Sci. 2012;1271(1):37–43. doi: 10.1111/j.1749-6632.2012.06750.x.
13. Gild P, Ehdaie B, Kluth LA. Effect of obesity on bladder cancer and renal cell carcinoma incidence and survival. Curr Opin Urol. 2017;27(5):409–14. doi: 10.1097/MOU.0000000000000425.
14. Goodwin PJ, Stambolic V. Impact of the obesity epidemic on cancer. Annu Rev Med. 2015;66:281–96. doi: 10.1146/annurev-med-051613-012328.
15. Tutor AW, Lavie CJ, Kachur S, Milani RV, Ventura HO. Updates on obesity and the obesity paradox in cardiovascular diseases. Prog Cardiovasc Dis. 2023;78:2–10. doi: 10.1016/j.pcad.2022.11.013.

16. Kalantar-Zadeh K, Rhee CM, Chou J, Ahmadi SF, Park J, Chen JLT, et al. The obesity paradox in kidney disease: how to reconcile it with obesity management. Kidney Int Rep. 2017;2(2):271–81. doi: 10.1016/j.ekir.2017.01.009.

17. Komura K, Inamoto T, Black PC, Koyama K, Katsuoka Y, Watsuji T, et al. Prognostic significance of body mass index in Asian patients with localized renal cell carcinoma. Nutr Cancer. 2011;63(6):908–15. doi: 10.1080/01635581.2011.594207.

18. Kim LH, Doan P, He Y, Lau HM, Pleass H, Patel MI. A systematic review and meta-analysis of the significance of body mass index on kidney cancer outcomes. J Urol. 2021;205(2):346–55. doi: 10.1097/JU.0000000000001377.

19. Lee WK, Hong SK, Lee S, Kwak C, Oh JJ, Jeong CW, et al. Prognostic value of body mass index according to histologic subtype in nonmetastatic renal cell carcinoma: a large cohort analysis. Clin Genitourin Cancer. 2015;13(5):461–8. doi: 10.1016/j.clgc.2015.04.012.

20. Byun SS, Hwang EC, Kang SH, Hong SH, Chung J, Kwon TG, et al. Sex-specific prognostic significance of obesity in nonmetastatic clear-cell renal-cell carcinoma in Korea: a large multicenter cohort analysis. Clin Genitourin Cancer. 2018;16(1):e173–9. doi: 10.1016/j.clgc.2017.08.015.

21. Ohno Y, Nakashima J, Nakagami Y, Satake N, Gondo T, Ohori M, et al. Sex and the clinical value of body mass index in patients with clear cell renal cell carcinoma. Br J Cancer. 2013;109(7):1899–903. doi: 10.1038/bjc.2013.512.

22. Hakimi AA, Furberg H, Zabor EC, Jacobsen A, Schultz N, Ciriello G, et al. An epidemiologic and genomic investigation into the obesity paradox in renal cell carcinoma. JNCI J Natl Cancer Inst. 2013;105(24):1862–70. doi: 10.1093/jnci/djt310.

23. Donin NM, Pantuck A, Klopfer P, Bevan P, Fall B, Said J, et al. Body mass index and survival in a prospective randomized trial of localized high-risk renal cell carcinoma. Cancer Epidemiol Biomarkers Prev. 2016;25(9):1326–32. doi: 10.1158/1055-9965.EPI-16-0226.

24. Sung HH, Jeon SS, Park SY, Jeong BC, Seo SI, Lee HM, et al. Impact of body mass index in Korean patients with renal cell carcinoma. Cancer Causes Control. 2012;23(3):505–11. doi: 10.1007/s10552-012-9910-4.

25. Brookman-May S, Kendel F, Hoschke B, Wieland WF, Burger M, Rossler W, et al. Impact of body mass index and weight loss on cancer-specific and overall survival in patients with surgically resected renal cell carcinoma. Scand J Urol Nephrol. 2011;45(1):5–14. doi: 10.3109/00365599.2010.515610.

26. Waalkes S, Merseburger AS, Kramer MW, Herrmann TRW, Wegener G, Rustemeier J, et al. Obesity is associated with improved survival in patients with organ-confined clear-cell kidney cancer. Cancer Causes Control. 2010;21(11):1905–10. doi: 10.1007/s10552-010-9618-2.

27. Sunela KL, Kataja MJ, Kellokumpu-Lehtinen PLI. Influence of body mass index and smoking on the long-term survival of patients with renal cell cancer. Clin Genitourin Cancer. 2013;11(4):458–64. doi: 10.1016/j.clgc.2013.04.017.

28. Song M, Giovannucci E. Preventable incidence and mortality of carcinoma associated with lifestyle factors among while adults in the United States. JAMA Oncol. 2016;2(9):1154. doi: 10.1001/jamaoncol.2016.0843.

29. Reeves GK, Pirie K, Beral V, Green J, Spencer E, Bull D. Cancer incidence and mortality in relation to body mass index in the million women study: cohort study. BMJ. 2007;335(7630):1134. doi: 10.1136/bmj.39367.495995.AE.

30. Lee HW, Jeong BC, Seo SI, Jeon SS, Lee HM, Choi HY, et al. Prognostic significance of visceral obesity in patients with advanced renal cell carcinoma undergoing nephrectomy. Int J Urol. 2015;22(5):455–61. doi: 10.1111/iju.12716.

31. Bahadoram S, Davoodi M, Hassanzadeh S, Bahadoram M, Barahman M, Mafakher L. Renal cell carcinoma: an overview of the epidemiology, diagnosis, and treatment. G Ital Nefrol Organo Uff Della Soc Ital Nefrol. 2022;39(3):2022-vol3. PMID: 35819037.