Urological manifestations in obesity

Badhrinath Jayapal[a] *and Nitin Kapoor*[b,c]

Purpose of review
The global prevalence of obesity continues to rise, with effects ranging across urology. This review summarizes recent evidence on obesity and its impact on various urological conditions, including malignancy, infections, stone disease, functional urology and male reproductive health, chronic kidney disease, and pediatric urology, to guide comprehensive management in this population.

Recent findings
Obesity has a paradoxical survival benefit in patients with renal cell carcinoma, while its impact drives aggressive prostate cancer. Obesity increases kidney stone risk, with sodium-glucose co-transporter 2 inhibitors and glucagon-like peptide-1 (GLP-1) receptor agonists showing divergent effects on lithogenesis and urinary tract infections. It is a modifiable risk factor for overactive bladder and stress urinary incontinence, both of which improve with weight loss/ bariatric surgery. Robotic surgery mitigates obesity-related perioperative risk. Hypogonadism secondary to obesity is a major reversible cause of male infertility. Glucagon-like peptide-1 receptor agonists (GLP-1 RA) reverse metabolic hypogonadism and helps in slowing chronic kidney progression. Maternal and childhood obesity may impair testicular descent and can also increase the risk of nocturnal enuresis.

Summary
Obesity's urological effects are heterogeneous, paradoxical from pediatric to adults, mediated by insulin resistance, chronic inflammation, and hormonal dysregulation. Weight loss and the use of GLP-1 RA may offer a unique strategy for optimizing and treating urological conditions.

Keywords
functional urology, glucagon-like peptide-1 receptor agonists, male infertility, obesity, urology

INTRODUCTION

Obesity is a major healthcare burden worldwide. Approximately 43–44% of the adults were overweight, and 16% were obese. These numbers have been significantly increasing since the last decade. Similarly, the prevalence of obesity has increased from 8% to 20% over the past two decades among children and adolescents [1]. The Obesity rates have increased significantly in low- and middle-income countries, which were previously associated only with high-income countries. From an economic perspective, overweight and obesity are projected to cost US$ 3 trillion per year by 2030 and US$ 18 trillion by 2060 [2]. Obesity has a significant impact on various urological conditions, including urological malignancies, infections, kidney stone disease (KSD), infertility, and functional urology. This review examined recent evidence on various urological conditions and their outcomes in relation to obesity.

OBESITY AND UROLOGICAL MALIGNANCIES

Obesity has a certain influence on the treatment outcomes of urological malignancies. It differs across the malignancies and with their staging. BMI may function as an independent prognostic factor in renal cell carcinoma (RCC), with higher BMI associated with increased risk, and patients with higher BMI often demonstrate improved overall and progression-free survival. In bladder cancer, it's poorly defined with a modestly increased risk with higher BMI. Obesity is associated with more aggressive prostate cancer, with higher cancer-specific mortality [3▪▪].

OBESITY AND PROSTATE CANCER

Higher BMI patients had reduced concordance rates across staging, Gleason scoring and grading from

[a]Department of Urology, Christian Medical College, Vellore, Ranipet Campus, [b]Department of Endocrinology, Diabetes and Metabolism, Christian Medical College, Vellore, Tamil Nadu, India and [c]Non-Communicable Disease unit, Baker Heart and Diabetes Institute, Melbourne, Victoria, Australia

Correspondence to Assistant Professor Badhrinath Jayapal, Department of Urology, Christian Medical College, Vellore, Ranipet Campus, Tamil Nadu, 632517, India. Tel: +91 9944187879; e-mail: badhrinj@gmail.com

Curr Opin Endocrinol Diabetes Obes 2026, 33:288–297
DOI:10.1097/MED.0000000000000986

KEY POINTS

- Obesity has a paradoxical effect on uro-oncology, with improved survival in renal cell carcinoma and immunotherapy, and more aggressive, increased mortality in Prostate cancer.
- Obesity has a significant effect on benign urological conditions such as stone disease, male infertility, incontinence, and functional urology by promoting insulin resistance, chronic inflammation, and hormonal dysregulation.
- Emerging pediatric urology evidence links maternal/childhood obesity to obesity and impaired testicular function, antibiotic-prophylaxis-associated weight gain in vesicoureteric reflux, and higher rates of nocturnal enuresis and overactive bladder.
- Glucagon-like peptide-1 receptor agonists are emerging as a unifying therapy across urology in terms of reduced cancer risk, fewer kidney stones, slower chronic kidney disease progression, and reversal of metabolic hypogonadism.
- Robotic surgery reduces obesity-related perioperative risk across the procedures, with weight loss and metabolic optimization in managing obesity-related urological conditions.

preop assessment who underwent radical prostatectomy. Active surveillance in these patients must be carefully monitored [4]. Despite a higher incidence of pT3a disease at surgery, there is no significant difference in biochemical recurrence or positive surgical margins. Class III morbidly obese patients showed no differences in functional or oncological outcomes compared with normal-weight patients. Class III obesity patients who underwent robotic-assisted radical prostatectomy (RARP) had longer console times, higher rates of estimated blood loss and higher rates of lymphocele formation. Full nerve sparing was less frequent [5]. In patients who underwent bilateral nerve-sparing procedure, BMI did not significantly affect the outcomes, pathology or blood loss [6]. Obesity adversely impacts perioperative outcomes, short-term incontinence recovery rates, and mid-term emergency department (ED) recovery rates, but did not affect mid-term oncological outcomes after RARP [7]. The early and 1-year continent rates were comparable in patients who underwent retzius-sparing radical prostatectomy [8]. Higher BMI was associated with prolonged console and instrument active times during RARP, indicating a procedure-specific effect of obesity on efficiency that was not observed in RAPN [9]. In both metastatic hormone-sensitive and castration-resistant prostate cancer, the risk of death was lower in patients with higher BMI [10,11]. A recent study showed potential cancer-risk reduction with semaglutide [glucagon-like peptide-1 receptor agonist (GLP-1 RA] in obese patients, including Prostate cancer, potentiating future studies [12]. Use of GLP-1 RA did not affect perioperative safety in terms of morbidity and complications [13].

OBESITY AND RENAL CANCER

Almost 20% of kidney cancer is linked to obesity, and its risk is expected to increase beyond 2030. Significant male burden in the 55–79 age group noted in high-SDI (Socio-Development Index) countries, with recent trends suggesting increased rates in low-SDI countries [14▪▪]. The obesity paradox was termed when patients with higher BMI have a higher risk of developing renal cell carcinoma (RCC) but with better outcomes, particularly in males with high-risk localized disease [15,16]. A recent study showed a U-shaped association between relative visceral fat area (rVFA) and recurrence-free survival (RFS) in localized ccRCC, with a significant effect at high rVFA and a nonsignificant trend at low rVFA [17]. Recently, in addition to the existing Leibovich prognostic score for localized RCC patients, body composition parameters (BCP), such as visceral adipose tissue density and intramuscular adipose tissue density, independently predicted reduced recurrence-free survival (RFS) [18▪▪]. Even in metastatic RCC patients receiving tyrosine kinase inhibitors (TKI) or immune checkpoint inhibitors (IO/ICI) combination therapy, outcomes are favorable as first-line therapy in higher BMI [19]. In combination therapies, IO showed longer overall survival (OS) than IO-TKI regimens [20]. The BMI polygenic score study found no effect on mortality, concluding that the observational obesity paradox is likely reverse causation of preclinical cancer, causing weight loss rather than a real biological benefit of fat [21].

Among obese patients who underwent robot-assisted, laparoscopic and open partial nephrectomies, Robotic partial nephrectomy was associated with the lowest adjusted odds of complications, mortality and shorter hospitalization [22]. Obesity is also associated with major complications (Clavien-Dindo III–IV), along with coronary disease, COPD and Chronic kidney disease [23]. Obesity also attenuates the benefits of the prone position during robotic partial nephrectomy compared with the lateral position [24]. Obesity and diabetes contributed to worse renal functional recovery at 12 months after robotic partial nephrectomy [25]. In patients undergoing radical nephrectomy and inferior vena cava thrombectomy, obesity is associated with a three-fold increased risk of moderate to severe acute kidney injury (AKI) [26].

1752-296X Copyright © 2026 Wolters Kluwer Health, LLC. All rights reserved.

OBESITY AND BLADDER CANCER

In overweight/ obese patients with bladder cancer who are ≥65 years of age, there is a significant difference in better overall survival compared to normal-weight or underweight patients. Severely obese patients had the highest activities of daily living (ADL) disability rates with significantly worse physical/ mental HRQOL, showing dissociation between living longer and living well in this population [27]. Patients who underwent radical cystectomy had significant perioperative complications of Clavien-Dindo ≥2, such as paralytic ileus, wound infections, Acute kidney injury, and longer hospital stay [28]. The overweight and obese bladder cancer patients who underwent Radical Cystectomy had better outcomes within the first 5 years but showed no difference in long-term survival – debunking the bladder "Obesity Paradox" [29]. Patients with a BMI ≥34 kg/m^2 are at high risk of complications, which helps us strategize prehabilitation for this group [30]. In patients undergoing robotic-assisted radical cystectomy, the incidence of urinary tract infection (UTI) risk increases up to 39% with increasing age, female sex and obesity, in whom short-course regimens can be effective, considering the rising antibiotic resistance [31].

OBESITY AND UROTHELIAL CARCINOMA

Obesity, along with dyslipidemia, did not significantly affect overall survival compared to poorer outcomes with Hypertension and Diabetes [32▪▪]. In advanced urothelial carcinoma, patients on combination therapy with enfortumab vedotin plus pembrolizumab (EV+P) who experienced early weight loss of ≥5% by day 28 had inferior overall survival, regardless of preoperative BMI or albumin levels. Its association is similar to cancer-associated cachexia in patients with gastric cancer who had worse clinical outcomes with early on-treatment weight loss. Prospective studies evaluating early nutritional interventions to mitigate this risk should be conducted [33].

OBESITY AND PENILE CANCER

Obesity in penile cancer is not extensively studied as some other urological cancers. In obese patients, groin examination for inguinal nodes is challenging, where the imaging modalities such as USG, CT or MRI are used. These modalities certainly cannot detect micrometastasis, whereas FDG PET cannot reliably detect nodes of <10 mm. In patients undergoing surgery for penile cancer, the higher the BMI, the greater the risk of early postoperative complications within the first 30 days, such as wound infection (26%) and bleeding (1.5%) in partial penectomies and any type of inguinal procedures [34].

OBESITY AND TESTICULAR CANCER

Obesity is not a strong driver of testicular malignancies compared to other urological malignancies such as kidney, prostate, or bladder [35]. There was a nonlinearly increased risk in young patients (<30 years of age) with a BMI <30, for which further studies are warranted [36].

OBESITY AND INFECTIONS

Among urological disorders, it's known that higher BMI is associated with a higher risk of Fournier's gangrene, whereas low BMI is a risk factor for genitourinary tuberculosis. Among newer evidence, sodium-glucose co-transporter 2 (SGLT2) Inhibitors co-prescribed in patients with diabetes mellitus carry an FDA warning for urinary tract infection. Still, the association is with fungal rather than bacterial infection [37]. Abnormal genitourinary pathology with SGLT2i is an additive risk for fungal UTI but not for nonfungal UTI [38]. In the setting of recurrent and complicated urinary tract infection with patients requiring high-dose use, its use warrants considerable caution, even though the evidence suggests any excess risk is modest [39] (Figs. 1–3).

OBESITY AND KIDNEY STONE DISEASE (UROLITHIASIS)

Metabolic syndrome was more common in stone formers than in nonstone formers. Hypertension and impaired glucose tolerance showed the strongest associations with KSD, but guidelines do not mandate routine metabolic screening [40]. Males with BMI >30 kg/m^2 have a higher risk of developing stones compared to the normal population, especially aged between 40 and 49 years. The risk increases with a history of chronic disease such as hypertension, diabetes, malignancy and weak/failing kidneys [41]. A 30-year systematic review in the UK has found that high BMI, overweight, and obesity have an increased association with stone risk. Still, due to high heterogeneity and imbalanced cohorts, the evidence was rated very low [42]. Novel anthropometric visceral adiposity-focused indices, such as the A body shape index (ABSI) and body roundness index (BRI), show a stronger positive association with KSD than traditional measures such as BMI and waist circumference [43]. Obesity, metabolic syndrome, and diabetes mellitus are linked to KSD through insulin resistance and low-grade systemic inflammation. These impair renal ammoniagenesis and lower urinary pH, thereby driving uric acid crystallization and promoting hypercalciuria, hyperoxaluria, and hypocitraturia, thereby favoring calcium oxalate stones [44]. In view of this, KSD should

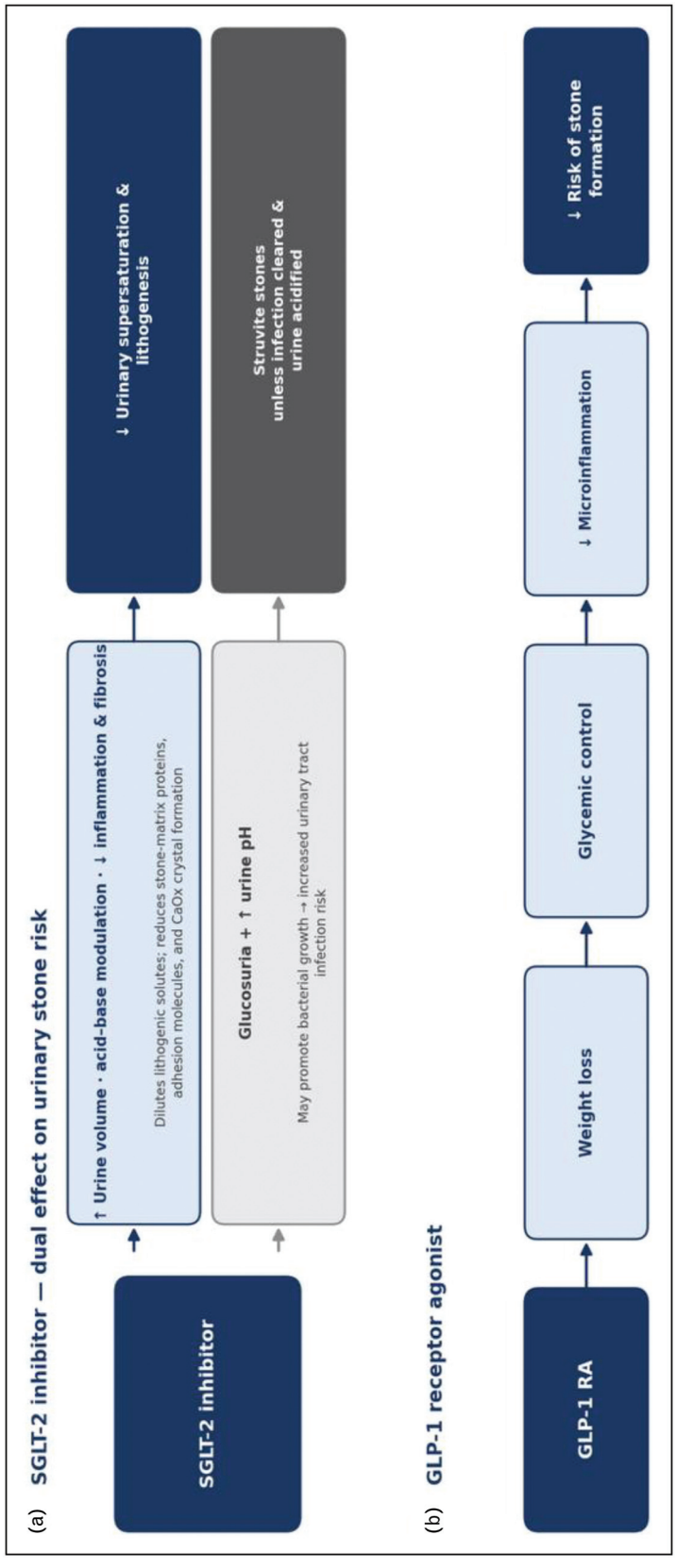

FIGURE 1. (a) SGLT-2 inhibitors reduce urinary supersaturation and lithogenesis; conversely, glucosuria and elevated urine pH precipitate struvite stones unless treated. (b) GLP-1 receptor agonists reduce the risk of stone formation [44].

1752-296X Copyright © 2026 Wolters Kluwer Health, LLC. All rights reserved.

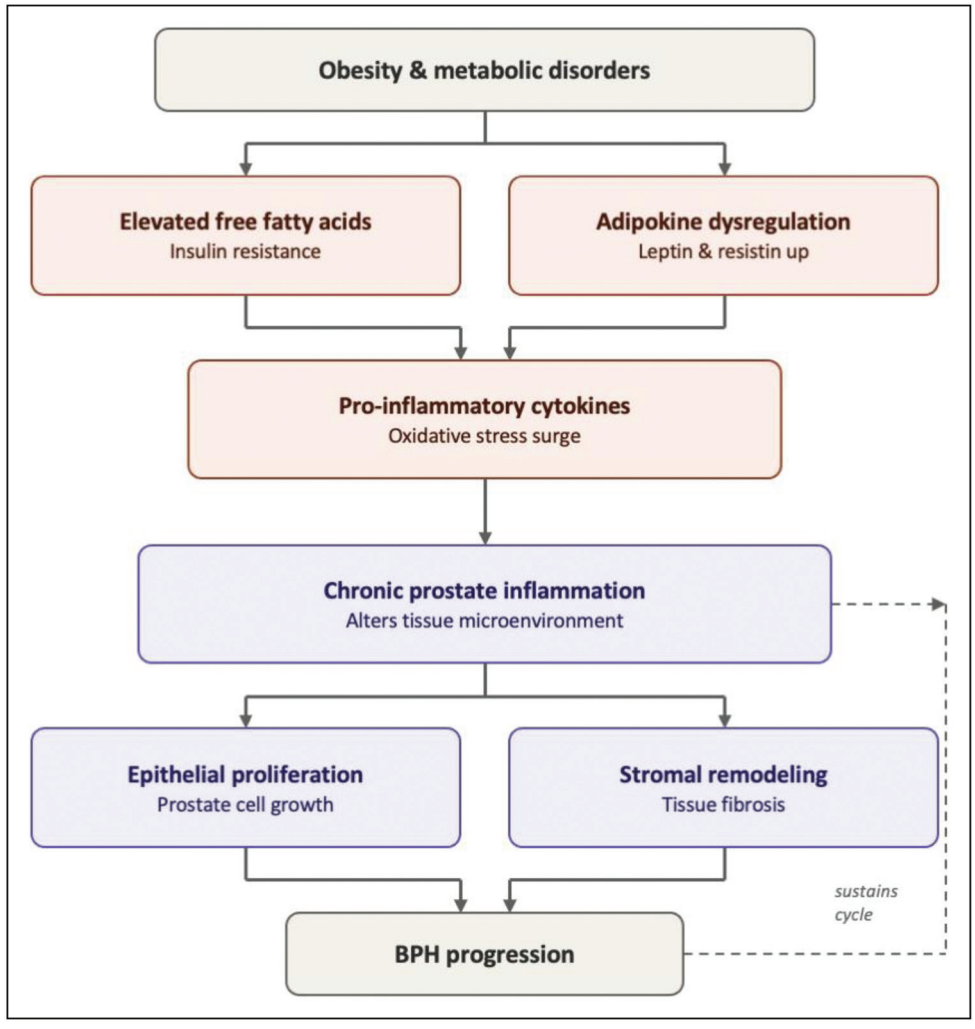

FIGURE 2. Pathway linking obesity and metabolic dysregulation to BPH progression. Local prostatic effects (purple) are driven by systemic metabolic and inflammatory changes (coral); the dashed line indicates the self-reinforcing feedback loop between BPH-related tissue injury and systemic inflammation [54].

be managed as a metabolic disease alongside associated comorbidities [42] (Table 1).

SGLT-2i lowers urinary supersaturation by increasing urine volume and by modulating acid–base status in a phenotype-specific manner, thereby reducing lithogenesis. It also exerts anti-inflammatory and antifibrotic effects, reducing the expression of inflammatory markers, stone core matrix proteins, and adhesion molecules, as well as the formation of calcium oxalate crystals. Conversely, the combination of glucosuria and higher urine pH may promote bacterial growth and increase the risk of urinary tract infections, which can precipitate struvite stones unless the infection is cleared and the urine is acidified [44]. GLP-1 RA lower blood sugar, promote weight loss, and have local effects on microinflammation, thereby reducing the risk of stone formation [44].

In terms of surgical options in obese patients with KSD, supine Percutaneous Nephrolithotomy is preferred for unilateral low & moderate complex stone disease in comparison to Prone PCNL for large staghorn complex stone disease [45].

OBESITY AND FUNCTIONAL UROLOGY

Obesity is a potentially modifiable adverse factor in overactive bladder (OAB) as per the recent AUA/SUFU OAB guideline [46]. Positive association of obesity with the risk of wet OAB in both sexes, which may be avoided by controlling BMI, waist to stature ratio (WSR), body fat percentage (BFP), fat mass and strengthening lean mass [47]. There is also a significant association between the elevated weight-adjusted waist index (WWI) and OAB. An anti-inflammatory diet may help prevent OABas, as

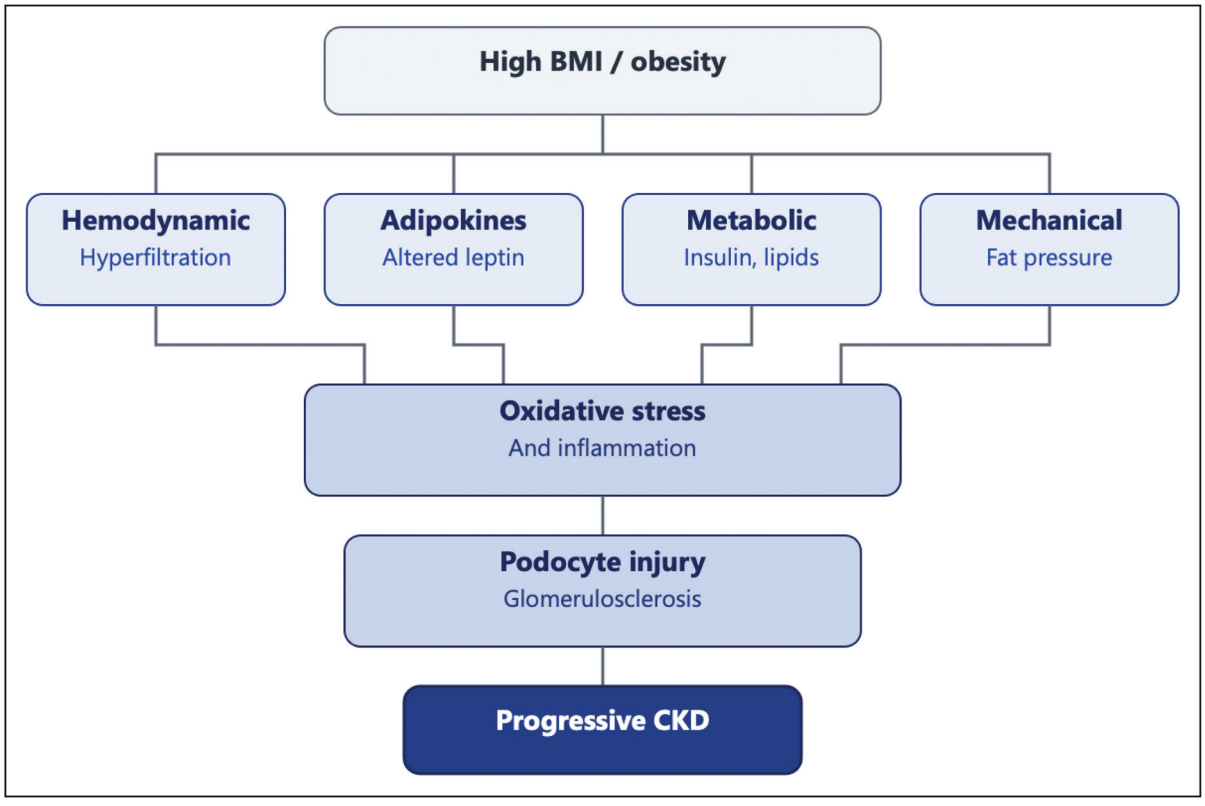

FIGURE 3. Pathway linking obesity to renal damage via multiple pathways leading to progressive chronic kidney disease [67].

indicated by a higher dietary Inflammatory Index (DII) [48]. Among patients with GLP-1 RA, the impact on OAB remains unclear. Patients with more baseline symptoms showed greater symptomatic improvement, but there is no direct evidence that these medications are more effective than weight-loss-related improvements [49]. In nondiabetic patients undergoing onabotulinumtoxin A (BTX–A) for refractory OAB, concurrent use of GLP-1 RA was associated with a lower risk of urinary retention and urinary tract infection, exerting beneficial effects beyond weight loss and metabolic outcomes [50]. Conversely, patients undergoing GLP-1 RA therapy reported Chronic pelvic pain syndrome/chronic prostatitis-like symptoms with negative infectious evaluation, which resolved after discontinuing medications [51]. Obese women with high intra-abdominal pressure are at higher risk of stress urinary incontinence (SUI), the pathophysiology of which is still not fully understood [52]. Bariatric procedures, especially Laparoscopic sleeve gastrectomy, have a beneficial secondary effect on urinary incontinence, more profoundly seen with a history of SUI in previous vaginal childbirth [53]. Obese patients who underwent mid urethral sling surgery for SUI experience lower cure and satisfactory rates than nonobese women after 10 years [52].

OBESITY AND MALE LUTS/BPE

Obesity increases fatty acid levels in the body, which, in turn, activate pro-inflammatory cytokines and oxidative stress pathways[54]. Obesity and elevated insulin levels alter the metabolic environment, thereby enhancing the proliferative response of prostatic tissues [55]. The Chinese visceral adipose index (CVAI) findings suggest that managing visceral fat content and maintaining a healthy fat distribution pattern help reduce the risk of benign prostatic enlargement, along with insights for early prevention and intervention of benign prostatic hypertrophy (BPH) [56]. Sarcopenic Obesity, which includes muscle mass, muscle strength and physical performance, is associated with a higher prevalence of Lower urinary tract symptoms/Benign Prostatic Enlargement among older men than Sarcopenia or Obesity alone [57▪▪]. Obesity and Insulin resistance potentially induce systemic metabolic changes that exacerbate the local inflammatory and oxidative stress responses in the prostate. The systemic factors, along with local tissue changes, accelerate prostatic hypertrophy, thereby creating a cycle in which metabolic factors continuously fuel the progression of prostate enlargement [54].

OBESITY AND MALE INFERTILITY

Obese patients have increased peripheral adipose tissue, which leads to increased aromatase activity,

1752-296X Copyright © 2026 Wolters Kluwer Health, LLC. All rights reserved.

Table 1. Overview of common urological conditions in relation to obesity

Urological conditions	Key findings	Points for takeaway
Kidney cancer	Obesity paradox in RCC – Higher risk with better survival.	Robotic Approach in obese patients; Weight monitoring around therapy. Risk stratification by type rather by BMI
Prostate, bladder & urothelial carcinoma	Aggressive, higher mortality prostate cancer; Modestly increased bladder cancer risk; Worse outcomes for Advanced urothelial carcinoma on-treatment weight loss.	
Surgical options	Robotic approach – lowest complication rate across urological procedures.	
Infections	SGLT-2 inhibitors – Fungal risk, modest risk in recurrent/ complicated UTI	Exercise caution with SGLT2-inhibitors
Kidney stones	Metabolic syndrome, Hypertension, and impaired glucose tolerance – Strong association for stone formation BMI >30 raises stone risk; Novel indices (ABSI, BRI) outperform BMI for stone prediction SGLT2 inhibitors – reduce lithogenesis, may raise struvite/ UTI risk.	Management as a metabolic disease; to consider SGLT2-i/ GLP-1RA as risk-modifiers
Functional urology	Obesity – Modifiable OAB risk factor; Obese patients have lower cure rates after mid-urethral sling at 10 years	Anti-inflammatory diet reduces risk; Consider weight loss/ bariatric surgery as first line adjuncts
Male infertility	Obesity driven Pseudo-hypogonadism; GLP-1 RA improved outcomes in Diabetes/ Metabolic syndrome	Addressing reversible factors before initiating testosterone
Chronic kidney disease	GLP-1 RA (FLOW trial) reduces renal failure risk & Cardiovascular death in T2DM with CKD	Consider GLP-1 RA therapy with CKD & DM
Renal transplant	Robotic approach – comparable outcomes with lower SSIs & shorter hospital stay	Offer Robotic approach where available
Reconstructive urology	Pyeloplasty patients show greatest eGFR improvement with comparable outcomes as normal individuals	Safe and effective in Obese patients
Paediatric urology	Maternal/ childhood obesity may impair testicular descent and Leydig cell function Obese children show higher urinary uric acid excretion, and Nocturnal enuresis	Metabolic screening at-risk children

CKD, chronic kidney disease; DM, diabetes mellitus; GLP-1 RA, Glucagon-like peptide-1 receptor agonists; RCC, renal cell carcinoma.

resulting in the conversion of circulating Testosterone to estrogen, leading to adult-onset hypogonadotropic hypogonadism. The Aromatase inhibitors help restore normal circulating testosterone levels in these patients [58]. Chronic inflammation associated with obesity can disrupt the hypothalamic–pituitary–gonadal (HPG) axis, leading to reduced testosterone production and, in ageing, late-onset hypogonadism, increasing the risk of chronic metabolic diseases [59]. Obesity-related reductions in Sex hormone binding globulin led to reduced testosterone concentrations with normal luteinizing hormone (LH) and follicle-stimulating hormone (FSH), causing pseudo-hypogonadism of obesity. Weight reduction, management of type 2 diabetes mellitus, obstructive sleep apnea, depression, and other comorbidities are part of the reversible mechanism of the obesity induced hypogonadism, avoiding unnecessary testosterone supplementation [60]. The addition of testicular replacement therapy (TRT) to lifestyle therapy mitigates weight-loss-induced reductions in muscle mass and bone mineral density by downregulating genes involved in muscle and bone anabolism [61]. Nondiabetic Obese men who received semaglutide (GLP1-RA) were more likely to develop erectile dysfunction and testosterone deficiency requiring medical therapy [62]. In contrast, in patients with Type 2 Diabetes Mellitus and Obesity who desire fertility, it markedly improved sperm morphology, total testosterone levels, and functional hypogonadism, and promoted weight loss; however, it did not demonstrate a direct action on testicular function [63–65]. Recently introduced Tirzepatide, a dual agonist of glucose-dependent insulinotropic peptide (GIP) and

GLP-1 receptors, is effective in improving metabolic parameters, erectile dysfunction, and gonadal hormone levels in obesity and metabolic hypogonadism [66].

OBESITY AND CHRONIC KIDNEY DISEASE

High BMI accounted for 23.4% of all CKD deaths and 27.3% of CKD-related DALYs globally, with a doubling trend by 2050. The age-standardized death rate nearly doubled from 2.69 to 5.06 per 100 000 [67]. The FLOW trial showed that a dose of 1 mg subcutaneous semaglutide reduced the risk of clinically important kidney outcomes, such as the onset of kidney failure, a 50% reduction in eGFR from baseline, and death from cardiovascular causes, in patients with Type 2 Diabetes Mellitus and chronic kidney disease [68].

OBESITY AND RENAL TRANSPLANT

Obese patients who underwent robotic-assisted kidney transplant have comparable outcomes to those undergoing open kidney transplant, including cold ischemia time, warm ischemia time, estimated blood loss, 1-year and 3-year survival, lower SSI incidence, and shorter hospital stay [69,70].

OBESITY AND RECONSTRUCTIVE UROLOGY

High BMI was not associated with inferior early perioperative outcomes, pain, complications or surgical success after single-port robot-assisted pyeloplasty for Uretero-pelvic junction obstruction (UPJO). In contrast, obese patients showed the greatest improvement in eGFR postoperatively [71].

OBESITY AND PAEDIATRIC UROLOGY

Childhood obesity is a serious public health concern with a wide array of urological conditions. Maternal obesity and gestational metabolic disorders may be associated with cryptorchidism, which may impair Leydig cell function and testicular development. Postnatal and Adolescent obesity may be an added risk factor for the shift in sex steroid balance in these children [72]. Continuous antibiotic prophylaxis (CAP) used in children with vesicoureteric reflux (VUR) to prevent recurrent urinary tract infections was significantly associated with an increased risk of overweight and obesity, a finding that should be interpreted cautiously in light of CAP's benefits [73]. Obesity is a potential risk factor for lower urinary tract symptoms, especially nocturnal enuresis and OAB [74▪▪]. Obese children with stone disease demonstrate higher urinary uric acid excretion, but BMI was not considered a risk factor for pediatric stone disease [75].

CONCLUSION

Obesity has a broad paradoxical effect across the urological spectrum, where higher BMI worsens prostate cancer outcomes yet has a protective effect in renal cell carcinoma and the recovery of eGFR post-pyeloplasty. It has modifiable outcomes in stone disease, functional urology, male infertility, pediatric urology, chronic kidney disease and renal transplantation driven by insulin resistance, chronic inflammation and hormonal dysregulation, representing challenges in modern urological practice. Recent evidence in children suggests its impact early in life, with relevance extending beyond adolescence. Incretin-based therapy, such as GLP-1 RA, and robotic surgery have a positive influence on the management of this spectrum of diseases, with a focus on future prevention and treatment strategies to improve overall health.

Acknowledgements

We would like to acknowledge the Mentorship Program of Christian Medical College, Vellore, Tamil Nadu, India, for bringing the authors together to draft this manuscript.

Financial support and sponsorship

None.

Conflicts of interest

There are no conflicts of interest. Badhrinath Jayapal and Nitin Kapoor have no financial or nonfinancial relationships or activities to declare in relation to this article.

REFERENCES AND RECOMMENDED READING

Papers of particular interest, published within the annual period of review, have been highlighted as:

- ▪ of special interest
- ▪▪ of outstanding interest

1. Obesity and overweight. https://www.who.int/news-room/fact-sheets/detail/obesity-and-overweight (Accessed 6 July 2026).
2. Okunogbe A, Nugent R, Spencer G, *et al.* Economic impacts of overweight and obesity: current and future estimates for 161 countries. BMJ Glob Health 2022; 7:e009773.
3. Handke A, Silberg M, Orf C, *et al.* Impact of obesity on systemic treatment outcomes in metastatic urological malignancies. Obes Sci Pract 2026; 12:e70164.

▪▪ A comprehensive review of the renal, bladder and prostate malignancies in relation to Obesity.

4. Does body mass index affect the proper preoperative grade and stage assessment in patients with prostate cancer undergoing radical prostatectomy? http://ceju.online/journal/2025/prostate-cancer-2502.php (Accessed 23 July 2026).
5. Jaber AR, Saikali S, Moschovas MC, *et al.* Impact of class III obesity (morbid obesity) on the perioperative, functional, and oncological outcomes of robotic-assisted radical prostatectomy. Cancers 2025; 17:709.
6. Sakthivel DK, Prabhakar P, Iyub MJR, *et al.* CLO26-117: from lean to obese: BMI's role in functional and cancer outcomes after nerve-sparing robotic-assisted radical prostatectomy. J Natl Compr Canc Netw 2026; 24: CLO26-117.

1752-296X Copyright © 2026 Wolters Kluwer Health, LLC. All rights reserved.

7. Kilic S, Sambel M. Impact of obesity on perioperative and clinical outcomes after robotic assisted radical prostatectomy. Sci Rep 2025; 15:225.
8. Fransis K, Tappero S, Dautricourt S, *et al.* Perioperative and clinical outcomes in patients undergoing Retzius-sparing robot assisted radical prostatectomy stratified by the degree of obesity. Int Urol Nephrol 2026; 58:4203–4212.
9. Drobot RB. Procedure-specific impact of body mass index on robot-derived intraoperative telemetry: a retrospective single-surgeon cohort study. Wideochir Inne Tech Maloinwazyjne 2026; 21:37–44.
10. Swami U, Jo Y, Narang A, *et al.* Correlation of body mass index with overall survival among patients with metastatic hormone-sensitive prostate cancer: analysis of patient-level data from SWOG-1216. Eur Urol Oncol 2025; 8:612–615.
11. Martini A, Shah QN, Waingankar N, *et al.* The obesity paradox in metastatic castration-resistant prostate cancer. Prostate Cancer Prostatic Dis 2022; 25: 472–478.
12. Levy S, Attia A, Elshazli RM, *et al.* Differential effects of GLP-1 receptor agonists on cancer risk in obesity: a nationwide analysis of 1.1 million patients. Cancers 2024; 17:78.
13. Gallagher M, Taylor B, Mirro I, *et al.* Perioperative glucagon-like peptide-1 receptor agonist use and surgical outcomes in patients with morbid obesity (BMI > 35) undergoing robotic urologic surgery: a propensity score-matched analysis. J Robot Surg 2026; 20:551.
14. ■■ Lu Y, Zhang S, Ma J, *et al.* Global spatio-temporal evolution and health inequalities in high BMI-associated kidney cancer burden from 1990 to 2021 and burden prediction to 2040. Front Oncol 2025;15:1630856.

The article highlights the increased impact of obesity in renal malignancies across the many countries.

15. Scelo G, Larose TL. Epidemiology and risk factors for kidney cancer. J Clin Oncol 2018; 36:3574–3581.
16. Koterazawa S, Sumiyoshi T, Shibasaki N, *et al.* Sex differences in the prognostic value of body mass index after curative nephrectomy for high-risk renal cell carcinoma: a multicenter cohort study. Int J Clin Oncol 2026; doi:10.1007/s10147-026-03134-8.
17. Feng Z, Wu Y, Hu Z, *et al.* Visceral fat measured with CT helps predict recurrence-free survival in patients with localized clear cell renal cell carcinoma. Int Urol Nephrol 2025; 58:501–510.
18. ■■ Chen H, Cai L, Zhuang J, *et al.* A nomogram including body composition parameters for predicting recurrence of pT1 clear cell renal cell carcinoma: a multicenter retrospective study. Insights Imaging 2026; 17:30.

An impactful review about body parameters apart from BMI for prognostication of renal malignancies.

19. Sato K, Takemura K, Oki R, *et al.* Prognostic significance of body mass index in patients with metastatic renal cell carcinoma receiving first-line therapies. Urol Oncol Semin Orig Investig 2025; 43:269.e1–269.e6.
20. Anami T, Motoshima T, Matsushita Y, *et al.* Impact of body mass index on the efficacy of immune combination therapy in metastatic renal cell carcinoma: a multicenter study in Japan. Int J Clin Oncol 2025; 30:2079–2086.
21. Deng Z, Graff RE, Batai K, *et al.* Polygenic score for body mass index in relation to mortality among patients with renal cell cancer. Int J Obes 2024; 48: 1735–1740.
22. Faheem MSB, Shahabi M, Ayub A, *et al.* Impact of obesity on peri-operative outcomes at robotic-assisted, laparoscopic and open partial nephrectomy using national inpatient sample data. J Robot Surg 2026; 20:272.
23. O'Hara J, Okhawere K, Soputro NA, *et al.* External validation of the preoperative risk evaluation for partial nephrectomy (PREP) score. BJUI Compass 2026; 7:e70239.
24. Yang X, Chen H, Yu H, *et al.* Prone retroperitoneal versus lateral retroperitoneal robotic assisted partial nephrectomy: a propensity score-matched comparison. World J Urol 2026; 44:127.
25. Pumfrey N, Azhar F, Harwood R, *et al.* Anatomy and patient comorbidity in robotic partial nephrectomy: predictors of complexity and outcomes. J Robot Surg 2026; 20:605.
26. Ghoreifi A, Dudinec JV, Ivey MC, *et al.* Acute kidney injury following radical nephrectomy and inferior vena cava thrombectomy: a tertiary referral center experience. Urol Oncol Semin Orig Investig 2026; 44:111029.
27. Rajpurohit M, Golzy M, Chen N-W, *et al.* Relationship among body mass index, survival, cancer treatment and health-related quality of life among older patients with bladder cancer. Cancers 2025; 17:1200.
28. Arulshankar S, Wei X, Ting C, *et al.* Impact of body mass index on outcomes after radical cystectomy: a retrospective Australian cohort study. BJUI Compass 2026; 7:e70238.
29. Arthuso FZ, Fairey AS, Boulé NG, *et al.* Associations between body mass index and bladder cancer survival: is the obesity paradox short-lived? Can Urol Assoc J 2022; 16:E261–E267.
30. Obesity and complication risk from radical cystectomy: identifying a body mass index threshold. https://www.auajournals.org/doi/epdf/10.1097/JU.0000000000002988 (Accessed 5 July 2026).
31. Khan SM, Hussain JM, Dar FF, *et al.* Prophylactic antibiotics and urinary tract infections after robot-assisted radical cystectomy: a narrative review. Urol J 2026; 03915603261456339.
32. ■■ Manta A, Roumpou A, Gerogianni M, *et al.* The impact of metabolic syndrome on survival outcomes in urothelial carcinoma: a retrospective cohort study. Front Oncol 2026; 16:1858817.

This article highlights the association of metabolic syndrome and its varied impact on the urothelial malignancies.

33. Chow RD, Mamtani R. Association between early on-treatment weight loss with enfortumab vedotin plus pembrolizumab and survival in advanced urothelial carcinoma. Clin Genitourin Cancer 2025; 23:102417.
34. Juliebø-Jones P, Nordanger IM, Beisland C, *et al.* Early and late complications associated with penile cancer surgery and the impact of human papillomavirus status: findings from a retrospective norwegian cohort study. Eur Urol Open Sci 2025; 72:29–35.
35. Tateo V, Thompson ZJ, Gilbert SM, *et al.* Epidemiology and risk factors of testicular cancer: a systematic review. Eur Urol 2025; 87:427–441.
36. Dieckmann K-P, Hartmann JT, Classen J, *et al.* Is increased body mass index associated with the incidence of testicular germ cell cancer? J Cancer Res Clin Oncol 2009; 135:731–738.
37. Aboukaoud M, Morhi Y, Osher E. Sodium-glucose co-transporter 2 inhibitors and severe urinary tract infections: real-world meta-analysis of cohort studies. Ann Pharmacother 2025; 59:891–903.
38. Xu J, Sun A, Yang Y, *et al.* Risk of urinary tract infections with sodium-glucose transport protein-2 inhibitors in subpopulations with abnormal genitourinary pathology. Clin J Am Soc Nephrol 2025; 20:820–828.
39. Ren J, Yang S, Wang Y, *et al.* Sodium–glucose cotransporter 2 inhibitor increases risk of urinary tract infection: evidence from mendelian randomization and meta-analysis. Br J Clin Pharmacol 2025; 91:2621–2627.
40. Dassanayake SN, Lafont T, Somani BK. Association and risk of metabolic syndrome and kidney stone disease: outcomes from a systematic review and meta-analysis. Curr Opin Urol 2025; 35:377.
41. Li W, Huang J, Liu Z, *et al.* Trends in the prevalence of kidney stones among U. S. adults with obesity from 2007 to 2020. Int J Surg Lond Engl 2025; 111: 5843–5851.
42. Garg M, Joseph P, Johnson H, *et al.* Obesity and BMI score as risk factors for urolithiasis: a systematic review over 30 years. Curr Opin Urol 2025; 35:338.
43. Hu X, Li X, Ye N, *et al.* Association of novel anthropometric indices with prevalence of kidney stone disease: a population-based cross-sectional study. Eur J Med Res 2024; 29:204.
44. Colin IM, Pozdzik A. Obesity, metabolic syndrome, diabetes and kidney stones: strengthening links over time?. touchREV Endocrinol 2026; 22:29–36.
45. Tee KTL, Loo CH, Goh YK, *et al.* Supine versus prone: a comparison of percutaneous nephrolithotomy outcomes in obese patients. Open J Urol 2025; 15:263–275.
46. The AUA/SUFU guideline on the diagnosis and treatment of idiopathic overactive bladder. https://www.auajournals.org/doi/epdf/10.1097/JU.0000000000003985 (Accessed 4 July 2026).
47. Zhang J, Chen W, Tang Z, *et al.* The association between obesity and wet overactive bladder: results from 2005 to 2020 National Health and Nutrition Examination Survey. Obes Facts 2025; 18:489–502.
48. Huang S, He Q, Zhao J, *et al.* Association of weight-adjusted waist index (WWI) with overactive bladder (OAB): a cross-sectional study from NHANES. Sci Rep 2025; 15:13207.
49. Sandler MD, Williams AD, Wein A, *et al.* Effects of glucagon like peptide-1 agonists on patients with overactive bladder: a pilot study. Cont Rep 2025; 14: 100083.
50. Hammad MAM, Quesada SG, Belczyk AL, *et al.* Beyond glycemic control: concurrent GLP-1 receptor agonist use is associated with reduced urinary adverse events following onabotulinumtoxina treatment in non-diabetic adults with overactive bladder. Toxins 2025; 17:542.
51. Tariq A, Garza Gangemi A, Herrera Caceres J. Beyond weight loss: effect of GLP-1 receptor agonist therapy on the urological health. Urol Case Rep 2026; 65:103368.
52. Lundmark Drca A, Westergren Söderberg M, Ek M. Obesity as an independent risk factor for poor long-term outcome after mid-urethral sling surgery. Acta Obstet Gynecol Scand 2024; 103:1657–1663.
53. Sternau M, Czajkowski M, Wierzbicki P, *et al.* Bariatric surgery has a long-term beneficial impact on urinary incontinence in women with obesity. Medicina (Mex) 2025; 61:564.
54. Zhou Y, Luo Q, Wang R, *et al.* Integrated management strategies for benign prostatic hyperplasia. Front Urol 2025; 5:1641171.
55. Ratajczak W, Walczakiewicz K, Laszczyńska M, *et al.* The profile of oxidative stress markers (arachidonic and linoleic acid derivatives) in patients with benign prostatic hyperplasia in relation to metabolic syndrome. Aging 2025; 17:116–130.
56. Li B, Li J, Sun C, *et al.* Association between the Chinese visceral adiposity index and the risk of benign prostatic hyperplasia: a national prospective cohort study. Sci Rep 2025; 15:222.
57. ■■ Zhou Z, Liu G, Ji J, *et al.* Association of sarcopenic obesity with benign prostatic hyperplasia/lower urinary tract symptoms: a nationwide cross-sectional study. Eur Urol Open Sci 2025; 82:81–87.

An Impactful review of Sarcopenic obesity in lower urinary tract symptoms when compared to Sarcopenia or obesity alone.

58. Pozzi E, Corsini C, Salonia A. Medical therapy for male infertility. Curr Opin Urol 2025; 35:157.
59. Tsutsumi T, Tsuchiya K. Testosterone and obesity in an aging society. Biomolecules 2025; 15:1521.
60. Muir CA, Wittert GA, Handelsman DJ. Approach to the patient: low testosterone concentrations in men with obesity. J Clin Endocrinol Metab 2025; 110: e3125–e3130.

61. Viola V, Samanta T, Nava MLD, *et al.* Testosterone modulation of muscle transcriptomic profile during lifestyle therapy in older men with obesity and hypogonadism. J Cachexia Sarcopenia Muscle 2025; 16:e13697.
62. Able C, Liao B, Saffati G, *et al.* Prescribing semaglutide for weight loss in nondiabetic, obese patients is associated with an increased risk of erectile dysfunction: a TriNetX database study. Int J Impot Res 2025; 37: 315–319.
63. Semaglutide improved sperm morphology in obese men with type 2 diabetes mellitus and functional hypogonadism. https://dom-pubs.pericles-prod.literatumonline.com/doi/epdf/10.1111/dom.16042 (Accessed 20 July 2026).
64. Deameh MG, Ramez M, Rowaiee R, *et al.* Effects of glucagon-like peptide-1 receptor agonists on male reproductive hormones, semen parameters, and metabolic outcomes: a systematic review. J Sex Med 2026; 23:qdaf381.
65. Salvio G, Ciarloni A, Ambo N, *et al.* Effects of glucagon-like peptide 1 receptor agonists on testicular dysfunction: a systematic review and meta-analysis. Andrology 2025; 13:2022–2034.
66. La Vignera S, Cannarella R, Garofalo V, *et al.* Short-term impact of tirzepatide on metabolic hypogonadism and body composition in patients with obesity: a controlled pilot study. Reprod Biol Endocrinol 2025; 23:92.
67. Ying J, Lan X, Zhou H, *et al.* Global, regional, and national burdens of chronic kidney disease attributable to high body mass index from 1990 to 2021, with future forecasts up to 2050: a systematic analysis for the global burden of disease study. Front Public Health 2025; 13:1612300.
68. Perkovic V, Tuttle KR, Rossing P, *et al.* Effects of semaglutide on chronic kidney disease in patients with type 2 diabetes. N Engl J Med 2024; 391:109–121.
69. Pratama ADM, Shenelo SA, Kamila RAF. Efficacy of robot-assisted versus open kidney transplantation in obese patients: a systematic review and meta-analysis. Clin Transplant Res 2026; 40:39–54.
70. Rondot A, Levy S, Mercier J, *et al.* Living-donor kidney transplantation: comparison of robotic-assisted versus conventional open technique in obese recipients. World J Urol 2026; 44:181.
71. Qi J-C, Yang H-X, He Y, *et al.* Navigating the challenge: does obesity compromise outcomes in single-port robot-assisted pyeloplasty? J Robot Surg 2026; 20:633.
72. Zhang D, Li Q, Yang Z, *et al.* Front Endocrinol 2026; 17:
73. Fujii T, Harada N, Katami H, *et al.* Oral antibiotic exposure and the risk of overweight and obesity in children with vesicoureteral reflux. J Pediatr Surg 2026; 61:163003.
74. ■■ Rho BY, Lee SW, Lee YS, *et al.* Association between overweight and lower urinary tract symptoms in children and adolescents: a systematic review and meta-analysis. J Pediatr Urol 2025; 21:1087–1097.
The article highlights the impact of obesity in children in relation to storage lower urinary tract symptoms.
75. Zhong J, Yang B, Liu Y, *et al.* The impact of body mass index on quantitative 24-h urine chemistries in pediatric urolithiasis: a systematic review and meta-analysis. Urol J 2026; 23:37–47.

1752-296X Copyright © 2026 Wolters Kluwer Health, LLC. All rights reserved.