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The Impact of Obesity on Lower Urinary Tract Function: a Literature Review

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Abstract Traditionally, lower urinary tract dysfunction was attributed to poor viscoelasticity of the bladder, neurogenic causes, and the enlarged prostate. The etiology of lower urinary tract symptoms (LUTS) extends well beyond prostate enlargement and bladder outlet obstruction. There are many publications of the impact of obesity on diverse urological conditions, malignant and nonmalignant, in terms of patient clinicopathological parameters and outcomes. The literature is characterized by often conflicting results. However, there is increasing evidence showing that metabolic syndrome and, in particular, obesity can adversely affect lower urinary tract function. This review article will study the effect of obesity and metabolic syndrome on LUTS, BPH, overactive bladder (OAB), stress urinary incontinence (SUI), and post-prostatectomy outcomes.

Keywords Obesity · Metabolic syndrome · LUTS

Introduction

The lower urinary tract is involved in the storage and elimination of urine. The micturition cycle is divided into two phases: (i) bladder filling and (ii) voiding. There is coordination of contraction and relaxation of muscles of the urinary bladder and urethral sphincters during the micturition cycle. When there is dyssynergia between the bladder and sphincter, lower urinary tract dysfunctions occur.

Traditionally, lower urinary dysfunction was attributed to poor viscoelasticity of the bladder, neurogenic causes, and the enlarged prostate. There has been a growing interest in the relationship between benign prostatic hyperplasia (BPH), lower urinary tract symptoms (LUTS), obesity, body mass index (BMI), and metabolic syndrome. The literature increasingly shows an association between lower urinary tract dysfunction, obesity, and metabolic syndrome.

Obesity and Metabolic Syndrome

Obesity and overweight are defined as abnormal or excessive fat accumulation that may impair health. BMI is a widely accepted index that classifies adults as obese or overweight and is defined as a person's weight in kilograms divided by the square of his height in meters (kg/m^2). The World Health Organization (WHO) definitions are: overweight BMI≥25 and obesity BMI≥30. Obesity is a worldwide health problem which has more than doubled since the 1980s. In 2014, nearly 40 % of the worldwide adult population were overweight, and more than 10 % were classified as obese. Obesity is preventable [1]. In 2011–2012, the prevalence of obesity in the United States was 16.9 % in youth and 34.9 % in adults [2].

Metabolic syndrome has been recognized since the 1920s and includes an assortment of fatal heart attack risk factors including diabetes, abdominal obesity, high cholesterol, and high blood pressure. The United States Third National Health and Nutrition Examination Survey estimated the age-adjusted prevalence of metabolic syndrome to be 24 % and showed an increasing prevalence of metabolic syndrome with age. More than 40 % of the cohort above 60 years old suffered from metabolic syndrome [3]. There has been a plethora of

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definitions for metabolic syndrome. The exact pathogenesis of metabolic syndrome is complex and not fully elucidated. However, central obesity and insulin resistance are postulated to be important causative factors.

The International Diabetes Federation (IDF) in 2006 came out with a comprehensive definition that is the most widely accepted internationally [4]. A person is defined to be suffering from metabolic syndrome if they have:

Central obesity using waist circumference (definition differs for different ethnic groups, Europid men ≥94 cm, Europid women ≥80 cm) and two of the following four factors:

(i) high triglyceride (TG) level: ≥150 mg/dL (1.7 mmol/L), or receiving treatment for hypertriglyceridemia
(ii) low high-density lipoprotein (HDL) cholesterol: <40 mg/dL (1.03 mmol/L) for males and <50 mg/dL (1.29 mmol/L) for females, or receiving treatment for low HDL levels
(iii) hypertension: systolic BP≥130 or diastolic BP≥85 mmHg, or receiving anti-hypertensives or having been previously diagnosed to suffer from hypertension
(iv) high fasting plasma glucose (FPG)≥100 mg/dL (5.6 mmol/L), or previously having being diagnosed to have type 2 diabetes.

Traditionally, obesity is associated with cardiovascular diseases, diabetes, and an assortment of musculoskeletal disorders. But nowadays, the effect of obesity and metabolic syndrome is recognized to be beyond cardiovascular effects and extending to quality of life, social, behavioral, and emotional domains.

There are many publications of the impact of obesity on diverse urological conditions, malignant and nonmalignant, in terms of patient clinicopathological parameters and outcomes. The literature is characterized by often conflicting results. However, there is increasing evidence showing that metabolic syndrome, especially obesity, can adversely affect the function of the lower urinary tract.

Benign Prostatic Hyperplasia (BPH)

Lower urinary tract symptoms are common among middle age and elderly men.

Approximately 90 % of men in their seventies have LUTS secondary to BPH [5]. There is a one-third chance of a man in his fifties to undergo treatment for BPH in his lifetime. The etiology of LUTS extends beyond prostate enlargement and bladder outlet obstruction [6].

Data from the Boston Area Community Health Survey examined the relationship between metabolic syndrome and LUTS. The severity of LUTS was quantified by the American Urological Association Symptom Index (AUASI). Prevalence of metabolic syndrome was 30 % and the prevalence of moderate or severe LUTS defined as an AUASI 8 or greater to be 20 %. The prevalence of metabolic syndrome was 20 % for men with minimal LUTS and was double at 40 % in those reporting LUTS (AUASI symptom score 2 or above). Metabolic syndrome was significantly associated with incomplete emptying, intermittency, and nocturia. The association between the presence of LUTS and metabolic syndrome supports common underlying factors between lower urinary tract symptoms and extra-urinary chronic conditions [7].

Similar findings are seen from the Health Professionals Follow-up Study (HPFS)—a prospective analysis using a cohort of more than 50,000 American men 40–75 years old over a 16-year period. This large prospective study showed that abdominal adiposity as well as adult weight gain was associated with both increased LUTS incidence and progression [8•].

The Air Force Health Study studied 1206 participants over 15.6 years. The risk of BPH was higher with increasing age, height, and fasting blood glucose levels. However, there was no significant relationship between metabolic syndrome, weight, body mass index, and lipid levels with BPH [9].

In view of conflicting results from epidemiological studies relating obesity to BPH and LUTS, Wang et al. conducted a meta-analysis to study the association between obesity and BPH. A total of 9 studies with good study design were included and showed a positive association of BMI with BPH and LUTS. The odds ratio was 1.27, and the 95 % confidence intervals were 1.05–1.53 [10•].

He et al. looked specifically at the relationship of central obesity with lower urinary tract symptoms. The authors postulated that BMI was not specific enough, only representing overall body fat, whereas waist circumference was a better representative of central adiposity. They performed meta-analysis of all case control or cohort studies on obesity and LUTS. Twelve studies met the inclusion criteria, representing 83,304 cases. A 10 cm increase in waist circumference was statistically associated with a 2.5 % increase in the risk of LUTS [11•].

The studies mentioned above suggest an association between obesity, prostate volume, and lower urinary tract symptoms. Thus, assuming that the association that we observed between BMI and the risk of LUTS is causal, almost 20 % of severe LUTS cases in the United States could be prevented if men maintained a normal body weight. As mentioned previously, obesity is reversible, thus making it an extremely attractive target for LUTS prevention and perhaps treatment [8•]. So, from another angle, can weight loss improve LUTS?

There is little data on whether weight loss can prevent and improve LUTS. A recent randomized controlled trial with 130 patients over a 1-year period showed that a comprehensive intensive weight reduction program was no better than a standard advice at weight reduction and improvement in lower

urinary tract symptoms. In view of the negative results between the two groups, the investigators studied the whole cohort of obese male subjects. However, there was still no significant difference in LUTS between the obese and non-obese groups. The authors postulated that the subjects were of Chinese ethnicity with a small proportion of subjects belonging to the obese group which might have masked subtle effects and poor compliance of patients to a weight loss program, with weight regained at subsequent follow-ups [12].

At present, the literature suggests a positive relationship between obesity, physical inactivity, high BMI, and metabolic syndrome with LUTS. Conflicting data may be due to incomplete definitions, as some studies just focus on BMI, which just takes account of total body weight or general obesity, and not abdominal girth or abdominal obesity, which appears to be the culprit behind. Further data is needed to confirm that weight loss can bring about LUTS prevention and perhaps treatment.

Post-Prostatectomy Incontinence

Carcinoma of the prostate is the most common cancer in men from developed countries, with more than 1.1 million new cases in 2012 worldwide [13].

The most common treatment for organ-confined disease is radical prostatectomy. Urinary incontinence is a common and distressing side effect for patients undergoing radical prostatectomy. Rates of post-prostatectomy incontinence (PPI) vary in the current literature and can be as high as 80 % [14]. Functional outcomes after radical prostatectomy greatly influence patients' quality of life, and urinary continence has been shown to have a higher impact on quality of life than sexual functioning [15]. Traditionally, urinary continence had been heavily attributed to anatomical factors and surgical techniques including bladder neck preservation, bladder neck, anterior and posterior reconstruction, urethral length preservation, preservation of the endopelvic fascia, and nerve-sparing approach [16]. More recently, there is an increasing realization that patient factors including obesity is related to PPI.

Wiltz et al. studied oncological and functional outcomes in more than 700 overweight and obese men undergoing robotic radical prostatectomy. At both the 12 and 24 months follow-up, obese men had significantly lower urinary continence and potency rates. Seventy-five percent of normal weight men were continent, in contrast to 57 % of obese men at 24 months, and this was statistically significant [17].

In an observational study of 600 patients, Wolin et al. studied whether there was a difference in postoperative continence between someone who vigorously exercises and is overweight/obese vs someone who is overweight/obese but not physically active (defined by Wolin et al. as exercising 1 h or greater per week). The incontinence rate was 59 % in obese and physically inactive men at 1 year [18].

In the MIRROR study (Multicenter Italian Report on Radical Prostatectomy), an independent Italian multicenter prospective observational study on radical prostatectomy, abdominal obesity (using above 102 cm as a cutoff) was associated with a higher intraoperative complication rate, higher perioperative complication rate, and worse functional outcome. Obese men were three times more likely to suffer from intraoperative complications and to receive blood transfusions and were 2.5 times more likely to use two or more pads per day [19•].

However, in a prospective Canadian observational study with more than 300 subjects, BMI was not a statistically significant predictor of early continence at 1-month post-prostatectomy [20].

There is conflicting literature on the association between obesity and PPI. However, the majority support an association of high BMI and low physical activity with post-prostatectomy incontinence.

Overactive Bladder

Overactive bladder (OAB) is a clinical syndrome defined as "urgency, with or without urgency incontinence, usually with frequency and nocturia" [21]. In a 2011 prevalence study in the United States, the prevalence of OAB was more than 30 %. Detrusor overactivity which is defined as a urodynamic observation of involuntary detrusor contractions during the filling phase is postulated to arise from increased cellular excitability with abnormal propagation either in the detrusor muscle (myogenic), other bladder cell types (integrative), or the central nervous system (neurogenic) [22].

The Finnish National Nocturia and Overactive Bladder (FINNO) Study looked at the prevalence and risk factors of LUTS in an adult population based sample of more than 3700 subjects. There was no significant association between obesity and urgency. However, there was a threefold increase risk of urgency urinary incontinence in obese women [23].

In view of paucity of literature on OAB, obesity and metabolic syndrome in women, a systematic review of 27 studies was performed by Bunn et al. to study the association of metabolic syndrome with LUTS and OAB in women. The authors found that that there was limited evidence on this subject and that the available evidence were often of poor quality. However, the limited available evidence supported metabolic syndrome as both contributors and predictors of LUTS in women [24].

A recent case control study from Turkey which included 204 women showed that the presence of metabolic syndrome, waist circumference, and neck circumference were statistically associated with OAB ($p<0.001$ for all 3 factors). Cutoff values for OAB were 35.25 cm for neck circumference and 98.5 cm for waist circumference. This is the first study in which neck circumference was used as an indicator for obesity in the study of the association with OAB [25].

Overactive bladder and obesity are not conditions that solely occur in the adult population. A Taiwanese study showed that 24 % of obese children suffered from overactive bladder, as compared to 14.5 % of normal weight children, and was statistically significant [26]. Childhood obesity is increasing at an alarming rate worldwide, with a doubling in childhood obesity prevalence.

The data on the association between high BMI with urge urinary incontinence and OAB is not mature. However, the available evidence suggests that there is a positive association of OAB with metabolic syndrome and obesity.

Nocturia

Nocturia, defined by the International Continence Society (ICS) as urinating one or more times a night [21], is a common problem for the elderly. Population studies show that nocturia is prevalent, affecting 25 % of women and 20 % of men [27]. Nocturia is multifactorial and is associated with obesity, diabetes, cardiac disease, hypertension, lifestyle factors, and obstructive sleep apnea.

The Boston Area Community Health Survey—a large cohort study including more than 1800 random individuals—found an association of nocturia with a metabolic syndrome, and this association was more marked in those younger than 60 years old [28]. The FINNO Study also showed that obese subjects were twice as likely to suffer from nocturia as compared to their normal weight counterparts [23].

Nocturia is a common condition with apparent association with metabolic syndrome and obesity.

Stress Urinary Incontinence

Stress urinary incontinence (SUI) is multifactorial, and previous pregnancies, pelvic trauma, hormonal factors, prior pelvic surgeries, and radiotherapy have been postulated to be contributing factors. An association between obesity and urinary incontinence has been shown in epidemiological studies, and an odds ratio of 1.6 per 5 unit increase in body mass index has been shown. In the population-based study FINNO, the risk of SUI was double in obese women [23].

Chen and colleagues showed that obese women contemplating bariatric surgery experienced statistically significant higher urinary incontinence severity score using the Sandvik incontinence severe scale, as compared to a control group of non-obese women. Over 705 of women who were considering bariatric surgery suffered from urinary incontinence, with 60 % having stress incontinence, 53 % with urgency incontinence, and 42 % with mixed incontinence [29].

A secondary analysis of the RCT data on female mid-urethral slings from Canada compared surgical outcomes for obese and non-obese cohorts. Obese women had both a significantly lower 12-month objective (68 % vs 86 %) and subjective cure rates (70 % vs 85 %) as compared to normal weight subjects after mid-urethral sling surgery. The intraoperative and postoperative complications were similar in both groups [30].

Contrasting data was found in a study that looked at the whether BMI affected the success rate of mid-urethral sling surgery. BMI was not independently associated with failures in 243 women who underwent mid-urethral sling surgery over a 36-month follow-up period [31]. In view of the equivocal results, a meta-analysis was performed to compare the efficacy of mid-urethral slings in obese women with SUI. The meta-analysis studied 13 studies and showed that the objective and subjective cure rates were not statistically different in both obese and non-obese women [32].

Multiple studies have shown a clear relationship between weight loss from lifestyle modification and improved urinary continence. A meta-analysis of 6 articles including 2352 subjects showed that non-surgical weight loss intervention improved urinary continence and concluded that non-surgical weight loss should be first-line management of urinary incontinence in obese and overweight women [33].

Urinary continence improves in more than 805 of morbidly obese patients undergoing bariatric surgery, with a significant improvement in mean Sandvik severity index score from 5.4 to 2.3 postoperatively [34].

The literature supports a higher incidence of urinary incontinence in obese and overweight patients. Both non-surgical weight loss and surgically-induced weight loss improve urinary continence rates. However, there is conflicting data on whether the outcomes are worse in obese patients undergoing surgical treatment of urinary incontinence.

Mechanism of Obesity on LUT Function

In view of some conflicting results from the observational and cohort studies, it is important to know the mechanism and the pathophysiology in which obesity brings about LUTS. However, the exact mechanism by which obesity is linked to lower urinary tract function is not clearly elucidated. A few theories including increased chronic inflammation, obesity-associated perturbations in energy and insulin regulation, sympathetic activity, and increased estrogen to testosterone ratio have been postulated.

Obesity may be linked to increased sympathetic nervous system activity, leading to increased storage LUTS from smooth muscle contraction [35]. Hammarsten did some interesting work on the relationship of diabetes and obesity with BPH. Data from 158 consecutive LUTS patients suggested that BPH was a component of the metabolic syndrome, and that the metabolic abnormality of a defective insulin-mediated

glucose uptake and secondary hyperinsulinemia were both seen in patients with BPH and those with metabolic syndrome [36]. An increased sympathetic nerve activity in BPH men has also been hypothesized.

Obese men also have an increased estrogen to testosterone ratio, which can contribute to prostatic tissue hyperplasia. It is well recognized that sexual hormones are essential for normal prostate development and growth. Androgens are also important in the maintenance of BPH as demonstrated by the size reduction of an enlarged prostate with androgen deprivation therapy [37]. However, it has not been confirmed whether changes in serum concentrations of androgens are directly associated with the onset of LUTS or clinical BPH.

However, researchers from the Boston Area Community Health (BACH) Survey could not find any correlation of circulating levels of sex hormones with urological symptoms. The study showed that circulating levels of sex hormones did not significantly predict LUTS in men after age adjustment. The pathophysiology of LUTS is complex and likely includes other factors on top of circulating sex steroid levels [38].

A study investigating the role of insulin as a promoter of BPH showed prostate gland volume to correlate with fasting serum insulin. The association was found to be statistically significant. Moreover, the study showed that insulin and free oestradiol were independent risk factors for BPH, further affirming that insulin and free oestradiol were BPH promoters. The authors concluded that hyperinsulinemia which is an important feature of the metabolic syndrome may be an underlying primary event in BPH [39].

Data from the Third National Health and Nutrition Examination Survey (NHANES III)—a cross-sectional study representative of the US population—showed an association between biochemical markers of the metabolic syndrome with LUTS. Men with LUTS had higher glycosylated hemoglobin as compared to men without symptoms. A higher level of glycosylated hemoglobin was associated with higher odds of LUTS. These results were statistically significant [40].

Metabolic syndrome has been associated with an elevated circulating C-reactive protein (CRP) level. A Taiwanese study showed that storage LUTS and sensory bladder disorders were associated with a higher CRP level. The serum CRP levels were significantly positively associated with age and serum PSA levels; and negatively associated with voided volume and maximum flow rate. The authors suggested that CRP levels were associated with storage symptoms, suggesting that chronic inflammation was associated with LUTS/BPH [41].

For the relationship between obesity and stress urinary incontinence, it is postulated that obese women have increased intra-abdominal pressure (IAP) which adversely stress the pelvic floor and, together with neurophysiological mechanisms, contribute to stress urinary incontinence [42]. An increased abdominal pressure has been linked to urgency urinary incontinence through causing detrusor overactivity [43]. Moreover, obesity may be associated with neurogenic disease, which could further impact the pelvic floor and cause urethral dysfunction [44].

Conclusion

This review has shown that there is a positive association between obesity, metabolic syndrome, and different aspects of lower urinary tract function.

There is abundant evidence that indicates a correlation between obesity and several urological diseases. Increasing data show that there is an effect of long-term weight loss on urinary incontinence, but there is still conflicting data on the effect of weight loss on the treatment and prevention of BPH, LUTS, and overactive bladder. The data is exciting in that obesity and metabolic syndrome are modifiable risk factors for lower urinary tract dysfunction, and that we can comfortably advise patients to have lifestyle changes and a trial of conservative management prior to medical and surgical treatment for urological conditions.

The limitation of the review is that the majority of the studies are observational studies/cohorts, and that a causality relationship cannot be established. Moreover, the exact mechanism of how obesity causes lower urinary tract dysfunction is not yet clearly established. More basic science studies and randomized controlled trials need to be conducted in order to allow us to clearly understand the exact pathogenesis and mechanism of this effect.

Compliance with Ethics Guidelines

Conflict of Interest Ada T. L. Ng and M. K. Yiu declare that they have no conflict of interest.

Human and Animal Rights and Informed Consent This article does not contain any studies with human or animal subjects performed by any of the authors.

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