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Adiposity-Induced Voiding Dysfunction: Unraveling the Association Between Overweight Status and Symptom Severity in Non-Retentive Benign Prostatic Hyperplasia

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A B S T R A C T

Benign prostatic hyperplasia (BPH) has historically been viewed through a prostato-centric lens, attributing lower urinary tract symptoms (LUTS) primarily to prostatic volume and mechanical obstruction. However, this model fails to account for the substantial symptom burden observed in patients without significant retention or massive enlargement. Emerging evidence suggests that systemic metabolic dysregulation, particularly adiposity, plays a crucial role in the pathophysiology of LUTS. This study aims to evaluate the association between Overweight status and the subjective severity of LUTS in a specific cohort of non-retentive BPH patients, thereby isolating metabolic contributors from acute mechanical failure. We conducted an observational analytic cross-sectional study at the Urology Polyclinic of RSUD Dr. Moewardi, Surakarta, Indonesia, from June 2024 to January 2025. The study enrolled 110 men diagnosed with BPH who met strict criteria for non-retentive status (post-void residual <150 mL, no indwelling catheter). Participants were stratified into normal BMI (<25 kg/m²) and Overweight (≥25 kg/m²) groups. Symptom severity was quantified using the International Prostate Symptom Score (IPSS). Data were analyzed using the Mann-Whitney U test and Chi-square analysis. The cohort comprised 72 (65.5%) normal-weight and 38 (34.5%) overweight patients. A statistically significant disparity in symptom severity was observed. The overweight group exhibited a significantly higher mean IPSS (17.87 ± 5.18) compared to the normal group (11.54 ± 4.71) ($p<0.001$). Notably, 44.7% of overweight patients presented with severe LUTS, compared to only 5.6% of normal-weight patients. Conversely, 90.9% of patients with mild symptoms belonged to the normal-weight group. In conclusion, overweight status is significantly associated with increased LUTS severity in non-retentive BPH patients. The findings suggest that adiposity exacerbates voiding dysfunction through systemic inflammatory, hormonal, and autonomic pathways independent of urinary retention. These results advocate for the integration of weight management as a core therapeutic strategy in BPH care.

1. Introduction

Benign prostatic hyperplasia (BPH) represents one of the most prevalent progressive conditions in the aging male population, creating a substantial global health burden. Histologically defined by the non-malignant proliferation of epithelial and stromal cells within the transition zone of the prostate, the clinical manifestation of BPH is characterized by lower urinary tract symptoms (LUTS). These symptoms are traditionally bifurcated into storage symptoms

(urgency, frequency, nocturia) and voiding symptoms (hesitancy, intermittency, weak stream), which collectively deteriorate the patient's quality of life (QoL).[1]

For decades, the urological community has operated under a volume-centric paradigm. This model posits that symptom severity is a direct function of prostate size and the resulting Bladder Outlet Obstruction (BOO). Consequently, therapeutic algorithms have prioritized the reduction of prostate volume via 5-alpha reductase inhibitors or the mechanical alleviation of obstruction through alpha-blockers and surgical intervention.[2] However, clinical practice frequently presents a paradox that this model cannot explain: patients with massive prostatic enlargement often report minimal symptoms, while those with relatively small glands suffer from debilitating voiding dysfunction. This discordance strongly implies the existence of extra-prostatic drivers of symptomatology.

In recent years, the pathophysiological understanding of BPH has undergone a paradigm shift toward a systemic metabolic model. Metabolic syndrome (MetS)—encompassing central obesity, insulin resistance, dyslipidemia, and hypertension—has been identified as a potent promoter of prostatic inflammation and growth. Central to this metabolic cluster is adiposity. Adipose tissue is no longer viewed merely as an inert energy storage depot but as a dynamic endocrine organ. It secretes a vast array of bioactive substances, including pro-inflammatory cytokines, adipokines, and growth factors, which can induce a state of chronic systemic inflammation. This inflammatory milieu is hypothesized to infiltrate the prostate and bladder, sensitizing afferent nerves and altering smooth muscle tone, thereby generating symptoms independent of physical obstruction.[3-5]

Despite the growing recognition of this metabolic BPH phenotype, a critical gap remains in the literature regarding the non-retentive population. Most existing studies utilize mixed cohorts that include patients with acute urinary retention or decompensated bladders. The inclusion of retention introduces a significant mechanical confounder, as retention represents a catastrophic failure of the bladder pump that maximizes symptom scores regardless of the patient's metabolic status. By focusing strictly on a non-retentive cohort—patients who maintain the ability to void but suffer from significant symptoms—we can more accurately isolate the contribution of metabolic factors to symptom generation.[4]

This research distinguishes itself by strictly isolating a non-retentive population to investigate the metabolic tipping point of LUTS. Unlike previous studies that conflate obstructive complications with symptom burden, this research focuses on the compensated phase of BPH where metabolic interventions could be most effective. Furthermore, it provides specific data on the Southeast Asian phenotype, utilizing the Asia-Pacific BMI cutoff (≥25 kg/m^2) for overweight status, which offers higher sensitivity for metabolic risk in this population compared to Western standards. The primary objective of this research is to evaluate the association between overweight status and LUTS severity, as quantified by the International Prostate Symptom Score (IPSS), in a homogenous cohort of non-retentive benign prostatic hyperplasia patients. By establishing this correlation, we aim to validate adiposity as a critical, modifiable determinant of symptom burden, thereby supporting a holistic treatment approach that extends beyond the prostate gland itself.

2. Methods

We employed an observational analytic study with a cross-sectional design to assess the relationship between body mass index (BMI) and symptom severity at a single point in time. The study was conducted at the Urology Polyclinic of Dr. Moewardi General Hospital, Surakarta, Indonesia, a tertiary academic referral center serving a diverse population in Central Java. The data collection period extended from June 2024 to January 2025. The study population consisted of male patients presenting to the urology clinic with clinical symptoms suggestive of BPH. A consecutive sampling technique was utilized to recruit 110

respondents who met the specific eligibility criteria designed to eliminate mechanical confounders. Inclusion Criteria: Male patients diagnosed with BPH based on clinical history, digital rectal examination (DRE) revealing benign consistency, and ultrasonography confirming prostatic enlargement; Willingness to participate and sign informed consent; Literacy and cognitive capability to complete the IPSS questionnaire independently. Exclusion criteria: to ensure the isolation of metabolic factors from mechanical failure and other pathologies, the following exclusions were strictly applied: urinary retention: defined as a post-void residual (PVR) volume >150 mL measured via transabdominal ultrasonography, or the presence of an indwelling catheter; structural abnormalities: presence of urethral stricture, bladder stones, or history of prior lower urinary tract surgery (such as TURP, open prostatectomy); neurological and renal comorbidities: neurogenic bladder (secondary to stroke, spinal cord injury) and chronic kidney disease; active infection: active urinary tract infection, which would artificially inflate irritative symptom scores.

Independent variable (overweight status): anthropometric measurements including weight (kg) and height (m) were obtained using calibrated instruments. BMI was calculated as weight divided by height squared (kg/m²). Patients were stratified according to the Asia-Pacific classification for metabolic risk: normal: BMI < 25.0 kg/m²; overweight: BMI ≥ 25.0 kg/m² (This category encompasses both overweight and obese classifications to capture the full spectrum of excess adiposity). Dependent variable (LUTS severity): symptom severity was assessed using the validated Indonesian version of the International Prostate Symptom Score (IPSS). This instrument comprises seven questions evaluating incomplete emptying, frequency, intermittency, urgency, weak stream, straining, and nocturia. Each item is scored from 0 to 5, yielding a total score range of 0–35. Severity was categorized as: mild: score 0–7; moderate: score 8–19; severe: score 20–35. Data were processed using SPSS version 22.0. Descriptive statistics were generated to characterize the demographic profile of the cohort. The normality of the data distribution was assessed using the Kolmogorov-Smirnov test. Due to the non-normal distribution of the IPSS data, non-parametric statistical tests were employed. The Mann-Whitney U test was utilized to compare the mean ranks of IPSS scores between the normal and overweight groups. Chi-square analysis was performed to evaluate the association between categorical BMI status and LUTS severity tiers. A p-value of ≤ 0.05 was considered statistically significant.

3. Result and Discussion

Figure 1 serves as the demographic and clinical baseline, defining the parameters of the cohort (N=110) prior to the stratification analysis. The data presented here is crucial for understanding the context of the findings, as it characterizes a specific, rigorously selected phenotype of patients presenting to a tertiary academic medical center in Indonesia: the non-retentive male with Benign Prostatic Hyperplasia (BPH). By visualizing the fundamental distribution of metabolic status (Body Mass Index) and clinical symptom burden (International Prostate Symptom Score), Figure 1 establishes the heterogeneity present within this defined group, setting the stage for investigating how these two primary variables interact. The metabolic profile (body mass index distribution) panel A of Figure 1 delineates the anthropometric landscape of the cohort, specifically focusing on metabolic risk as quantified by body mass index (BMI). The study utilized the Asia-Pacific classification system, setting the threshold for overweight status at ≥ 25 kg/m², a cut-off recognized for its higher sensitivity in detecting metabolic comorbidities in this specific ethnic population compared to Western standards. The graphical representation reveals that the study successfully captured a metabolically diverse group of patients. The majority of the cohort, comprising 65.5% (n=72) of the participants, fell within the normal weight category (< 25 kg/m²). This subgroup represents the baseline urological patient in whom metabolic drivers are theoretically minimized, allowing their symptoms to be attributed primarily to

age-related prostatic enlargement or other non-metabolic factors. Conversely, a substantial and statistically meaningful proportion of the cohort, 34.5% (n=38), was classified as overweight (≥ 25 kg/m²). This segment encompasses patients across the spectrum of excess adiposity, ranging from pre-obesity to established clinical obesity. The presence of over one-third of the study population in this high-metabolic-risk category is significant. It reflects the increasing prevalence of metabolic syndrome prevalent in modern urban Indonesian society and ensures that the study is adequately powered to detect potential differences in symptom symptomatology driven by adiposity. This distribution confirms that the study is not examining a niche population but rather a representative cross-section of patients presenting for urological care in a tertiary setting, allowing for results that have broader clinical applicability. The clinical symptom burden (LUTS Severity) Panel B focuses on the dependent variable of the study: the subjective severity of LUTS, assessed via the validated Indonesian version of the IPSS. This panel is critical for characterizing the clinical reality of the non-retentive patient. Crucially, this visualization dispels the potential misconception that patients without urinary retention are relatively asymptomatic or in a sub-clinical phase of the disease. The data demonstrates that the vast majority of this cohort experiences significant voiding dysfunction and QoL impairment. Only a minority of patients, 20.0% (n=22), reported mild symptoms (IPSS 0-7), a category often managed with watchful waiting. The dominant clinical presentation, encompassing 60.9% (n=67) of participants, was moderate symptoms (IPSS 8-19). This represents the typical patient requiring medical therapy (alpha-blockers or 5-ARIs) to manage bothersome symptoms that interfere with daily activities. Furthermore, a nearly equal proportion to the mild group, 19.1% (n=21), presented with severe symptoms (IPSS 20-35). These patients suffer from debilitating frequency, urgency, nocturia, and voiding difficulty, despite having post-void residual volumes below 150 mL.

We have a cohort of 110 aging men with confirmed BPH who have maintained bladder compensation (absence of retention). Within this clinically homogeneous group regarding retention status, there is significant heterogeneity in both metabolic health and symptom burden. We observe a population that is far from asymptomatic, with nearly 80% reporting moderate-to-severe distress. Simultaneously, we see a population split between metabolically healthy (normal weight) and metabolically at-risk (overweight) individuals. This specific constellation of demographic and clinical characteristics—a symptomatic, non-retentive, metabolically diverse cohort—provides the ideal substrate for testing the study's central hypothesis: that the observed variation in symptom severity (Panel B) is not random but is significantly influenced by the patient's metabolic status (Panel A).

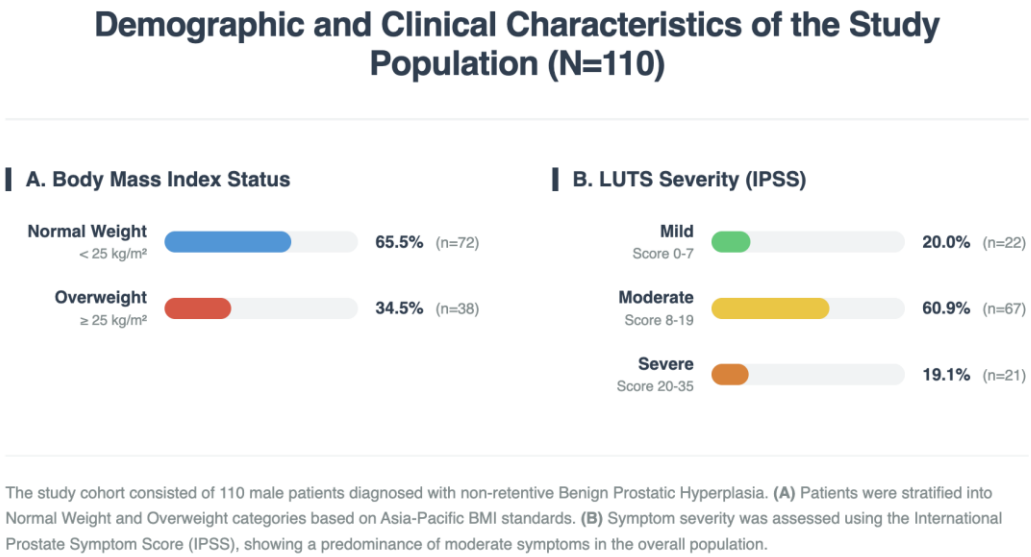


Figure 1. Demographic and clinical characteristics of the non-retentive BPH.

Figure 2 encapsulates the core empirical findings of the study, presenting a multi-panel graphical analysis of the association between overweight status and LUTS severity. Building upon the baseline characteristics established in Figure 1, this figure visualizes the direct statistical comparison between the normal BMI cohort (n=72) and the overweight cohort (n=38). Through two distinct visualizations—a proportional stacked bar analysis (Panel A) and a comparison of central tendencies (Panel B)—Figure 2 demonstrates a profound and statistically significant divergence in the clinical trajectories of these two groups. The data presented here provides robust evidence rejecting the null hypothesis, illustrating that in a non-retentive BPH population, adiposity is not merely a comorbid feature but a potent determinant of symptom worsening. Panel A utilizes stacked bar charts to provide a striking visual representation of how patients in each BMI category are distributed across the tiers of LUTS severity (mild, moderate, severe). This visualization reveals a dramatic severity migration driven by overweight status. The normal BMI bar serves as the clinical baseline. In this group, the symptom distribution follows a standard pattern often seen in early-to-mid-stage BPH. The largest segment is moderate symptoms (66.7%), representing the typical manageable burden of disease. A substantial proportion maintains mild symptoms (27.8%), indicating successful bladder compensation and minimal bothersomeness.

Notably, the severe segment is small, comprising only 5.6% (n=4) of normal-weight patients. This profile suggests that in the absence of significant metabolic dysregulation, non-retentive BPH tends to manifest with manageable symptomatology. In sharp contrast, the overweight bar reveals a fundamentally different clinical reality. The distribution shifts markedly toward the severe end of the spectrum. The mild symptom category nearly vanishes, representing only 5.3% (n=2) of overweight patients, suggesting that it is rare for an overweight patient with BPH to remain minimally symptomatic. While half the group (50.0%) falls into the moderate category, the most significant finding is the explosion of the severe segment. A remarkable 44.7% (n=17) of overweight patients were classified as having severe LUTS.

The juxtaposition of these two bars highlights a powerful epidemiological finding from the study: whereas a normal-weight patient has a roughly 1 in 18 chance of having severe symptoms, an overweight patient has nearly a 1 in 2 chance. Furthermore, a supplementary annotation highlights a critical statistic derived from this distribution: 81.0% of all severe cases in the entire study population originated

from the overweight group. This indicates that excess adiposity is highly overrepresented among the most severely affected patients. While panel A visualizes categorical shifts, panel B quantifies the magnitude of this difference using continuous data. It presents a side-by-side comparison of the mean total IPSS scores for both groups, complete with error bars representing the standard deviation (SD), providing insight into both central tendency and variability. The normal group exhibited a mean IPSS of 11.54 (SD ±4.71). This score sits squarely in the lower-middle range of the moderate category, consistent with the distribution seen in panel A. The error bar indicates that while there is variability, the majority of these patients cluster around a manageable symptom baseline.

Conversely, the overweight group recorded a significantly elevated mean IPSS of 17.87 (SD ±5.18). This score is approaching the threshold for "Severe" (20+). The absolute difference in means is 6.33 points. In the context of the IPSS, a change of ≥3 points is generally considered clinically perceptible to the patient. Therefore, the observed difference is not only statistically meaningful but represents a substantial, clinically relevant deterioration in quality of life. The standard deviation in the overweight group is slightly larger, suggesting a wider spectrum of symptom presentation, but the entire distribution is shifted upwards. Above the bars, a bracket annotates the result of the non-parametric Mann-Whitney U test, confirming that this difference is highly statistically significant with a p-value of < 0.001. This extreme level of significance indicates that there is less than a 0.1% probability that the observed difference in symptom scores between normal and overweight patients is due to random chance alone. Taken together, the panels of Figure 2 provide a comprehensive statistical argument. Panel A shows where the patients end up clinically (a massive shift to severe symptoms), and Panel B shows how much worse their symptoms are on average (a clinically significant 6-point increase). The combination of these graphical analyses offers compelling evidence that in patients who have not yet succumbed to urinary retention, overweight status acts as a major aggravator of benign prostatic obstruction, effectively accelerating the symptomatic progression of the disease from a manageable state to a debilitating one.

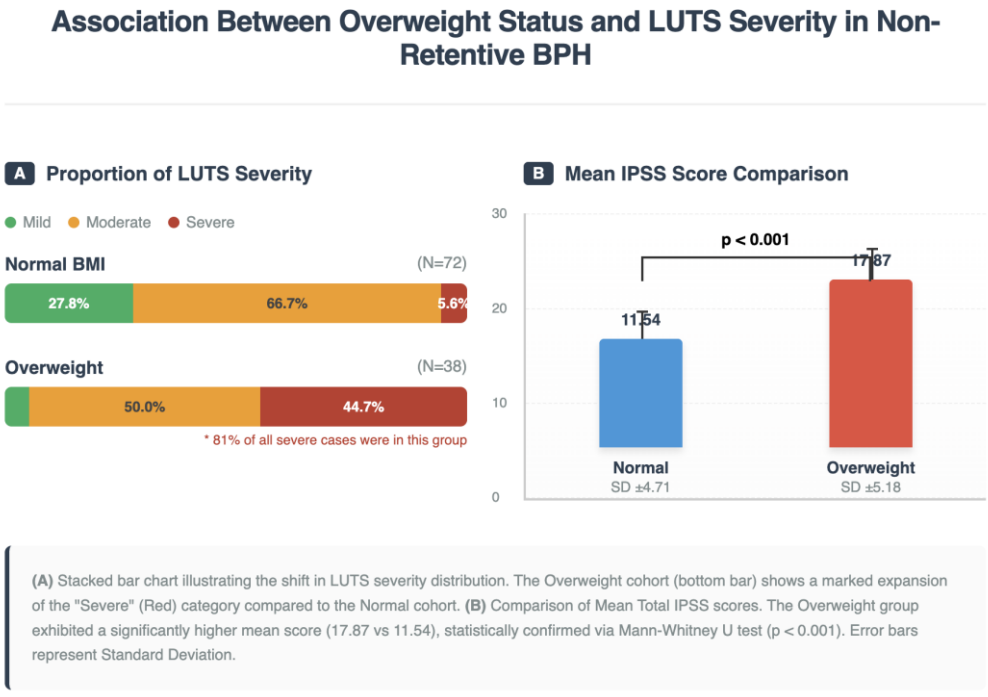
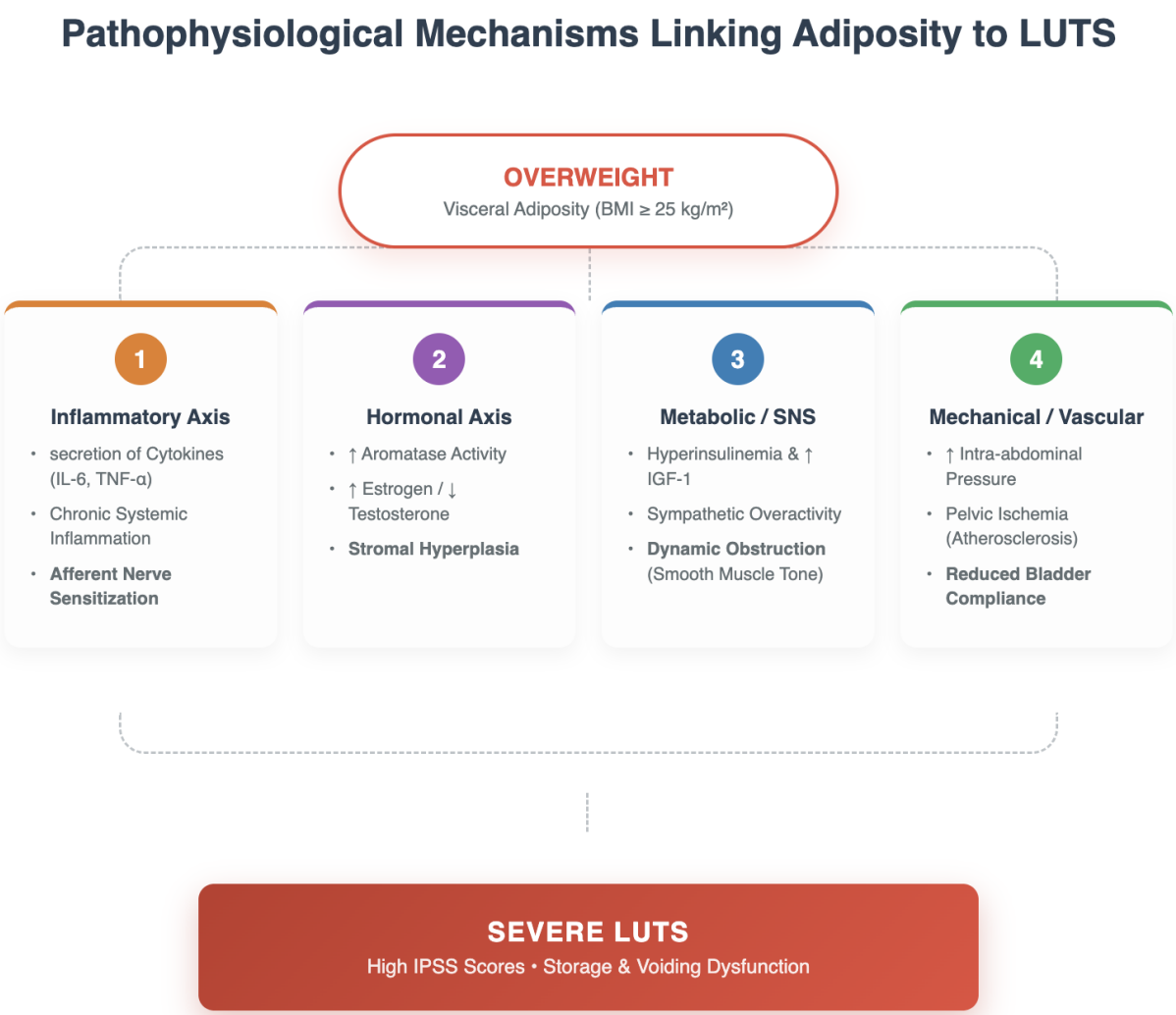


Figure 2. Association between overweight status and LUTS severity in non-retentive BPH.

The results of this study provide compelling evidence of a robust association between overweight status and the exacerbation of lower urinary tract symptoms (LUTS) in patients with benign prostatic hyperplasia (BPH). Our analysis demonstrated that the mean IPSS score in overweight patients (17.87) was significantly higher than in normal-weight patients (11.54), with a remarkable shift toward severe symptomatology in the overweight cohort. This finding is not merely a statistical correlation but a reflection of complex underlying pathophysiological interactions between adipose tissue, systemic metabolism, and the lower urinary tract environment. By isolating the non-retentive population, we have effectively highlighted the "metabolic burden" on the compensated bladder. Figure 3 translates the statistical associations observed in Figure 2 into a coherent pathophysiological framework. It serves as a schematic representation of the "multi-hit" hypothesis proposed in the manuscript's discussion, illustrating the complex biological mechanisms through which systemic adiposity translates into localized lower urinary tract dysfunction. This diagram moves beyond simple correlation to explore causation, mapping the theoretical pathways by which excess adipose tissue acts not as a passive bystander, but as an active, pathogenic driver of severe LUTS in the non-retentive patient. The figure is structured as a flow diagram, originating from the systemic source (overweight), branching into four distinct yet interacting pathological pathways, and converging on the final clinical outcome. This positioning emphasizes that the entire cascade is initiated by the metabolic state of the host. It is crucial to recognize that in this framework, adipose tissue is conceptualized not merely as increased body mass but as a dysregulated endocrine organ, particularly the visceral fat component central to metabolic syndrome. This central node acts as the generator for a variety of systemic signals that disrupt urological homeostasis.[6-8]

Visceral fat chronically secretes pro-inflammatory cytokines such as IL-6 and TNF-α. These mediators enter systemic circulation and induce a localized inflammatory response within the prostate and bladder. The key consequence highlighted here is afferent nerve sensitization.[9,10] Chronic inflammation lowers the nociceptive threshold of C-fibers in the bladder mucosa. This explains why non-retentive patients experience storage symptoms like urgency and frequency; their bladders are chemically irritated and signal a false need to void even at low volumes. Hormonal axis pathway addresses endocrine dysregulation. Adipose tissue contains high levels of aromatase, an enzyme that converts testosterone into estrogen. This leads to an altered hormonal milieu characterized by a high estrogen-to-androgen ratio. Estrogen is a potent mitogen for prostatic stromal cells. The primary outcome here is stromal hyperplasia, suggesting that adiposity drives a more biologically aggressive form of prostate growth, contributing to the static component of obstruction.[11,12] Metabolic/SNS axis pathway links hyperinsulinemia (a hallmark of insulin resistance in overweight states) to autonomic dysfunction. High insulin levels stimulate the Sympathetic Nervous System (SNS). The prostate gland is rich in alpha-adrenergic receptors responsible for smooth muscle contraction. Chronic sympathetic overactivity results in Dynamic Obstruction—a state where the prostate smooth muscle is chronically tense, restricting urine flow independent of the gland's physical size.[13] Mechanical axis pathway accounts for physical and vascular changes. Central obesity increases intra-abdominal pressure, exerting extrinsic force on the bladder. Simultaneously, the systemic atherosclerotic state associated with obesity leads to pelvic ischemia (chronic hypoperfusion). Ischemia induces fibrosis in the bladder wall, leading to reduced bladder compliance (a stiff bladder).[14,15] This inability to stretch properly further exacerbates storage symptoms like frequency and urgency. The diagram concludes with the convergence of these four pathways into a single outcome box: severe LUTS. This visual synthesis demonstrates that the severe symptomatology observed in overweight patients is rarely due to a single mechanism. Instead, it is the

cumulative effect of an irritated, sensitized bladder (inflammatory), an actively growing prostate (hormonal), a chronically contracted prostatic urethra (metabolic/SNS), and a stiff, compressed bladder (mechanical/vascular). Figure 3 provides a sophisticated biological rationale for the clinical data. It explains how an overweight patient without urinary retention (with a bladder pump that still works) can nonetheless suffer from severe symptoms. The dysfunction is not just about failing to empty; it is about a lower urinary tract that is chemically sensitized, hormonally stimulated, autonomically overactive, and vascularly compromised. This framework underscores the necessity of treating BPH in overweight patients as a systemic metabolic disease rather than a purely localized prostatic issue.[16]



Schematic Description: This figure illustrates the "Multi-Hit" hypothesis derived from the study findings. **(1)** Visceral fat generates inflammation, sensitizing bladder nerves (Urgency/Frequency). **(2)** Altered estrogen/androgen ratios drive tissue growth. **(3)** Metabolic dysregulation increases sympathetic tone and smooth muscle tension. **(4)** Physical compression and vascular ischemia reduce bladder compliance. Collectively, these pathways explain the high prevalence of severe symptoms in non-retentive overweight patients.

Figure 3. Pathophysiological mechanisms linking adiposity to LUTS.

The most robust theoretical framework explaining the high prevalence of severe symptoms in our overweight cohort is the concept of chronic systemic inflammation derived from visceral adiposity. Overweight individuals possess an expanded volume of visceral adipose tissue, which is metabolically active and functions as a secretory organ. It releases a variety of pro-inflammatory cytokines, including Interleukin-6 (IL-6), Interleukin-8 (IL-8), and tumor necrosis factor-alpha (TNF-α). These circulating cytokines induce a localized inflammatory response within the prostate gland, often referred to as metabolic prostatitis.[16-18] Histological studies have shown that BPH specimens from obese individuals often contain dense inflammatory infiltrates, primarily T-lymphocytes and macrophages. This chronic inflammation drives two pathological processes: stromal proliferation: inflammatory cells secrete growth factors that stimulate the proliferation of prostatic stromal cells, contributing to gland enlargement; afferent nerve sensitization: more importantly for symptom generation, inflammatory mediators sensitize the afferent C-fibers in the prostate and bladder neck. This lowering of the nociceptive threshold results in sensory urgency, where the patient feels a strong need to void even at low bladder volumes. This mechanism plausibly explains why the overweight patients in our study reported higher IPSS scores; the irritative or storage symptoms (frequency, urgency, nocturia) are driven by this inflammatory sensitization rather than mechanical obstruction alone.[18]

Our findings also align with the hormonal theory of BPH progression in the context of adiposity. Adipose tissue contains high levels of aromatase, an enzyme responsible for the peripheral conversion of testosterone to estrogen (estradiol). Consequently, overweight men often exhibit a paradoxical hormonal profile: reduced serum testosterone (due to feedback inhibition) and elevated estradiol levels. This elevated estrogen-to-androgen ratio is critical because estrogen is a potent stimulator of stromal cell proliferation in the prostate. Estrogen acts synergistically with dihydrotestosterone (DHT) to increase prostate volume. Furthermore, estrogen receptors (ER-alpha) are upregulated in inflamed prostatic tissue. This hormonal environment likely promotes a more aggressive, biologically active form of BPH. The significantly higher IPSS scores in our overweight group may reflect this accelerated, metabolically driven prostatic growth, which creates a more dynamic and resistant obstruction than the slower, age-related growth seen in lean men.[3,19]

Overweight status is intimately linked with hyperinsulinemia and insulin resistance, key components of the metabolic syndrome. Insulin itself is a growth factor, but it also increases the bioavailability of insulin-like growth factor 1 (IGF-1) by suppressing the production of IGF-binding proteins in the liver. Both insulin and IGF-1 receptors are upregulated in BPH tissue, promoting cellular mitosis and inhibiting apoptosis. Beyond growth, hyperinsulinemia stimulates the sympathetic nervous system (SNS). The prostate and bladder neck are rich in alpha-1 adrenergic receptors, which control smooth muscle tone. Chronic sympathetic overactivity, driven by high insulin levels in overweight patients, leads to increased smooth muscle tone in the prostate. This dynamic obstruction restricts urine flow even in the absence of massive glandular enlargement. This autonomic dysregulation offers a compelling explanation for why our non-retentive overweight patients experienced such severe symptoms—their prostates are in a state of chronic, hormonally-driven contraction.[18-20]

A critical, often overlooked mechanism supported by our results is the role of pelvic atherosclerosis and ischemia. Overweight status is a major risk factor for endothelial dysfunction and atherosclerosis. The pelvic ischemia hypothesis suggests that atherosclerotic narrowing of the pelvic arterial bed leads to chronic hypoperfusion of the prostate and bladder. Chronic ischemia creates a hypoxic environment that induces structural changes in the bladder wall. Specifically, it leads to smooth muscle fibrosis and collagen deposition, resulting in a non-

compliant or stiff bladder. This loss of compliance manifests clinically as detrusor instability, urgency, and frequency. This theory elucidates why overweight patients might experience severe symptoms even if their prostate volume is not massively enlarged; the bladder itself has become dysfunctional due to compromised blood flow.[17] Our finding that 44.7% of overweight patients had severe symptoms is likely driven by the compounding effects of prostatic obstruction and ischemic bladder dysfunction.

Finally, the physical mechanics of central adiposity contribute to symptom severity. Excess intra-abdominal fat exerts direct external pressure on the bladder. While this increased pressure can theoretically aid in voiding (via the Valsalva maneuver), it simultaneously reduces functional bladder capacity. The bladder is chronically compressed, meaning it reaches its fullness threshold at lower urine volumes. This manifests clinically as urinary frequency and urgency, independent of prostate size. This mechanical phenomenon acts as an independent aggravating factor, worsening the voiding parameters measured by the IPSS and contributing to the stark severity shift observed in our overweight cohort.[18]

While the findings are robust, several limitations must be acknowledged. First, the cross-sectional design allows for the identification of associations but cannot definitively establish causality between overweight status and symptom severity. Second, BMI was used as the sole surrogate for adiposity; while practical, it does not distinguish between visceral and subcutaneous fat, which have different metabolic profiles. Future studies utilizing waist circumference or visceral fat analysis would offer greater precision. Third, while we rigorously excluded retention, we did not perform invasive urodynamics (pressure-flow studies) to definitively differentiate between bladder outlet obstruction and detrusor underactivity. Lastly, as a single-center study in a tertiary referral hospital, selection bias may exist, as patients presenting to such centers often have more advanced disease than the general population.

4. Conclusion

This study establishes a definitive and significant association between overweight status and the severity of lower urinary tract symptoms in patients with benign prostatic hyperplasia without urinary retention. Patients with a BMI ≥ 25 kg/m² exhibit significantly higher IPSS scores and a markedly increased prevalence of severe symptoms compared to their normal-weight counterparts. These findings support a multifaceted pathophysiological model where adiposity exacerbates BPH through systemic inflammation, hormonal imbalance, sympathetic overactivity, pelvic ischemia, and mechanical compression. The clinical implications of this study are profound. Urologists should move beyond a purely prostate-centric view and adopt a holistic approach that includes metabolic evaluation. Weight status should be recognized as a key risk factor for severe symptomatology, and weight management strategies should be prioritized as essential, adjunctive interventions in the clinical management of BPH. By addressing the metabolic drivers of the disease, clinicians can potentially alleviate symptom burden and improve the quality of life for this growing patient population.

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