RESEARCH PAPER

Decoding Urinary Incontinence: Pathophysiological Basis and Approaches to Prevention

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ABSTRACT

Urinary incontinence affects people of all ages, and it is a significant public health issue across the globe. The pathophysiology of UI is complex and involves anatomical, neuromuscular, and hormonal mechanisms that result in bladder dysfunction. This review emphasises the importance of public awareness, the removal of associated stigma, and the integration of a holistic approach to enhance the quality of life of individuals with UI. Prevalence estimates are quite variable due to the lack of uniformity in the definitions and research methods. Still, they all convey the same message that it is a common problem, especially among the elderly and women after giving birth. The need for such frameworks in the clinical setting is evident, as they are useful in identifying the cause of the disease, as well as in implementing the most appropriate management strategies. In addition to standard health care, new insights into the genetic, microbiological, and physiological aspects of the condition hold promise for future preventive and personalized treatments. Bridging existing research gaps, especially those related to early detection markers, precision-based therapies, and microbiome-driven interventions, will be essential in transforming UI care in the coming years. Advances in genetics, microbiome research, and precision medicine highlight future directions for improved prevention and individualized care.

Keywords: UI, Pathophysiology, Diagnosis, Pelvic Floor Dysfunction, Management Strategies, Preventive Interventions.

Brief Summary: With a focus on prevention and better clinical results, this review provides an overview of the pathophysiology, kinds, risk factors, diagnostic techniques, and evidence-based therapeutic methods for UI.

List of abbreviations

UI = UI

MS = Multiple Sclerosis

COX 2= Cyclooxygenase-2

NSAIDs Non-steroidal anti-inflammatory drugs

ACE = Angiotensin-converting enzyme

ATFP = Arcus tendineus fascia pelvis

COPD = Chronic obstructive pulmonary disease

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1. INTRODUCTION

UI(UI) is an involuntary leakage of urine affecting adult women and men worldwide. According to the International Continence Society, UI is an involuntary leakage of urine and is a loss of bladder control. UI is most predominant in postmenopausal women (in the early postpartum period, (20-35%) and in pregnant women (30-40%) and is frequent [1]. Although UI has a greater impact on health, many individuals are unaware of available treatment options or are reluctant to seek advice [2]. Treating UI cannot be underscored, firstly, it has a high prevalence amongst all age groups across different demographics. Thus, it needs a widespread awareness and education. Secondly, this condition greatly impacts the quality of life, social interactions, daily activities, and emotional well-being [3,4]. Thirdly, there is a persistent requirement to remove the embarrassment that prevent many people from getting treatment [5]. Finally, effectively managing the condition can lead to considerable reductions in health care expenditures, and significantly emphasises the need for research and proactive treatment options [6]. As per reports, postpartum women also experience UI prevalence, and are influenced by factors like vaginal deliveries, age, and obesity [7]. The prevalence of UI among the male population is 11% (between 60-65 years), and 31% (older than 65 years) [8]. UI can significantly impact the quality of life, and physically it causes skin irritation due to urgency. Emotionally, UI can cause depression, anxiety, and social isolation, seriously affecting psychological well-being and self-esteem. Socially, UI restricts activities and relationships, causing embarrassment and social withdrawal. Day-to-day life is disrupted, reducing productivity and limiting occupational performance. Thus, addressing UI is crucial for improving overall well-being and quality of life [9]. The UI classification (exhibiting symptoms), and the guidelines in treatment are mentioned in Fig.1. This review provides a comprehensive overview of UI and addresses the knowledge gap in understanding and identifying the condition, thereby empowering individuals to manage and treat conditions effectively.

2. METHODS AND METHODOLOGY

2.1 Design and Scope

This review comprehensively examines the pathophysiology, diagnosis, and management of UI. It aims to consolidate the findings from existing literature and provides an in-depth understanding of the condition. Also, it discourses the knowledge gaps, and propose the future directions for clinical practice and research.

2.2 Literature Search Strategy and Inclusion

A systematic search of electronic databases, including PubMed, Scopus, and Web of Science, was conducted to identify relevant studies published up to 28/12/2024. Keywords such as “UI,” “pathophysiology,” “diagnosis,” “treatment,” “stress incontinence,” and “urge incontinence” were used in combination with Boolean operators (AND, OR) to refine search results. Additional articles were identified through manual searches of reference lists and relevant review papers. Studies were included if they were published in peer-reviewed journals. The review search was focused on human subjects and addressed UI’s pathophysiology, diagnostic methods, or treatment options. The data were considered if the provided quantitative or qualitative data were relevant to the review’s objectives. Non-English publications were not included, as well as studies with insufficient data or unrelated to the core aspects of UI were also avoided. Similarly, research that was focused exclusively on paediatric populations or non-human subjects was also omitted.

2.3 Data Extraction and Analysis

Relevant data were systematically extracted and organized into categories, including the types of UI, their risk factors, diagnostic approaches, and treatment modalities. Emphasis was placed on identifying emerging trends, gaps in existing knowledge, and areas for future research. Information from high-quality studies, including randomized controlled trials, observational studies, and meta-analyses, was prioritized.

3.0 TYPES OF UI AND THEIR CAUSES

3.1 Stress UI

Urine leakage that occurs suddenly and without conscious control during activities that create intra-abdominal pressure, like exertion, sneezing, coughing, laughing, or straining, is identified as stress UI. Several physiological and anatomical factors combine to cause this syndrome, which interferes with the regular mechanisms that regulate urine continence. White ethnicity and female sex are risk factors that cannot be changed; however, smoking, obesity, and chronic constipation are amongst major risk [10].

Stress UI in women is largely caused by several circumstances, including pregnancy, multiple vaginal deliveries, menopause, overweight and obesity, and pelvic floor treatments, including hysterectomy. Younger women who are active in sports may experience stress UI [11]. Furthermore, stress incontinence is frequently linked to pelvic organ prolapse, which often occurs after childbirth or menopause. Men are much less likely to get stress UI, which usually happens after prostate surgery [12].

3.2 Urine Incontinence Urge

Involuntary pee leaking induced by detrusor overactivity may be preceded or followed by a feeling of urgency in the urine. The patient's neurologic control loss or bladder irritation may induce contractions [13]. Detrusor instability, an overactive bladder, or neurological illnesses, including multiple sclerosis, parkinson's disease, or stroke, have a significant influence on the cause of UI [14].

3.3 Overflow UI

Overflow UI occurs when an expanded bladder leaks pee due to diminished detrusor contractility and bladder outlet blockage. Detrusor function can be affected by neurologic diseases such as diabetes, MS, and spinal cord injuries, causing a hypotonic neurogenic bladder. Urethral strictures, pelvic organ prolapse, and abdominal or pelvic

mass pressures can block bladder outlets. Benign prostatic hyperplasia is common in men [15]. Overflow UI is the only physically hazardous type since it can induce renal failure and irreparable bladder damage.

3.4 Mixed UI

Mixed UI is the condition of involuntary leakage of urine and is caused by a combination of urge and stress UI. In this type either urge incontinence or stress incontinence is more predominant [16].

3.5 Functional UI

Functional UI is defined as the involuntary flow of urine caused by environmental or physiological barriers to toileting and can create toileting difficulties.

4.0 PREVALENCE

UI is a prevalent disorder that considerably affects individuals' health and imposes a strain on healthcare systems [17]. Studies shows that approximately 10% of adult women develop UI, rising to over 40% at 70 years. The prevalence is significantly greater in nursing facilities. Despite the prevalence of UI, numerous women are reluctant to pursue medical consultation due to associated stigma. Nonetheless, awareness is increasing, encouraging more women to seek therapy [18]. The user interface can impact decisions related to elder care and institutionalization. Economically, it incurs significant expenses - Sweden, for instance, allocates almost 2% of its healthcare budget to UI [19]. Assessing prevalence is difficult due to disparate definitions, demographic variations, and methodological inconsistencies among research [20]. Cultural attitudes, reporting behaviors, and methodological variations can obfuscate the actual frequency among locations. Studies of superior quality employing defined criteria and verified instruments produce the most dependable estimates, with industrialized countries typically providing more robust data [21]. Several forms of UI, such as stress, urgency, and mixed, are more prevalent at different life stages. Supplementary risk factors encompass obesity, childbearing, pelvic operations, chronic illnesses, and neurological disorders. Evidence indicates racial disparities in incidence, with white women exhibiting elevated rates of stress UI; however, the findings are equivocal [22].

5. PATHOPHYSIOLOGY

5.1 Anatomical Characteristics of the Urethra in Women

a) Weakened Urethral Mucosa Sealing

Urethral mucosa seals, and due to submucosal blood vessels, normal urethral mucus-like discharges close. The blood vessels in the urethra's submucosa provide the mucous membrane with blood, boost cell proliferation, thicken it, encourage mucous secretion, and help the lumen close. The filling blood vessels in the submucosa may compress the urethral lumen to promote sealing. Thus, estrogen dilates and fills the urethra's submucosal blood vessels, making the mucosa highly vascularized. So, the urethral mucosa's sealing function is enhanced and its blood supply is finally increased [23]. Subsequently, when estrogen levels are lower, it shrinks the urethra's submucosal blood vessels, reduce mucosal blood flow, atrophy the mucosa, and impair urethral mucosal sealing [24]. In chronic urethritis, long-term inflammatory stimulation promotes additionally urethral mucosal fibrosis and increases the risk of UI.

Incontinence can result from urethral sphincter dysfunction. The inner longitudinal smooth muscle works as a central filler and contracts with the outer circular smooth muscle fiber to maintain static urethral tension and reduce lumen diameter.
In women, the external urethral sphincter is Ω-shaped, and the urethra's outermost layer, it covers almost 80% of its length [25]. The anterior vaginal wall acts as a "plate" to compress the urethra toward the posterior urethral wall, resulting in continence. Morgan et al. identified that slow-contractile muscle fibers can maintain the urethral pressure and tension by continual contraction [26]. When abdominal pressure rises, fast-contracting muscle fibers squeeze the urethra toward the posterior wall to stop urine flow and maintain urethral pressure. Frauscher et al. ultra sounded normal, and SUI women's external urethral sphincters and SUI patients had weak external urethral sphincters. The congenital urethral sphincter disorders are chiefly caused by central nervous system anomalies, while acquired ones are caused by delivery, surgery, and radiation therapy. [27]. The histology and architecture of a female bladder are shown in Fig. 2.

b) Lower Urethral Wall Elasticity

Normal female urethras are soft and tubular. The collagen, elastic fiber, loose connective tissue, and other components give the urethral wall extraordinary elasticity and flexibility. It efficiently deforms and closes the urethra in response to external stimuli, ensuring tight closure [28]. Zinner et al. showed in the mechanical model that lubricating the lumen surface improves pipe wall elasticity and flow resistance [29]. Thus, if the urethral wall is elastic, mucus in the lumen will increase the flow resistance. The urethral wall becomes hard and loses suppleness after repeated radiation therapy or surgery and can lessen the collagen and urethral elasticity. This results in decrease of urethral closure capacity and can lead to UI. Similarly, the fall in estrogen levels during post menopause causes urethral closure function problems and decreased collagen and elastic fiber synthesis in the urethral wall, which can lead to UI.

d) Reduced Functional Urethra Length

A correct urethral length is the most crucial part of female continence. The thickest part of the urethra is the middle urethral sphincter, which controls urine continence. The tension-free middle urethral suspension for SUI treatment improves urine continence and ensures the urethra's functional length when abdominal pressure rises. Pelsang et al. found that SUI patients' abdominal pressure causes the bladder neck and the proximal urethra to funnel downward [30]. This can reduce the functioning of urethra's relative length and hydrostatic pressure, which

might cause urethral resistance and UI. Its also detected that the fluid resistance increases with lumen effective length, and in turn can improve urine continence and urethral resistance.

5.2 Anatomical Factors Impacting the Urethra

Stress UI, is frequently attributed to compromised pelvic support structures. A significant factor is the impaired support of the bladder neck. The bladder neck typically resides above the pubococcygeal line, preserving a narrow posterior vesicourethral angle (90°–110°) to guarantee continence. Enhorning's theory predicts that a uniform pressure distribution between the bladder and urethra can sustain urine control. When bladder neck support diminishes, this angle either flattens or vanishes, leading to less pressure on the urethra and consequent avoidance of leakage, particularly during physical exercise [31]. The anterior vaginal wall provides support to the middle and lower urethra. When this wall deteriorates or prolapses, it inadequately supports the urethra during elevated abdominal pressure, resulting in UI. Likewise, disturbance of the pelvic fascia or the ATFP, a structural "hammock," can compromise the stability of the bladder, vagina, and urethra, hence complicating the maintenance of continence. Inadequate pubourethral ligaments, which anchor the urethra to the pubic bone, further exacerbate instability. Injury to these ligaments, as demonstrated in animal models, results in UI during elevations in abdominal pressure. Ultimately, failure of the levator ani muscle, particularly as a result of childbirth-related injury, can compromise the urethral closure mechanism [32]. This muscle group provides both sustained (type I fibers) and rapid (type II fibers) assistance, which is crucial for continence. Damage or weakening of the "hammock" support system increases the likelihood of postpartum or stress UI [33]. A remedy could be pelvic floor training and electrical stimulation, which are prevalent non-invasive therapies aimed at this muscle area.

5.3 Pelvic Floor Nerve Anatomical Factors

The female urinary continence is sustained by a complicated neuromuscular system that comprises sympathetic, parasympathetic, and somatic nerves. Parasympathetic fibers (S2–S4) modulate bladder detrusor contraction and urethral relaxation through acetylcholine (ACh) [34]. The sympathetic nerves (T10–L2) secrete norepinephrine (NE), stimulating receptors that constrict the bladder neck and urethra while inducing relaxation of the detrusor muscle. The pudendal nerve (S2–S4) regulates the external urethral sphincter via acetylcholine, facilitating voluntary control of urine. Damage to any of these nerves can disrupt normal urine function [35]. This injury frequently arises from external mechanical force, especially during parturition. Prolonged pressure and tension on pelvic nerves during the second stage of labor may result in hypoxia and nerve damage. Inadequately executed episiotomies may also damage the pudendal branches. Subsequent to nerve damage, neuromuscular alterations encompass diminished nerve fiber density, delayed nerve conduction, and prolonged action potentials. The affected muscles, including the urethral sphincter and levator ani, experience atrophy, changes in fiber type, and diminished contractility. These alterations compromise the urethral closure mechanism, particularly under heightened abdominal pressure, leading to UI. Comprehending the functions of sympathetic, parasympathetic, and somatic pathways is essential for diagnosing and treating UI associated with pelvic floor neuromuscular dysfunction. Nerves regulating the bladder and urethral sphincters are depicted in Fig.3.

5.4 Mechanism leading to incontinence

The bladder, urethra, and urinary sphincters combine to retain urine at low pressure and facilitate voluntary discharge. The detrusor muscle and internal sphincter are composed of smooth muscle, whereas the external sphincter and pelvic floor contain striated muscle. The urothelium protects the underlying tissue and facilitates communication with the brain. Sympathetic nerves regulate urine retention, whereas parasympathetic nerves (via S2–S4) control urination. The typical voiding volume ranges from 250 to 300 ml, with a maximum of seven voids each day. Despite anatomical understanding, the etiology of urgency incontinence remains ambiguous, encompassing alterations in muscle tone, neural regulation, urothelial functionality, supportive structures, and urine composition.

6. RISK FACTORS

6.1 Gender

Women experience UI at a higher rate than men do. One important aspect is the size of the urethra, which is shorter in women than in men. Urine leakage is, therefore, less likely to occur. Experiences such as pregnancy and childbirth can weaken the muscles supporting the bladder in women, increasing the risk of incontinence. Additionally, vaginal delivery may harm pelvic muscles or nerves, raise the risk of stress and urge incontinence. Prostate enlargement, prostate cancer, and therapies like radical prostatectomy (removal of the prostate) and radiotherapy (a form of cancer treatment) for prostate cancer are the most common risk factors for urine incontinence in men. In both sexes, UTIs can increase the risk of developing UI.

6.2 Age

People are more likely to experience UI as they age. This occurs as a result of the bladder's surrounding muscles gradually weakening. Similarly, age-related hormonal changes, especially during menopause, are particularly noticeable in women. Hormone levels, particularly those of estrogen, undergo major changes throughout menopause. During this stage, a reduction in estrogen may have detrimental effects on the bladder lining and increase the risk of incontinence.

6.3 Weight

According to studies, being overweight or obese dramatically raises the risk of incontinence, especially for women in their early and middle years. Both metabolic

health and the impact of being overweight on weakening the pelvic floor are concerns.

6.4 Smoking and Family History

Cigarette smoking is linked to a higher incidence of overactive bladder (OAB) and urine incontinence. Smoking can cause coughing spasms that cause or exacerbate stress incontinence and bladder leaks, as well as irritate the bladder's lining and cause urge incontinence. UI is more likely to occur in people with a family history of the condition.

7.0 DIAGNOSIS

7.1 Clinical Assessment

To properly determine the underlying reason or kind of incontinence, the doctor must do a more comprehensive history after diagnosing UI. An evaluation and elimination of the reversible causes of urine incontinence should be part of a patient's relevant history. Resnick developed the DIAPPERS mnemonic, which makes it simple to recall the common reversible causes of incontinence, such as: D: Delirium I: Infection A: Atrophy P: Pharmaceuticals, these include skeletal muscle relaxants, opioids, COX 2 selective NSAIDS, ACE inhibitors, calcium channel blockers, diuretics, alpha-adrenergic antagonists, alcohol, antidepressants, antipsychotics, antihistamines, anticholinergics, and thiazolidinediones [36,37] P: Psychological disorders, such as depression E: Excessive output of urine caused by taking an excessive number of fluids, taking too many medications, or having long-term illnesses such diabetes R: Reduced mobility S: Stool impaction [38]. The process of identifying the type of incontinence may be made simpler using standardized questionnaires demonstrating the sensitivity of stress incontinence and urge incontinence [39]. Also, the impact of medical and surgical histories needs to be evaluated. Cough-triggered incontinence due to COPD may also be examined by cough stress test in addition to a comprehensive history [40]. To screen out contributory reasons, a gynecologic or prostate examination should be part of the physical examination [41,42]. The diagnosis of stress incontinence is confirmed by a positive test, which is defined as leaking 5 to 15 seconds after coughing [43]. Laboratory tests are not usually recommended unless a specific cause, such as diabetes-related polyuria, is suggested by the history or physical examination. To identify the trends or triggers linked to a certain type of incontinence, patients may be recommended to avoid milk or dairy [44, 45]. While it is not necessary to perform imaging for every patient, a postvoid residual might help confirm the diagnosis if the physician detects overflow incontinence. After the patient has emptied their bladder, the amount of urine left in the bladder can be measured via intermittent bladder catheterization or ultrasound to determine the postvoid residual volume. A postvoid residual volume of less than 50 mL excludes overflow incontinence as a contributing factor to a patient's symptoms, while a residual volume of more than 200 mL is diagnostic of overflow incontinence.

7.2 Urological tests

Objective and semi-objective testing, including urinalysis, pad tests, urodynamic tests, and voiding diaries, may also be necessary for the diagnosis.

Urinalysis: Urinalysis employing colorimetric reagent test strips evaluates parameters including pH, protein, glucose, ketones, blood, bilirubin, urobilinogen, nitrite, leukocyte esterase, and specific gravity [46]. Urinalysis is advised in the evaluation of UI to detect UTIs and associated disorders and is performed by dipstick assay. Dipsticks can identify hematuria, glycosuria, and proteinuria, facilitating the screening for cancer, diabetes, or renal disorders. [47]. Positive dipstick findings should undergo mandatory microscopy, culture, and sensitivity (MCS) testing. Diagnostic thresholds comprise >100,000 CFU/ml in voided samples or >1,000 CFU/ml in catheterized specimens, both accompanied by pyuria. Reduced CFU counts may still be relevant in refractory lower urinary tract symptoms (LUTS). Asymptomatic bacteriuria is prevalent among the elderly, necessitating careful interpretation of urine data to prevent unwarranted antibiotic administration. Per the revised McGeer criteria, the diagnosis of symptomatic UTI necessitates a positive culture alongside localized genitourinary symptoms in patients devoid of indwelling catheters [48].

Post-void residual (PVR) volume assessment is recommended in patients presenting with bladder over-distension, pelvic organ prolapse, or voiding symptoms. PVR can be measured using in-out catheterization, ultrasound bladder scanners, or renal tract imaging [50]. While catheterization remains the gold standard, bladder scanning offers good sensitivity and specificity with improved patient comfort and fewer complications. A PVR of >30 mL immediately post-void is considered elevated; however, when measured after 10 minutes, up to 100 mL may be acceptable. Repeat testing is advised for borderline values ≥100 mL due to variability in clinical significance.

Although high PVR is not consistently linked to worsening storage symptoms or UI, it can be associated with recurrent UTIs and may influence treatment options, especially with interventions like onabotulinumtoxin A or stress incontinence surgery, which can impair voiding. Simple micturition charts, frequency–volume diaries, and comprehensive bladder diaries are particularly helpful in evaluating polyuria and nocturia.

The treatment outcomes are measured by Pad testing and are quantified by urine loss using perineal pads worn during daily activities. A weight gain of >1 g in 1 hour or >4 g in 24 hours indicates positive incontinence [51]. Pelvic floor imaging (via ultrasound or radiologic techniques) evaluates structural and functional abnormalities and supports clinical findings [52].

8.0 MANAGEMENT

8.1 Comprehensive Management Strategy for Female UI

The treatment strategy for adult female UI is inherently staged and customized to individual requirements and health conditions. For many otherwise healthy women, conservative approaches such as behavioral modification, fluid intake optimization, pelvic floor rehabilitation, medication, and, when warranted, surgical intervention are systematically investigated. Conversely, patients with substantial comorbidities may emphasize symptom alleviation over total remission, aiming to sustain dependent continence by assisted toileting or containment devices, rather than pursuing independent continence [53].

8.2 Multidisciplinary Care

Effective UI management significantly benefits from a multidisciplinary approach that incorporates physiotherapy, occupational therapy, urology, gynecology, and geriatrics [54-58]. Although the data does not yet endorse any one profession, collaborative expertise regularly correlates with enhanced patient outcomes.

8.3 Non-invasive Procedures

Lifestyle and behavioral therapies constitute the cornerstone of primary management. Modifications include weight reduction, bladder training, scheduled voiding, and customized hydration and caffeine consumption, which can provide significant symptom alleviation, particularly in instances with mixed UI. For overweight individuals, even minor weight reduction (3–5% of baseline) has demonstrated considerable decreases in both stress and urgency incontinence symptoms [59]. Pelvic floor muscle training (PFMT) is highly recommended as the primary treatment for stress, urgency, or mixed UI. Supervised high-intensity regimens are more efficacious than self-directed programs; nevertheless, home-based or electronic training methods may serve as cost-effective alternatives [60-64]. Behavioral therapies, including bladder retraining and prompted voiding, are crucial, particularly for those with cognitive or physical impairments. Adjunctive therapies such as electrical stimulation (ES) and percutaneous tibial nerve stimulation (PTNS) may be contemplated when traditional pelvic floor muscle training (PFMT) is difficult [65]. Electrical stimulation improves pelvic muscle activity and may alleviate overactive bladder symptoms, while the quality of evidence is inconsistent [66-67]. PTNS, a minimally invasive neuromodulatory procedure, is beneficial for urgency and overactive bladder symptoms, with acceptable safety and acceptability characteristics [68].

8.4 Pharmacological Intervention

Antimuscarinic drugs are the primary treatment for urgency UI, providing considerable symptom alleviation; however, they are limited by anticholinergic side effects and issues with patient adherence [69]. Mirabegron, a β3-adrenergic agonist, offers similar efficacy while exhibiting reduced anticholinergic side effects and enhanced tolerance (Table 1). Combination therapy, antimuscarinic and mirabegron, can further improve outcomes. Duloxetine, a serotonin-norepinephrine reuptake inhibitor (SNRI), may be indicated for stress incontinence; however, its administration necessitates careful consideration due to its side-effect profile [70-74]. The role of neurotransmitters in activating urothelial cells is depicted in Fig.4.

8.5 Surgical and Advanced Interventions

Women with recurrent or stress-predominant UI may find surgical interventions advantageous. Mid-urethral slings, autologous fascial slings, and colpo suspension techniques provide cure rates of 80% and are considered primary surgical options for simple cases [75-79]. However, the risks associated with mesh require thorough informed consent and evaluation of alternatives. In cases of urgent UI refractory to conservative and pharmacologic treatments, third-line interventions such as intravesical onabotulinumtoxin A injections and neuromodulation, either sacral (SNM) or tibial (PTNS), are recommended [80]. Both have shown effectiveness in decreasing incontinence episodes and enhancing quality of life; however, adherence to a structured therapeutic sequence and appropriate patient selection are essential.

8.6 Prospective avenues in Incontinence Research and Prevention

Recent research indicates that we may be approaching substantial advancements in the comprehension and prevention of urine incontinence, particularly in the domains of primary prevention and continence physiology [81]. Although conventional ideas have examined myogenic, neurogenic, and urothelial dysfunction as contributors to bladder disorders, the focus on detrusor overactivity has limited therapeutic advancements [82]. The precise function of detrusor overactivity in urgency incontinence is ambiguous, and its significance as a therapeutic goal is becoming scrutinized, as it frequently does not forecast outcomes of treatment. Novel biomarkers, biochemical (e.g., nerve growth factor) and imaging-based (e.g., bladder wall thickness) have not yet exhibited adequate reliability for therapeutic application [83]. Conversely, patient-reported outcomes, such as symptom severity and quality of life, are the most significant indications. Genetic research is uncovering variations linked to incontinence, providing insights into molecular subgroups and possible targeted therapies [84]. There is an increasing interest in prevention via microbiome modification and vaccinations aimed at uropathogens. Recognizing women at elevated risk, such as those with a history of childhood incontinence or traumatic vaginal births, enables timely management [85]. Studies such as URCHOICE are investigating individualized risk assessments during pregnancy [86-89]. However, balancing the dangers associated with caesarean sections and their potential advantages for incontinence prevention is complex and requires additional evidence from long-term, interventional research.

CONCLUSION

UI is a multifaceted illness that considerably diminishes

quality of life among multiple populations. Despite its widespread occurrence and related comorbidities, UI is inadequately recognized and treated, largely due to societal stigma and insufficient public and clinical knowledge. Progress in comprehending the pathophysiology of UI, together with enhancements in diagnostic techniques and treatment approaches, presents new opportunities for improved patient outcomes. A comprehensive, patient-focused strategy that incorporates pelvic floor therapy, pharmacological treatments, surgical procedures, and lifestyle changes emphasizes the significance of tailored multidisciplinary care. Emerging disciplines, including precision medicine, genetics, and microbiome research, offer significant opportunities for the formulation of preventive strategies and targeted medicines. To attain significant advancements in the management of UI, specifically fecal incontinence, three pivotal components are necessary: continuous research funding, extensive public education, and proactive de-stigmatization initiatives. Cooperative involvement of healthcare professionals, governments, and communities is essential for alleviating the societal and economic impact of incontinence. Such collaborations enable individuals to reclaim autonomy and enhance their overall health-related quality of life.

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The authors declare that no ethical approval was required for the study.

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Ethical standards

The manuscript does not contain clinical studies or patient data.

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List of Figures

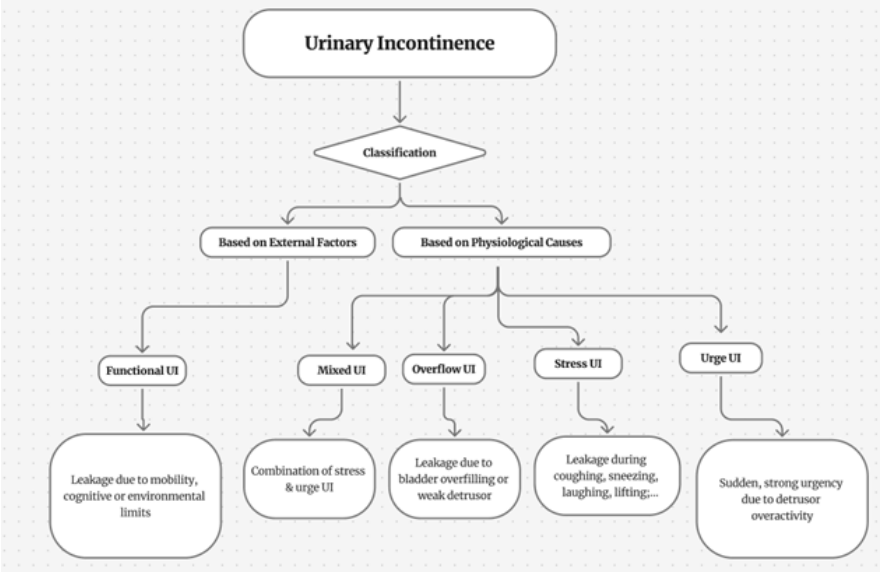


Fig. 1 A UI classification constructed on external and physiological factors

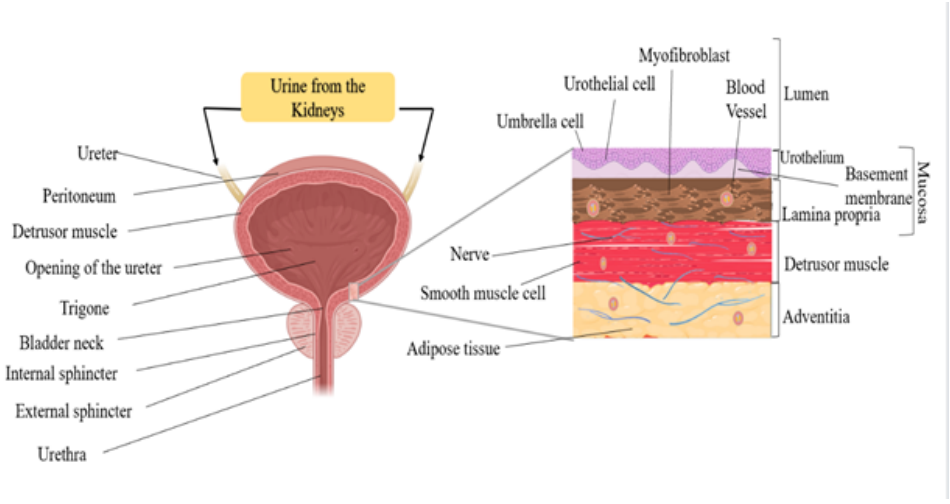


Fig. 2 Female bladder histology and architecture

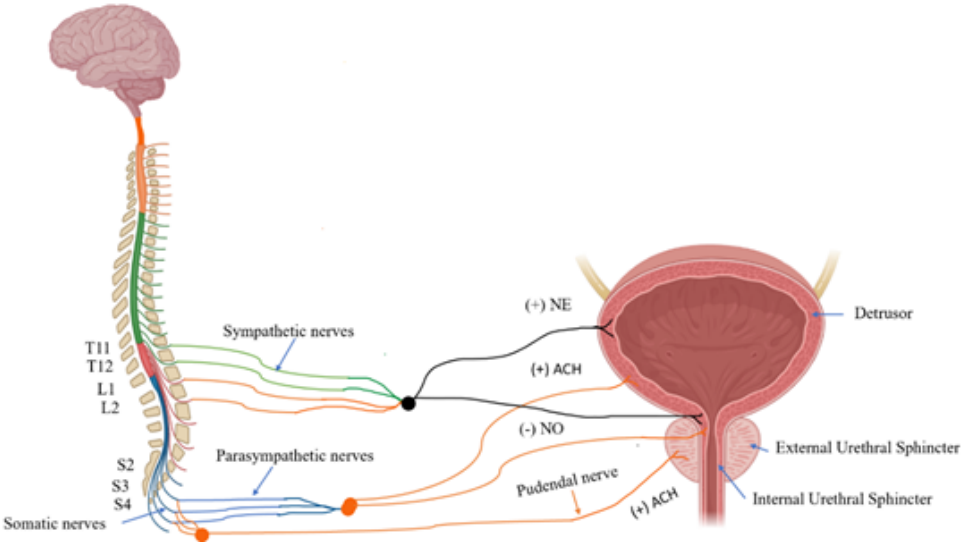


Figure. 3 Neural control of micturition, showing sympathetic (T11-L2), parasympathetic (S2-S4), and somatic nerves regulating the bladder and urethral sphincters.

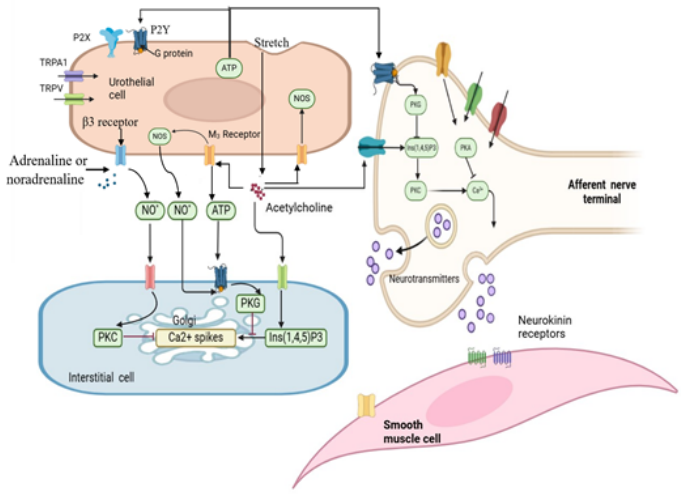


Fig. 4 Uroepithelial sensory web. The urotheliogenic theory of overactive bladder syndrome (BOX 1) suggests that neurotransmitters activate urothelial cells, signals from other cells, and autocrine or paracrine pathways

List of Tables

Table 1 Drugs licensed for UI[195-196]

Class	Medications	Formulation type and dose	Common adverse effects
Non-selective antimuscarinic	• Fesoterodine fumarate • Oxybutynin chloride • Tolterodine tartrate • Trospium chloride	• Tablet / 4-8 mg once a day • Tablet, syrup, transdermal gel/5-8 mg orally 2-3 times a day 10% dose for transdermal gel • Capsule, tablet/ 2-4 mg once a day • Capsule, tablet/ 40-60 mg once a day	Dry mouth, Blurred vision Constipation Impaired cognition Impaired memory

Selective M3 antimuscarinic	• Darifenacin • Imidafenacin • Solifenacin succinate	• Tablet / 7.5- 15 mg once a day • Tablet / 0.1 mg once a day • Tablet / 5-10 mg once a day	Dry mouth Dry eyes Constipation Blurred vision
β3-adrenergic agonist	• Mirabegron • Vibegron	• Tablet / 25-50 mg once a day • Tablet / 75 mg once a day	Headache Dizziness Dry mouth Hypertension