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Case Report

Nocturnal Enuresis in a Postmenopausal Woman: A Case Report and Review of Literature

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Abstract

Background: Nocturnal enuresis (NE) is a rare condition in adults, particularly in postmenopausal women. It is often linked to multiple factors including hormonal changes, comorbidities, and sleep disorders. Hormonal deficiency, particularly estrogen, has been implicated in the pathophysiology of lower urinary tract symptoms (LUTS) in postmenopausal women, contributing to bladder dysfunction and urethral instability. This case report presents a postmenopausal woman with NE, highlighting the importance of a thorough diagnostic approach, management strategies, and the underlying pathophysiological mechanisms.

Case Presentation: A 53-year-old multiparous woman, three years post menopause, presented with a two-week history of NE, experiencing 3-4 episodes of bedwetting per week. Urinalysis revealed no abnormalities, and hormonal profiling confirmed hypoestrogenism (FSH: 72 IU/L, estradiol: <20 pg/mL). MRI of the brain and polysomnography were recommended to exclude neurological issues and obstructive sleep apnea (OSA). The management plan included oral desmopressin (0.2 mg twice daily) and behavioral modifications.

Conclusion: Postmenopausal NE is associated with hormonal, neurological, and sleep-related factors. Local estrogen therapy and targeted investigations, such as sleep studies, are crucial for effective management. This case highlights the importance of a multidisciplinary approach integrating hormonal therapy, pharmacotherapy, and lifestyle adjustments. Further research is needed to establish evidence-based guidelines for managing NE in postmenopausal women.

Keywords: Nocturnal enuresis, Post menopause, Estrogen decline, Bladder dysfunction, Hormonal therapy, Case report

INTRODUCTION

Nocturnal enuresis (NE), defined as involuntary urination during sleep, is a well-documented condition in children but remains underrecognized and poorly characterized in adults, particularly postmenopausal women [1]. While childhood NE often resolves with age, adult-onset NE is typically secondary to underlying medical, hormonal, or neurological pathologies [2]. In postmenopausal women, the condition poses unique diagnostic and therapeutic challenges due to the interplay of aging, hormonal shifts, and comorbidities [3].

The decline in estrogen levels during menopause is a pivotal contributor to lower urinary tract dysfunction. Estrogen plays a critical role in maintaining the structural and functional integrity of the bladder, urethral mucosa, and pelvic floor muscles [4]. Its deficiency leads to urogenital atrophy, reduced collagen elasticity, and diminished blood flow to the urethral sphincter, resulting in decreased urethral closure pressure and detrusor muscle instability [5-7]. These changes predispose postmenopausal women to urinary incontinence, urgency, and, in rare cases, NE. Emerging evidence suggests that estrogen deficiency may also impair nocturnal antidiuretic hormone (ADH) secretion, exacerbating nighttime polyuria [8].

Beyond hormonal factors, adult NE is multifactorial, often linked to neurological disorders, obstructive sleep apnea (OSA), and obesity. OSA, prevalent in postmenopausal populations, contributes to NE through recurrent hypoxia-induced diuresis and increased intra-abdominal pressure during apneic episodes, which strain the bladder and urethra [9]. Obesity further compounds this risk by elevating intravesical pressure and promoting pelvic floor laxity. A large cohort study of 161,808 postmenopausal women found that 1.7% reported NE, with obesity (OR 2.29) and OSA-related symptoms (OR 2.01) being significant predictors [10].

Despite these associations, NE in postmenopausal women remains understudied, leading to gaps in clinical guidelines. Current management strategies often extrapolate from

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pediatric or general adult populations, overlooking the unique hormonal and physiological context of menopause [11]. This case report underscores the necessity of a tailored, multidisciplinary approach to NE in postmenopausal women, integrating hormonal therapy, neurological evaluation, and sleep disorder screening. By elucidating the complex etiology of this condition, clinicians can better address its profound impact on quality of life and mitigate associated stigma.

CASE REPORT

Patient History and Presentation

A 53-year-old multiparous woman presented to the medical clinic with a two-week history of nocturnal enuresis, reporting 3-4 involuntary bedwetting episodes weekly. The patient specifically denied accompanying urinary symptoms including urgency, dysuria, hematuria, or vaginal discharge. Her medical history was notable only for natural menopause at age 50 without hormone replacement therapy, and her obstetric history included three uncomplicated vaginal deliveries without documented pelvic floor trauma or dysfunction.

Physical Examination and Initial Investigations

Initial evaluation revealed a well-nourished woman with stable vital signs. Physical examination demonstrated no abdominal tenderness, masses, or organomegaly. Pelvic examination showed mild urogenital atrophy characterized by vaginal mucosal thinning and reduced rugae, but no evidence of pelvic organ prolapses or stress urinary incontinence. Initial laboratory workup with urinalysis was unremarkable (specific gravity 1.010, absent glucose/protein/leukocytes), and urine culture showed no bacterial growth.

Diagnostic Workup

The diagnostic workup was expanded given her postmenopausal status and absence of structural or infectious etiologies. Endocrine evaluation confirmed hypoestrogenism with markedly elevated FSH (72 IU/L) and suppressed estradiol (<20 pg/mL). Notably, serum desmopressin (ADH) levels were low at 0.5 pg/mL (normal nocturnal range 1-5 pg/mL), indicating deficient nighttime antidiuretic hormone secretion. Neuroimaging with brain MRI revealed no structural abnormalities, with particular attention to the normal appearance of the pituitary gland. Polysomnography was recommended to evaluate for potential obstructive sleep apnea as a contributing factor to nocturnal polyuria.

Treatment and Follow-Up

Treatment was initiated with oral desmopressin 0.2 mg twice daily to address the hormonal deficiency, supplemented by behavioral modifications including evening fluid restriction and scheduled pre-sleep voiding. At her four-week follow-up visit, the patient reported significant clinical improvement with only a single mild enuresis episode occurring during the preceding two weeks.

LITERATURE REVIEW

Nocturnal enuresis (NE) in postmenopausal women is a multifactorial condition influenced by hormonal, anatomical, and physiological changes associated with aging and menopause [12]. Estrogen deficiency, a hallmark of menopause, plays a central role in the pathogenesis of lower urinary tract symptoms (LUTS), including NE. Estrogen receptors are densely distributed in the bladder trigone, urethral mucosa, and pelvic floor muscles, where estrogen modulates collagen synthesis, vascular perfusion, and neuromuscular function [13]. Postmenopausal hypoestrogenism leads to urogenital atrophy, characterized by thinning of the urethral epithelium, reduced submucosal vascularity, and diminished collagen support for the bladder neck and urethra. These changes impair urethral closure pressure, increasing susceptibility to urinary incontinence and nocturnal leakage [14]. Additionally, estrogen deficiency may disrupt the circadian secretion of antidiuretic hormone (ADH), reducing nocturnal water reabsorption and contributing to polyuria, a key driver of NE [15,16].

Therapeutic strategies targeting estrogen deficiency have shown variable efficacy. Local estrogen therapy, such as vaginal estriol cream, directly restores urethral mucosal integrity and enhances urethral closure pressure. A meta-analysis by Mishra [7] demonstrated that localized estrogen therapy significantly improves urinary incontinence symptoms (OR = 1.45), whereas systemic hormone replacement therapy (HRT) is less effective (OR = 0.74). Systemic HRT's limited benefit may stem from its association with increased collagenase activity, which exacerbates pelvic floor laxity, or its negligible impact on urethral vascularity compared to topical applications [17]. These findings underscore the importance of route-specific estrogen therapy in managing postmenopausal LUTS.

Beyond hormonal factors, sleep-disordered breathing, particularly obstructive sleep apnea (OSA), has emerged as a critical contributor to NE in postmenopausal women. OSA is prevalent in this population due to age-related weight gain, reduced upper airway muscle tone, and hormonal shifts. Apneic episodes during OSA induce recurrent hypoxia and hypercapnia, triggering sympathetic activation and atrial natriuretic peptide (ANP) release. ANP promotes diuresis, leading to nocturnal polyuria, while intermittent hypoxia directly stimulates renal tubular sodium and water excretion [18]. Furthermore, OSA-related intra-abdominal pressure fluctuations during snoring or apneic events strain the bladder and urethra, exacerbating urinary leakage. A cohort study of 161,808 postmenopausal women identified OSA as an independent predictor of NE, with each additional OSA risk factor (e.g., snoring, obesity) increasing the odds of NE by 30-50% [19].

Obesity, a common comorbidity in postmenopausal women, compounds NE risk through multiple mechanisms. Excess adiposity elevates intravesical pressure, promotes pelvic floor laxity, and exacerbates OSA severity. Adipose tissue also secretes inflammatory cytokines, which may impair detrusor muscle function and bladder sensation. A dose-response relationship exists between body mass index (BMI) and NE prevalence, with obese women (BMI >30) facing a 2.3-fold higher risk than those with normal BMI [20].

Neurological and cognitive factors further complicate NE in older adults. Age-related neurodegeneration, cerebrovascular disease, or diabetic neuropathy may disrupt supraspinal inhibitory pathways governing bladder control, leading to detrusor overactivity or impaired arousal mechanisms. Cognitive decline, prevalent in elderly populations, can impair recognition of bladder fullness or delay waking responses to urinary urgency [21].

DISCUSSION

The management of nocturnal enuresis (NE) in postmenopausal women requires a nuanced understanding of its multifactorial etiology. In this case, localized hormone replacement therapy (HRT) was prioritized due to its targeted efficacy in addressing estrogen-deficient urogenital atrophy, a key contributor to bladder dysfunction. Systemic estrogen therapy, while beneficial for vasomotor symptoms, has shown limited effectiveness in improving urinary incontinence compared to local applications such as vaginal estriol [22]. Localized HRT enhances urethral mucosal integrity and collagen support, restoring urethral closure pressure without the systemic risks associated with oral estrogen, such as thromboembolism or breast cancer [23]. This approach aligns with clinical guidelines advocating for route-specific estrogen therapy in postmenopausal lower urinary tract symptoms (LUTS).

Neurological evaluation, particularly MRI of the brain, was integral to excluding silent pathologies such as cerebrovascular infarcts or demyelinating lesions, which may impair supraspinal bladder control mechanisms. MRI's superior soft tissue resolution makes it preferable to CT for detecting subtle neurological abnormalities, especially in patients without overt neurological deficits [24]. This step is critical in adult-onset NE, where undiagnosed neurological conditions could underlie sudden symptom onset. For instance, silent infarcts in the prefrontal cortex or pontine micturition centers may disrupt inhibitory pathways governing bladder continence, necessitating tailored interventions [25].

Lifestyle modifications, including evening fluid restriction and scheduled voiding, were advised to mitigate nocturnal polyuria and reduce bladder burden. Behavioral strategies are particularly relevant in postmenopausal women, as aging blunts the circadian rhythm of antidiuretic hormone (ADH), exacerbating nighttime urine production [26]. Obesity and obstructive sleep apnea (OSA), common in this population, further compound NE risk by increasing intra-abdominal pressure and promoting hypoxia-induced diuresis [27]. Weight loss and OSA management via continuous positive airway pressure (CPAP) may synergize with hormonal therapy to alleviate enuresis, though their efficacy requires validation in targeted studies.

A multidisciplinary approach is essential for optimizing outcomes. While initial management focused on HRT and behavioral changes, long-term follow-up must incorporate polysomnography results to address OSA and consider anticholinergics or beta-3 agonists if detrusor overactivity persists. The interplay of hormonal, neurological, and lifestyle factors underscores the need for personalized care. Future research should explore the longitudinal benefits of combining localized HRT with CPAP or pharmacotherapy, as well as the role of cognitive-behavioral interventions in improving adherence to lifestyle modifications.

CONCLUSION

Postmenopausal nocturnal enuresis is a complex condition necessitating holistic evaluation and individualized management. Hormonal therapy, neurological assessment, and lifestyle adjustments form the cornerstone of treatment, but long-term follow-up is critical to address evolving contributors such as OSA or neurogenic bladder dysfunction. Clinicians must remain vigilant to the multifactorial nature of NE, integrating emerging evidence on targeted therapies while prioritizing patient-Centered care. Further research is warranted to establish standardized protocols and improve quality of life for this understudied population.

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