# Original Research Paper



#### HINMAN SYNDROME: A NARRATIVE REVIEW

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ABSTRACT

Hinman syndrome, or non-neurogenic neurogenic bladder, is a complex voiding disorder characterized by functional bladder-sphincter dyssynergia without underlying neurological pathology. This narrative review examines its pathophysiology, clinical presentation, diagnostic approach, and management strategies. Patients typically present with urinary retention, recurrent UTIs, encopresis, and upper tract complications. Diagnosis requires exclusion of neurological causes through imaging and urodynamic studies demonstrating detrusor-sphincter dyssynergia. Treatment involves a multidisciplinary approach combining behavioral therapy (timed voiding, biofeedback), pharmacotherapy (anticholinergics, alpha-blockers), and psychological support. Advanced cases may require botulinum toxin injections or surgical intervention. Prognosis depends on early intervention, with untreated cases risking renal impairment. Future research should explore neurobiological correlates and optimize therapeutic algorithms to improve outcomes in this challenging disorder.

**KEYWORDS:** Urinary Bladder, Neurogenic; Urinary Retention; Vesico-Ureteral Reflux; Biofeedback, Psychology; Cholinergic Antagonists.

#### INTRODUCTION

Hinman syndrome, also known as non-neurogenic neurogenic bladder, is a rare voiding disorder characterized by functional bladder outlet obstruction mimicking neurogenic bladder but without underlying neurological pathology. It primarily affects children, presenting with symptoms such as enuresis, urinary retention, recurrent urinary tract infections (UTIs), and encopresis. Radiographic findings often include bladder wall thickening, hydroureteronephrosis, and a "Christmas tree" or trabeculated bladder appearance, yet neurological examinations and MRI results remain normal. The etiology is attributed to acquired behavioral and psychological factors, often linked to abnormal family dynamics or psychosocial stressors (1)(2).

Diagnosis requires exclusion of true neurogenic bladder through intact perineal sensation, normal lumbosacral imaging, and urodynamic studies showing detrusor-sphincter dyssynergia. Treatment is multidisciplinary, combining behavioral therapy (e.g., timed voiding, biofeedback), anticholinergics, and psychological support. Severe cases may require clean intermittent catheterization or surgical interventions like bladder augmentation to prevent renal damage (1)(2).

### Methods

A narrative review of the literature on Hinman syndrome was conducted using four databases: PubMed, Embase, Scopus, and Web of Science. The search strategy included keywords such as "Hinman syndrome," "non-neurogenic neurogenic bladder," "voiding dysfunction," and "pediatric urology," combined with Boolean operators (AND/OR). Articles in English and Spanish were screened without strict time restrictions. After removing duplicates and assessing relevance through title and abstract screening, 15 key references were selected based on their focus on etiology, diagnosis, and management. The synthesis prioritized clinical evidence and expert consensus.

## Pathophysiology and Etiology of Hinman Syndrome

Hinman syndrome, or non-neurogenic neurogenic bladder, is a functional voiding disorder characterized by bladder-sphincter dyssynergia in the absence of neurological pathology. The condition primarily affects children, leading to symptoms such as urinary retention, recurrent UTIs, and overflow incontinence. The pathophysiology is thought to involve learned voiding dysfunction, where abnormal pelvic floor contraction during micturition creates functional

obstruction (3). This behavior is often linked to psychological stressors, toilet-training difficulties, or dysfunctional family dynamics, reinforcing a maladaptive voiding pattern (4). Urodynamic studies typically reveal detrusor overactivity with poor sphincter relaxation, mimicking true neurogenic bladder but with intact sacral reflexes (5).

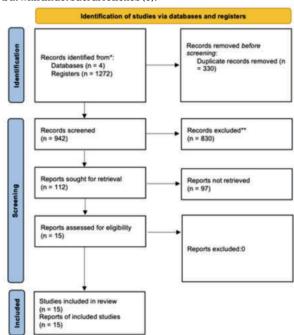


Figure. PRISMA.

The etiology remains multifactorial, with theories suggesting autonomic dysregulation, delayed maturation of pontine micturition centers, or conditioned reflex abnormalities. Some cases may also involve chronic constipation, which exacerbates pelvic floor dysfunction (3)(4). Unlike true neurogenic bladder (e.g., spina bifida), Hinman syndrome shows no structural spinal cord defects on MRI, emphasizing its behavioral origin. Early recognition is crucial to prevent complications such as hydronephrosis, vesicoureteral reflux, and renal scarring (5).

## Clinical Presentation of Hinman Syndrome

Hinman syndrome presents with a complex interplay of urinary and bowel dysfunction stemming from maladaptive

voiding patterns. The urinary manifestations typically include a triad of symptoms: daytime urinary incontinence, difficulty initiating voiding, and recurrent urinary tract infections (5). These symptoms arise from functional bladder outlet obstruction caused by involuntary contraction of the external sphincter during micturition. Approximately 30% of affected children develop vesicoureteral reflux as a secondary complication, potentially leading to more serious renal consequences if left untreated (6).

The bowel dysfunction component, present in 40-60% of cases, primarily manifests as encopresis resulting from paradoxical contraction of the anal sphincter during defecation (7). This dual dysfunction of urinary and bowel control often creates significant distress for both patients and caregivers. In advanced cases, patients may present with flank pain or laboratory evidence of renal insufficiency, reflecting the potential for progressive upper tract damage (8).

Diagnostic imaging reveals characteristic anatomical changes that develop over time. The bladder typically appears trabeculated with thickened walls, often assuming the pathognomonic "Christmas tree" configuration on cystography (5). Upper urinary tract evaluation frequently demonstrates varying degrees of hydroureteronephrosis, with approximately 85% of long-standing cases showing these dilatation changes (6). The most concerning finding, renal cortical scarring, indicates permanent damage and underscores the importance of timely diagnosis and management.

Urodynamic studies remain the gold standard for confirmation, demonstrating detrusor overactivity with simultaneous sphincter contraction during voiding attempts the hallmark finding that differentiates this condition from true neurogenic bladder disorders (7). These findings occur in the setting of normal neurological examination and intact sacral reflexes, highlighting the functional nature of the disorder (8).

#### Diagnosis of Hinman Syndrome

The diagnosis of Hinman syndrome requires a systematic approach that combines clinical evaluation with specialized testing to exclude neurological causes. A comprehensive assessment begins with detailed history-taking focusing on voiding patterns, bowel habits, and any psychological stressors. Physical examination must confirm intact perineal sensation and normal anal tone, as these findings help differentiate the condition from true neurogenic bladder disorders (8). Crucially, lumbar spine MRI must demonstrate normal anatomy to rule out occult spinal dysraphism or other neurological abnormalities that could explain the symptoms (9)

Urodynamic studies represent the cornerstone of diagnostic confirmation, typically revealing detrusor-sphincter dyssynergia (DSD)-the involuntary contraction of the external urethral sphincter during attempted voiding (10). This paradoxical activity creates functional bladder outlet obstruction despite the absence of neurological pathology. Additional urodynamic findings often include elevated postvoid residuals exceeding 200 mL, indicating incomplete bladder emptying (11). The combination of these urodynamic abnormalities with normal neurological imaging and examination findings establishes the diagnosis of Hinman syndrome.

A multidisciplinary approach is essential, involving pediatric urologists, neurologists, and sometimes psychologists to comprehensively evaluate all aspects of this complex condition. The diagnostic process must also assess for common comorbidities such as vesicoureteral reflux and renal scarring through appropriate imaging studies (12). Early and accurate diagnosis is critical to initiate timely interventions

that can prevent long-term renal damage and improve quality of life for affected children.

#### Management Strategies for Hinman Syndrome

The treatment of Hinman syndrome requires a multidisciplinary approach tailored to the severity of symptoms and potential complications. First-line therapies focus on behavioral modifications and pharmacotherapy. Behavioral interventions, including timed voiding schedules (every 2-3 hours) and pelvic floor biofeedback training, help retrain proper voiding mechanics and reduce dysfunctional holding patterns (12). These techniques are particularly effective in motivated children with mild to moderate symptoms. Pharmacological support often includes anticholinergics (oxybutynin) to manage detrusor overactivity and alphablockers (tamsulosin) to decrease bladder outlet resistance, though their use must be carefully monitored for side effects (13).

For refractory cases, more advanced interventions may be necessary. Botulinum toxin injections into the external urethral sphincter can temporarily relieve detrusor-sphincter dyssynergia (DSD), though the duration of effect varies and repeated procedures may be required (14). In severe cases with progressive hydronephrosis, vesicoureteral reflux (VUR), or declining renal function, surgical options such as bladder augmentation or Mitrofanoff catheterizable channels may be considered to protect upper urinary tract function (15).

Psychological support is a critical yet often overlooked component of management. Many children with Hinman syndrome experience anxiety, depression, or family-related stressors that exacerbate symptoms. Early involvement of a pediatric psychologist can address maladaptive behaviors, improve treatment adherence, and enhance overall outcomes (12). A holistic approach combining medical, behavioral, and psychological strategies offers the best chance for long-term success in managing this complex disorder.

#### Prognosis and Complications in Hinman Syndrome

The long-term outlook for Hinman syndrome depends largely on early recognition and intervention. Without proper treatment, persistent high-pressure voiding can lead to progressive renal damage, with some cases advancing to end-stage renal disease (ESRD) (13). While timely management improves outcomes, approximately 50% of severe cases ultimately require surgical intervention to preserve kidney function (14). Common complications include refractory hydronephrosis, recurrent pyelonephritis, and irreversible bladder dysfunction, emphasizing the need for close monitoring.

### CONCLUSION

Hinman syndrome presents complex diagnostic and therapeutic challenges. Optimal care requires a multidisciplinary team—including pediatric urologists, psychologists, and nephrologists—to address both physiological and behavioral aspects (15). Future research should investigate potential neurogenic underpinnings of this functional disorder while refining minimally invasive therapies to improve long-term outcomes.

#### REFERENCES

- Gampala S, Alkukhun L, Khan Z, et al. Hinman Syndrome: A Rare Entity With Neurogenic Bladder-Like Symptoms. Cureus. 2024;16(4):e58191. doi:10.7759/cureus.58191.
- Lee DH, Kim YS, Lee HY, Han SW. Hinman Syndrome: Long-Term Follow-Up of 14 Cases. Korean J Urol. 2007;48(10):1058-1063. doi:10.4111/kju. 2007.48. 10.1058.
- Franco I. Functional voiding disorders in children: Pathophysiology, diagnosis, and treatment. Pediatr Clin North Am. 2012;59(4):783-817. doi:10.1016/j.pcl.2012.05.007.
- Burgers RE, et al. Functional defecation disorders in children: Nonneurogenic versus neurogenic. J Pediatr Gastroenterol Nutr. 2013;56(2):204-211. doi:10.1097/MPG.0b013e318276bdbc.
- . Austin PF, et al. The standardization of terminology of lower urinary tract

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- function in children and adolescents. J Urol. 2014;191(6):1863-1865. doi:10.1016/j.juro.2014.01.110.
- Franco I. Functional voiding disorders in children: Pathophysiology, diagnosis, and treatment. Pediatr Clin North Am. 2012;59(4):783-817. doi:10.1016/j.pcl.2012.05.007.
- Koff SA, Wagner TT, Jayanthi VR. The relationship among dysfunctional elimination syndromes, primary vesicoureteral reflux, and urinary tract infections in children. J Urol. 1998;160(3 Pt 2):1019-1022. doi:10.1097/ 00005392-199809020-00011.
- Burgers RE, et al. Functional defecation disorders in children: Nonneurogenic versus neurogenic. J Pediatr Gastroenterol Nutr. 2013;56(2):204-211. doi:10.1097/MPG.0b013e318276bdbc.
- Austin PF, et al. The standardization of terminology of lower urinary tract function in children and adolescents. J Urol. 2014;191(6):1863-1865. doi:10.1016/j.juro.2014.01.110.
- Herndon CD, et al. The value of magnetic resonance imaging for the evaluation of neurogenic bladder in children. J Urol. 2003;170(4 Pt 1):1345-1348. doi:10.1097/01.ju.0000083730.56161.2d.
- Nørgaard JP, et al. Standardization and definitions in lower urinary tract dysfunction in children. Br J Urol. 1998;81 Suppl 3:1-16. doi:10.1046/j.1464-410x.1998.00003.x.
- Bauer SB. Neurogenic bladder: etiology and assessment. Pediatr Nephrol. 2008;23(4):541-551. doi:10.1007/s00467-008-0764-7.
- Koff SA. Relationship between dysfunctional voiding and reflux. J Urol. 1992;148(5 Pt 2):1703-1705. doi:10.1016/s0022-5347(17)37010-6.
   Steinhardt GF, Naseer S. Botulinum toxin: A novel treatment for dysfunctional
- Steinhardt GF, Naseer S. Botulinum toxin: A novel treatment for dysfunctional voiding in children. Curr Urol Rep. 2003;4(2):158-162. doi:10.1007/s11934-003-0043-x.
- Herndon CD, et al. The Kelly technique of bladder neck reconstruction for the treatment of refractory urinary incontinence. J Urol. 2003;170(4 Pt 2):1610-1613. doi:10.1097/01.ju.0000083730.56161.2d.