Case Report



# Severe Lower Urinary Tract Dysfunction in Otherwise Healthy Children: Report of Three Cases and Review of the Literature

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Posted Date: 15 October 2025

doi: 10.20944/preprints202510.1129.v1

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 doi:10.20944/preprints202510.1129.v1



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# Severe Lower Urinary Tract Dysfunction in Otherwise Healthy Children: Report of Three Cases and Review of the Literature

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## Highlights

**What are the main findings?**

- Severe lower urinary tract dysfunction can occur in neurologically and anatomically normal children, with distinct urodynamic phenotypes (overactivity, dyssynergia, hypo contractility) and cystoscopy remodeling (deep trabeculations, pseudo polypoid mucosa).
- A multimodal, function-first strategy (urotherapy ± biofeedback, targeted pharmacotherapy, and CIC when indicated) achieved infection control and functional recovery in all three cases.

**What is the implication of the main finding?**

- Early phenotyping with noninvasive tests and urodynamics helps prevent unnecessary surgeries (e.g., reimplantation, bulking) prompted by misattributed "structural" causes of rUTIs.
- Systematic identification and treatment of bladder–bowel dysfunction reduces rUTIs, protects the upper urinary tract, and improves long-term outcomes.

## Abstract

**Background:** Severe lower urinary tract dysfunction (LUTD) in neurologically and anatomically normal children is uncommon but clinically important, often presenting with recurrent urinary tract infections (rUTIs), incontinence, or urinary retention. We report three illustrative cases and integrate a structured narrative review to contextualize diagnostic and therapeutic strategies.

**Methods:** Retrospective review of three children (aged 3–10 years) evaluated at a tertiary pediatric urology center (2018–2024) for severe LUTD with rUTIs and no neuroanatomical or structural abnormalities. Assessment included ultrasound, voiding cystourethrography (VCUG), urodynamics, and cystoscopy. Management combined urotherapy, pelvic floor biofeedback, targeted pharmacotherapy, and, when indicated, continuous antibiotic prophylaxis (CAP) or clean intermittent catheterization (CIC). A structured narrative literature search (1970–2024) was undertaken to compare phenotypes and outcomes.

**Results:** Case 1 (7-year-old male) had detrusor overactivity with reduced capacity and cystoscopic remodeling (deep trabeculations, pseudopolypoid mucosa); symptoms resolved on urotherapy + trospium + biofeedback, with relapse after treatment withdrawal. Case 2 (3-year-old female) exhibited poor compliance, detrusor–sphincter dyssynergia, high post-void residuals, and secondary high-grade VUR, consistent with Hinman syndrome; CAP + trospium + CIC + biofeedback led to durable control of rUTIs. Case 3 (10-year-old male) had hypocontractile detrusor with recurrent retention; $\alpha$-blockade (tamsulosin) plus biofeedback achieved symptom resolution. Across cases, endoscopic findings correlated with urodynamic phenotypes, and a function-first multimodal approach was effective.

 doi:10.20944/preprints202510.1129.v1



**Conclusions:** Severe functional LUTD can occur in otherwise healthy children and may mimic structural disease. Early phenotyping with noninvasive testing, urodynamics, and selective cystoscopy, followed by structured conservative therapy, can control infections, restore function, and prevent unnecessary surgery and upper tract damage.

**Keywords:** lower urinary tract dysfunction; Hinman syndrome; pediatric urology; vesicoureteral reflux; dysfunctional voiding; biofeedback; antimuscarinics; clean intermittent catheterization

## 1. Introduction

Lower urinary tract dysfunction (LUTD) encompasses a heterogeneous spectrum of disorders characterized by abnormalities of bladder storage or voiding [1]. While mild or moderate LUTD—such as overactive bladder, dysfunctional voiding, or mild post-void residuals—is relatively common in pediatric populations, severe presentations involving persistently high post-void residual volumes, episodic or chronic urinary retention, intractable incontinence, or secondary upper urinary tract changes remain rare among neurologically intact children [2,3].

Traditionally, severe LUTD in children has been considered a marker of underlying neurogenic bladder, often associated with identifiable structural or developmental anomalies such as spinal dysraphism, myelomeningocele, tethered cord, sacral agenesis, or occult spinal lesions [3,4]. However, an emerging body of literature suggests that a subset of otherwise healthy children—i.e. with no detectable neuroanatomical, systemic, or congenital urinary tract defects—may present with severe lower urinary tract dysfunction [5,6]. The pathophysiology in these cases remains poorly understood and is likely multifactorial. Proposed mechanisms include maladaptive or learned voiding behaviors (such as chronic withholding), psychogenic and psychological stressors, subtle functional immaturities or dysregulation of bladder sensory/afferent pathways, and autonomic dysregulation of detrusor contractility [6,7].

An additional and clinically significant feature in many of these cases is the occurrence of recurrent urinary tract infections (rUTIs). Recurrent UTIs not only reflect impaired bladder emptying and stasis but also pose risks of renal scarring, hypertension, and long-term renal damage, especially in children with underlying voiding dysfunction [8,9].

Indeed, persistent residual urine, incontinence, high bladder pressures, and dysfunctional voiding patterns are recognized risk factors for bacterial colonization and recurrent infections[8,10]. Thus, the coexistence of severe LUTD and rUTIs in children without overt anatomical or neurological abnormalities presents both a diagnostic puzzle and a therapeutic challenge.

In this report, we describe three paediatric cases of severe lower urinary tract dysfunction (LUTD) occurring in otherwise healthy children who presented with recurrent urinary tract infections (UTIs) but had no identifiable neurological or structural urological abnormalities. Through these cases, we aim to illustrate the diagnostic challenges, outline a systematic approach to evaluation, and discuss individualized therapeutic strategies that led to favourable outcomes. We also review the current literature to contextualize these findings within the broader spectrum of functional and non-neurogenic bladder dysfunction in children.

## 2. Methods

We conducted a retrospective review of three pediatric patients evaluated between January 2018 and December 2024 at our tertiary referral pediatric urology center. Each patient presented with severe lower urinary tract dysfunction (LUTD) associated with recurrent urinary tract infections (UTIs), in the absence of any neurological or structural abnormalities. The study was reviewed and approved by the institutional ethics committee, and written informed consent for participation and publication was obtained from the parents or legal guardians of all patients.

 doi:10.20944/preprints202510.1129.v1



Patients were included if they met all of the following: Severe lower urinary tract dysfunction (LUTD) manifesting as: recurrent urinary retention, persistent post-void residual volume >20% of expected bladder capacity for age, or daytime incontinence refractory to initial behavioral therapy; recurrent urinary tract infections (rUTIs), defined as ≥2 febrile UTIs or ≥3 culture-confirmed UTIs within 12 months; absence of identifiable neurological, anatomical, or systemic causes following comprehensive evaluation. Children were excluded if they met any of the following conditions: neurological abnormalities affecting bladder function, including known spinal dysraphism, myelomeningocele, tethered cord, cerebral palsy, or peripheral neuropathies confirmed on clinical examination or spinal MRI; anatomical or structural urinary tract abnormalities such as posterior urethral valves, urethral strictures, ectopic ureter, ureterocele; previous lower urinary tract surgery or interventions (e.g., ureteral reimplantation, bladder augmentation, or endoscopic bulking injection); systemic conditions known to influence voiding behavior, including diabetes mellitus, chronic renal disease, or endocrine disorders affecting urine output; medication use that could alter detrusor or sphincter function (e.g., anticholinergics, $\alpha$-blockers, diuretics) within three months before evaluation (Table 1).

**Table 1.** Inclusion and Exclusion Criteria.

| Category | Criteria | Rationale / Notes |
|---|---|---|
| Inclusion Criteria | - Severe lower urinary tract dysfunction (LUTD) manifested by recurrent urinary retention, post-void residual (PVR) > 20 % of age-expected bladder capacity, or persistent daytime incontinence refractory to initial urotherapy. | Defines clinically significant LUTD requiring tertiary-level evaluation. |
| | - Recurrent urinary tract infections (rUTIs): ≥ 2 febrile or ≥ 3 culture-proven infections within 12 months. | Ensures selection of children with significant morbidity related to LUTD. |
| | - No detectable neurological or structural abnormalities on comprehensive evaluation (clinical exam, imaging, and cystoscopy). | Confirms non-neurogenic, functional etiology. |
| Exclusion Criteria | - Neurological disorders affecting bladder control (spinal dysraphism, myelomeningocele, tethered cord, cerebral palsy, peripheral neuropathies). | Eliminates neurogenic bladder causes. |
| | - Structural urinary tract abnormalities (posterior urethral valves, urethral stricture, ectopic ureter, ureterocele, bladder diverticulum, high-grade VUR ≥ III). | Excludes anatomical obstruction or reflux-related dysfunction. |
| | - Prior lower urinary tract or reconstructive surgery (e.g., ureteral reimplantation, bladder augmentation, endoscopic bulking). | Avoids postoperative or iatrogenic LUTD confounders. |

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| | |
|---|---|
| - Systemic diseases influencing micturition (diabetes mellitus, chronic kidney disease, endocrine or metabolic disorders). | Removes systemic confounders. |
| - Current or recent ( < 3 months ) use of medications affecting detrusor or sphincter function (anticholinergics, $\alpha$-blockers, diuretics). | Prevents pharmacologic bias in urodynamic interpretation. |

ChatGPT (model: GPT-5 Thinking; OpenAI) was used to assist with language drafting and document organization for this manuscript. Specifically, GenAI was employed to draft and refine prose in the Abstract, Highlights, and a structured Literature Review Methods paragraph; suggest in-text citation placement and MDPI-Children–style section headings; and help convert our draft into the MDPI-Children template structure. GenAI also suggested potential open-access references/figures for the authors to verify; all citations and sources were independently checked and curated by the authors. No GenAI tools were used for data collection, extraction from medical records, urodynamic calculations, statistical analysis, clinical decision-making, or interpretation of patient findings. All case data are real and were de-identified by the authors prior to drafting. The authors critically reviewed, edited, and accept full responsibility for all content.

## 3. Case Reports

Case 1

A 7-year-old boy was referred for evaluation of recurrent febrile urinary tract infections (6 episodes in the previous year), persistent daytime incontinence, urgency and enurezis. He had normal perinatal history, developmental milestones, and no prior urinary tract abnormalities. Physical and neurological examinations were unremarkable, with no spinal or perineal anomalies. Ultrasound revealed moderate bladder wall thickening (Figure) and no post-void residual urine (PVR). Voiding cystourethrography (VCUG) demonstrated a crenulated bladder contour, particularly during the voiding phase, and a mildly asymmetric (decentred) bladder configuration. No vesicoureteral reflux was identified. The posterior urethra appeared slightly dilated, yet bladder emptying was complete, with no post-void residual urine. Given the persistence of symptoms and recurrent infections, an exploratory cystoscopy was performed. The bladder was entered without difficulty. The bladder mucosa appeared markedly abnormal, displaying deep trabeculations with multiple cell-like and columnar ridges, giving a pseudo polypoid appearance. Small diverticula and dilated submucosal blood vessels were evident throughout the bladder wall. The extent of these mucosal changes rendered visualization of the ureteric orifices difficult. During withdrawal of the cystoscope, the bladder neck was noted to be high-set, while the verumontanum was normal, and the posterior urethra showed no signs of congenital posterior urethral valves. Urodynamic studies confirmed detrusor overactivity with reduced functional bladder capacity (120 mL vs. expected 200 mL) and uninhibited contractions during the filling phase, supporting a diagnosis of severe functional lower urinary tract dysfunction.

The patient was started on standard urotherapy (timed voiding, adequate hydration, and bowel management) combined with trospium chloride (0.2 mg/kg twice daily) and continuous antibiotic prophylaxis with Nitrofurantoin (50mg once daily). After four weeks, pelvic floor biofeedback training was introduced to improve sphincter coordination.

At six-month follow-up, he achieved full daytime continence with resolution of urgency and no new UTIs. Follow-up urodynamic assessment at 12 months showed improvement of bladder capacity and suppression of detrusor overactivity. Continuous antibiotic prophylaxis was withdrawn after 12 months, while trospium chloride was progressively tapered and discontinued at 24 months. Six months after discontinuation of all treatment, the patient developed a febrile urinary tract infection

 doi:10.20944/preprints202510.1129.v1



and recurrence of enuresis. The infection was treated with appropriate antibiotics, and combination therapy with trospium chloride and biofeedback was reinstated.

Case 2

A 3-year-old girl presented to the Emergency Department (ED) of our tertiary pediatric referral center with fever and pollakiuria. Her history revealed that symptoms had begun two months earlier with dysuria and daytime incontinence, for which a urinary tract infection (UTI) had been diagnosed and treated with antibiotics. Despite appropriate therapy, her symptoms persisted. A renal and bladder ultrasound subsequently revealed left-sided ureterohydronephrosis, with dilatation of the renal pelvis and calyces and a crenulated bladder wall. In view of these findings and ongoing symptoms, the patient was referred to the Emergency Department for further evaluation and management. Voiding cystourethrography (VCUG): The study demonstrated a grade V left-sided vesicoureteral reflux, with a dilated and tortuous ureter, dilated renal pelvis, and convex calyces. Voiding was not achieved during the examination. DMSA renal scintigraphy demonstrated a small, irregularly contoured left kidney with marginal cortical scarring and thinned parenchyma, showing markedly reduced tracer uptake. The differential renal function was 81.3% for the right kidney and 18.7% for the left, indicating significant loss of function on the affected side. In this case, the patient underwent surgical correction with Cohen cross-trigonal ureteral reimplantation, performed through a standard open (transvesical) approach. Despite an initially favourable postoperative evolution, the patient developed recurrent febrile urinary tract infections and episodes of intermittent incontinence during medium- and long-term follow-up. Exploratory cystoscopy demonstrated marked bladder mucosal remodelling, characterized by deep trabeculations and a coarse, cell-like and columnar architecture, rendering identification of the reimplanted ureteric orifices challenging. Following comprehensive evaluation and multidisciplinary case review, the patient was diagnosed with Hinman syndrome, representing a non-neurogenic neurogenic bladder secondary to severe functional voiding dysfunction.

Urodynamic Findings: Urodynamic evaluation was performed after resolution of urinary infection and bladder preparation. The filling cystometry demonstrated reduced bladder compliance with early detrusor overactivity, producing sustained rises in intravesical pressure exceeding 40 cm $H_2O$ during the filling phase. Bladder sensation was present but inconsistent, and the patient reported urgency before reaching expected bladder capacity.

During the voiding phase, pressure–flow analysis revealed simultaneous detrusor contraction and external urethral sphincter activity, consistent with detrusor–sphincter dyssynergia. The uroflowmetry curve exhibited a staccato flow pattern, with low maximum flow rate (Qmax 5 mL/s) and prolonged voiding time. Post-void residual urine was elevated at 120 mL, indicating incomplete bladder emptying despite adequate detrusor effort. These urodynamic findings—poor compliance, detrusor overactivity, sphincter dyssynergia, and high post-void residuals—were highly suggestive of Hinman syndrome (non-neurogenic neurogenic bladder), correlating with the patient's clinical picture of recurrent febrile urinary tract infections and incontinence. The patient was started on a comprehensive treatment regimen that included continuous antibiotic prophylaxis (CAP), trospium chloride, clean intermittent catheterization (CIC), and pelvic floor biofeedback therapy. Clinical evolution was slowly favourable, with progressive improvement in bladder function and complete resolution of recurrent urinary tract infections over time.

Case 3

A 10-year-old boy presented to the Emergency Department (ED) of our tertiary pediatric referral center with acute urinary retention. He had a total of three prior emergency presentations for similar symptoms before being admitted for comprehensive evaluation and further investigations. Renal and bladder ultrasound: The urinary bladder, evaluated in a partially filled state, showed marked wall thickening (up to 7–8 mm) with an irregular inner contour and heterogeneous intravesical content, suggestive of chronic inflammatory changes. Voiding cystourethrography (VCUG): The urinary bladder demonstrated a large capacity with a slightly crenulated contour and uniform opacification.

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A grade I vesicoureteral reflux was noted. Voiding was difficult, intermittent, and incomplete, with a significant post-void residual volume. The urethra appeared normal throughout its course.

Urodynamic studies: The evaluation demonstrated a hypocontractile detrusor, with a maximum urinary flow rate of 4 mL/s and a weak, low-pressure urinary stream. A significant post-void residual volume was recorded, consistent with incomplete bladder emptying.

Exploratory cystoscopy: The bladder was entered without difficulty. The bladder mucosa appeared markedly altered, showing cellular and columnar ridges with pronounced trabeculation and a pseudodiverticular appearance at the trigone. The posterior urethra was normal, with no evidence of anatomical obstruction. The patient was treated with tamsulosin (Omnic-Tocas) and pelvic floor biofeedback therapy, resulting in a gradually favourable clinical evolution and complete resolution of acute urinary retention episodes.

Summary of Patient Characteristics (Figure 1 and Table 2):

The three pediatric patients (two males, one female; aged 3–10 years) presented with severe lower urinary tract dysfunction (LUTD) manifesting as recurrent febrile urinary tract infections, incontinence, or urinary retention, in the absence of neurological or structural abnormalities. Imaging and urodynamic evaluations revealed diverse functional patterns ranging from detrusor overactivity and detrusor–sphincter dyssynergia to hypocontractility, each associated with bladder wall remodeling evident on cystoscopy (trabeculation, pseudopolypoid mucosa, diverticula).

All patients underwent multimodal management, including combinations of urotherapy, anticholinergic or $\alpha$-blocker therapy, biofeedback, and in selected cases continuous antibiotic prophylaxis or clean intermittent catheterization. Clinical outcomes were favorable in all cases, with resolution of infections and improvement in continence or bladder emptying, although one patient (Case 1) experienced recurrence after discontinuation of therapy.

**Table 2.** Summary Table of Cases.

| Characteristic | Case 1 | Case 2 | Case 3 |
|---|---|---|---|
| Age / Sex | 7 years / Male | 3 years / Female | 10 years / Male |
| Presenting Symptoms | Recurrent febrile UTIs, daytime incontinence, urgency, enuresis | Fever, pollakiuria, dysuria, incontinence | Acute urinary retention (recurrent episodes) |
| Prior History | Multiple UTIs over the preceding year | Persistent LUTS after treated UTI | Three prior ED visits for retention |
| Ultrasound Findings | Moderate bladder wall thickening; no residual urine | Left ureterohydronephrosis; crenulated bladder wall | Bladder wall thickened (7–8 mm), irregular contour, heterogeneous content |
| VCUG Findings | Crenulated bladder, no reflux, normal emptying | Grade V left VUR, dilated tortuous ureter, no voiding achieved | Large-capacity bladder, mild crenulation, grade I VUR, incomplete emptying |
| Renal Scintigraphy (DMSA) | – | Left kidney: small, irregular, cortical scarring; function 18.7% | – |

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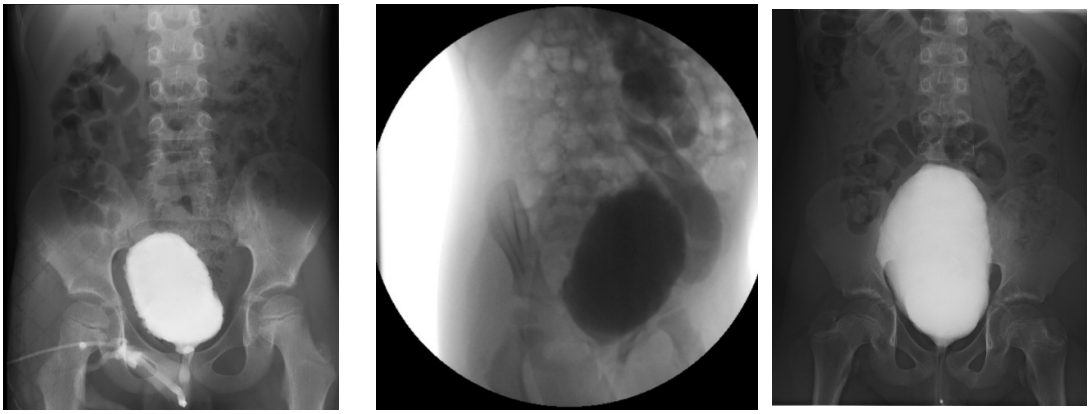
|---|---|---|---|
| Cystoscopy Findings | Deep trabeculations, pseudopolypoid mucosa, diverticula, dilated vessels | Marked trabeculation, cell-like and columnar mucosa, difficult ureteric visualization | Trabeculated, pseudodiverticular bladder mucosa, normal urethra |
| Urodynamic Pattern | Detrusor overactivity, reduced capacity, uninhibited contractions | Poor compliance, detrusor–sphincter dyssynergia, high PVR | Hypocontractile detrusor, weak flow (Qmax 4 mL/s), high PVR |
| Diagnosis | Severe functional LUTD | Hinman syndrome (non-neurogenic neurogenic bladder) | Functional LUTD with hypocontractile bladder |
| Treatment | Urotherapy, trospium chloride, CAP, biofeedback | CAP, trospium chloride, CIC, biofeedback | Tamsulosin (Omnic-Tocas), biofeedback |
| Outcome | Initial remission; recurrence after withdrawal, improved with retreatment | Gradual improvement; resolution of UTIs, stable bladder function | Gradual recovery; resolution of acute retention episodes |



(**A**) (**B**) (**C**)

**Figure 1.** VCUG images.

Representative imaging and endoscopic findings from the three pediatric patients with severe functional LUTD and recurrent urinary tract infections.

- (A) *Case 1:* Voiding cystourethrography showing a crenulated, asymmetric bladder contour without reflux;
- (B) *Case 2:* Voiding cystourethrography demonstrating grade V left vesicoureteral reflux with a tortuous, dilated ureter and dilated pelvis and calyces;
- (C) *Case 3:* Voiding cystourethrography depicting a large-capacity bladder with mild crenulation and grade I reflux, associated with incomplete voiding and high post-void residual;

## 4. Discussion

Severe lower urinary tract dysfunction (LUTD) in neurologically and anatomically normal children remains a diagnostic and therapeutic challenge. While mild functional disorders such as overactive bladder or dysfunctional voiding are common in paediatrics, severe presentations involving high-pressure voiding, recurrent febrile urinary tract infections (UTIs), and bladder wall remodelling are rare. The three cases presented here illustrate distinct phenotypes within the

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spectrum of severe functional LUTD, ranging from detrusor overactivity and detrusor–sphincter dyssynergia to detrusor hypo contractility, all occurring in neurologically intact children without structural obstruction.

*1. Pathophysiologic considerations*

The pathophysiology of severe functional LUTD in such patients remains incompletely understood. Historically, similar presentations have been described under the term Hinman syndrome, or non-neurogenic neurogenic bladder, first characterized by Hinman and Bauman (1973) as a functional voiding disorder in which children develop detrusor–sphincter discoordination and bladder wall changes typical of neurogenic bladder, but without any detectable neurological lesion [11,12]. Chronic maladaptive voiding behaviour—often secondary to voluntary withholding, pain, or psychosocial stressors—can lead to persistently elevated detrusor pressures, incomplete emptying, and bladder remodelling. Over time, the resulting high-pressure bladder dynamics can induce trabeculation, pseudodiverticula, and mucosal vascular congestion, as observed cystoscopically in all three of our patients.

*2. Clinical and Diagnostic Correlation*

In our series, Case 1 presented with severe detrusor overactivity and reduced bladder capacity, leading to recurrent UTIs and incontinence, but with preserved bladder emptying. Case 2 demonstrated the classical features of Hinman syndrome, including detrusor–sphincter dyssynergia, poor compliance, and secondary high-grade vesicoureteral reflux (VUR), ultimately resulting in unilateral renal scarring and loss of function. Case 3, conversely, displayed a hypocontractile detrusor and high residual volumes, suggesting a late-stage, decompensated functional bladder. Despite these distinct urodynamic profiles, all cases shared endoscopic evidence of severe bladder wall remodeling—deep trabeculations, pseudopolypoid mucosa, and submucosal vascular changes—supporting a continuum of functional injury resulting from chronic dyscoordination of voiding.

Exploratory cystoscopy played a pivotal diagnostic role in excluding anatomical causes such as posterior urethral valves, urethral strictures, or postoperative obstruction. The absence of these findings, combined with characteristic urodynamic patterns, confirmed a functional etiology in all patients. This reinforces the importance of a structured diagnostic algorithm combining imaging, urodynamics, and endoscopic assessment in children with severe, atypical LUTD.

*3. Hinman Syndrome and Its Relevance to Severe Functional LUTD*

The presentation of severe lower urinary tract dysfunction (LUTD) in neurologically and anatomically normal children shares many pathophysiological and clinical features with Hinman syndrome, also known as the non-neurogenic neurogenic bladder first described by Hinman and Bauman in 1973 [11]. In this condition, children exhibit the urodynamic and morphological characteristics of neurogenic bladder—such as detrusor–sphincter dyssynergia, trabeculated bladder wall, and progressive upper urinary tract changes—yet lack any identifiable neurological lesion[12–14]. The underlying mechanism is believed to involve maladaptive learned voiding behaviors, chronic contraction of the external urethral sphincter during voiding, and consequent elevation of intravesical pressures. Over time, these functional disturbances lead to detrusor hypertrophy, loss of compliance, and secondary vesicoureteral reflux, closely mirroring the cystoscopic and urodynamic findings observed in our cohort [2,7].

Recent evidence suggests that Hinman syndrome may represent the severe end of the non-neurogenic LUTD spectrum, wherein prolonged behavioral dysfunction, emotional stressors, and delayed diagnosis culminate in irreversible bladder remodeling and renal compromise[15,16]. Recognition of this continuum is essential, as early identification and targeted functional therapy—combining urotherapy, biofeedback, pharmacologic modulation (e.g., trospium chloride), and psychological support—can halt or even reverse detrusor deterioration in many cases [17]. Conversely, misinterpretation of these findings as structural pathology and subsequent surgical interventions, such as reimplantation or bladder outlet procedures, frequently result in persistence or worsening of symptoms. Therefore, awareness of the Hinman paradigm reinforces the need for a

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function-first diagnostic approach and comprehensive multidisciplinary management to prevent progression to upper tract damage and preserve long-term renal function.

*4. Management implications*

Management of severe functional LUTD requires a multimodal approach, addressing both bladder dynamics and behavioral contributors. In all three patients, therapy was based on the integration of pharmacologic modulation, pelvic floor retraining (biofeedback), and, when indicated, continuous antibiotic prophylaxis (CAP) or clean intermittent catheterization (CIC).

Anticholinergic therapy with trospium chloride, used in Cases 1 and 2, effectively reduced detrusor overactivity and improved bladder compliance, consistent with previously reported pediatric outcomes. In Case 3, tamsulosin (Omnic-Tocas) was chosen to reduce functional outlet resistance and facilitate voiding in the setting of a hypocontractile bladder. Biofeedback training proved beneficial across all cases, enhancing sphincter relaxation and voiding coordination, which are often impaired in these children. The gradual but consistent clinical improvement, including resolution of recurrent UTIs and continence restoration, highlights the efficacy of this non-surgical, function-oriented therapy.

The therapeutic use of trospium chloride in children with bladder dysfunction has been increasingly documented, although data remain limited compared to adult populations. Controlled studies, such as that by Pereira et al. on children with detrusor instability, demonstrated significant symptomatic and urodynamic improvement with trospium compared to placebo, supporting its efficacy in pediatric overactive bladder [18]. Subsequent reviews and clinical overviews have confirmed its favorable safety profile and rapid therapeutic response, with up to 90% of children responding within the first week of treatment [19]. Importantly, trospium's quaternary ammonium structure limits its penetration across the blood–brain barrier, resulting in fewer central nervous system adverse effects relative to other antimuscarinics, a key advantage in pediatric use [20,21].
Although most published studies involve idiopathic or neurogenic detrusor overactivity, rather than severe functional LUTD or Hinman syndrome, their findings support the pharmacologic rationale for antimuscarinic therapy in such cases. In our series, trospium chloride was combined with biofeedback-based pelvic floor retraining, leading to marked symptomatic improvement and resolution of infections in all three patients. These outcomes align with prior evidence suggesting that bladder relaxation, improved compliance, and reduction of detrusor overactivity are achievable in children when trospium is used as part of a structured conservative regimen [18–21]. Given the paucity of reports in severe, non-neurogenic LUTD, our experience expands on existing literature by demonstrating that trospium chloride may play a useful adjunctive role in preventing recurrent urinary tract infections and preserving bladder compliance in this complex subgroup.

*5. Literature Review Methods*

To contextualize our findings within existing evidence, we performed a structured narrative review of the literature on severe lower urinary tract dysfunction (LUTD) and Hinman syndrome in neurologically intact children. A comprehensive search was conducted in PubMed, Scopus, and Web of Science for studies published between 1970 and December 2024, using combinations of the following terms: *"Hinman syndrome," "non-neurogenic neurogenic bladder," "functional lower urinary tract dysfunction," "voiding dysfunction," "children,"* and *"pediatric."* Eligible publications included case reports, case series, and observational studies describing severe non-neurogenic LUTD in otherwise healthy children. Studies were excluded if they involved neurogenic, anatomical, or postoperative bladder dysfunction. Reference lists of relevant papers were screened to identify additional reports. Extracted data included patient demographics, neurological findings, key imaging and urodynamic features, management strategies, and clinical outcomes. Representative studies were summarized and compared with the present cases (Table 3).

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**Table 3.** Comparison of Present Cases with Published Reports on Severe Functional Lower Urinary Tract Dysfunction and Hinman Syndrome.

| Study / Source | Age / Sex | Neurological Findings | Key Imaging / Cystoscopic Findings | Urodynamic Pattern | Management | Outcome / Remarks |
|---|---|---|---|---|---|---|
| Present Report – Case 1 | 7 y / M | Normal | Crenulated bladder, trabeculated mucosa, pseudopolypoid changes | Detrusor overactivity, small capacity, uninhibited contractions | Trospium chloride, CAP, biofeedback | Resolution of incontinence and UTIs; relapse after withdrawal, improved with retreatment |
| Present Report – Case 2 | 3 y / F | Normal | Grade V VUR, trabeculated pseudopolypoid mucosa, difficult ureteric visualization | Poor compliance, detrusor–sphincter dyssynergia, high PVR | CAP, trospium chloride, CIC, biofeedback | Gradual functional improvement; resolution of UTIs; diagnosed as Hinman syndrome |
| Present Report – Case 3 | 10 y / M | Normal | Large-capacity bladder, mild crenulation, trabeculated trigone | Hypocontractile detrusor, low Qmax (4 mL/s), high PVR | Tamsulosin (Omnic-Tocas), biofeedback | Slow but complete resolution of acute retention |
| Hinman & Bauman, 1973 (J Urol 109:727–732) | 6–15 y / Mixed | Normal | Trabeculated, thick-walled bladder; VUR common | Detrusor–sphincter dyssynergia; high pressures | Behavioral retraining, catheterization | Variable; some progressed to renal failure |
| Lee et al., 2007 (Korean J Urol 48:1058) | 5–17 y / 14 pts | Normal | Trabeculation, diverticula, VUR in 50% | Poor compliance; DSD | CIC ± anticholinergics | Improved bladder compliance; preserved renal function in most |
| Chaichanamongkol et al., 2008 (Clin Exp Nephrol 12:145) | 1.5 y / M | Normal | VUR, hydronephrosis | DSD; poor compliance | CIC, CAP | Recovery from renal failure; long-term follow-up stable |
| Gampala et al., 2024 (Cureus 16:e55684) | 14 y / M | Normal | Bilateral VUR, trabeculated bladder | DSD; incomplete voiding | Anticholinergic, CIC | Improved voiding and infection control |
| Jayanthi et al., 1997 (J Urol 158:1282) | <2 y / Mixed | Normal | Thickened bladder wall; reflux | DSD, poor compliance | CIC, behavioral therapy | Early infancy presentation; good outcome with early management |

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| | | | | | | |
|---|---|---|---|---|---|---|
| Psychogenic Urinary Retention (Wan et al., 2010) | 9 y / F | Normal | Normal bladder and urethra | Normal detrusor; voluntary retention | Psychological counseling | Complete recovery; illustrates differential |

*6. Outcomes and comparison with literature*

All three patients fit well within the functional–non-neurogenic spectrum described in prior reports of Hinman syndrome and severe LUTD. Like historical cases, all demonstrated bladder wall trabeculation and abnormal voiding dynamics without neurogenic lesions. However, unlike many earlier series where renal impairment was common, timely recognition and functional management (biofeedback, pharmacologic modulation, CAP/CIC) in your cases led to favorable outcomes and renal preservation. This underscores the evolution in diagnostic awareness and conservative treatment efficacy over the past two decades (Table 3).

The clinical spectrum and outcomes of severe functional lower urinary tract dysfunction (LUTD) and Hinman syndrome have been documented in several case reports and small series over the past five decades. The original description by Hinman and Bauman (1973) established the concept of a *non-neurogenic neurogenic bladder*, in which chronic functional obstruction and detrusor–sphincter discoordination produce bladder wall trabeculation, diverticula, and progressive renal injury despite normal neurological findings [11]. Subsequent studies, including Lee et al. (2007), reported long-term follow-up of 14 pediatric cases, demonstrating that poor bladder compliance and persistent high detrusor pressures are common and may lead to vesicoureteral reflux (VUR) and upper tract deterioration if untreated [22]. Jayanthi et al. (1997) expanded the understanding of this entity to infancy, describing similar urodynamic findings even before toilet training [23].

Individual case reports, such as those by Chaichanamongkol et al. (2008) and Gampala et al. (2024), reinforced that Hinman syndrome can manifest with recurrent urinary retention, hydronephrosis, and even renal failure, but that timely initiation of clean intermittent catheterization (CIC) and pharmacologic therapy can stabilize or reverse renal dysfunction [24,25]. Hoebeke et al. (2010) and Kocvara et al. (2007) emphasized the role of detailed urodynamic assessment in distinguishing functional from neurogenic causes of LUTD and advocated early behavioral and biofeedback-based interventions to improve detrusor–sphincter coordination [16,26]. In addition, rare presentations of psychogenic urinary retention in otherwise normal children, such as that reported by Wan and Yang (2010), highlight the potential contribution of psychosocial and learned behavioral factors in this functional spectrum [27].

Overall, the literature supports the interpretation that severe LUTD in neurologically intact children represents a continuum ranging from dysfunctional voiding to the full expression of Hinman syndrome, with outcomes closely linked to the timing and adequacy of diagnosis. Early multidisciplinary intervention — combining urotherapy, pharmacologic modulation, and biofeedback retraining — remains the cornerstone of successful management and prevention of irreversible renal damage.

*7. Combination Therapy with Trospium Chloride and Biofeedback*

In our institution, we have increasingly adopted a combination of biofeedback training and pharmacologic modulation with trospium chloride for children presenting with severe non-neurogenic LUTD, particularly those with detrusor overactivity or dysfunctional voiding unresponsive to standard urotherapy alone. Trospium chloride, a quaternary ammonium antimuscarinic, exhibits limited central nervous system penetration due to its hydrophilic nature, making it particularly well-suited for pediatric patients who may experience cognitive or behavioral side effects with tertiary amines such as oxybutynin. In our three cases, trospium chloride was introduced (at a dose of 0.2–0.3 mg/kg twice daily) in two patients showing persistent urgency and detrusor overactivity despite initial urotherapy. Both patients underwent concurrent pelvic floor biofeedback sessions, emphasizing relaxation training, sphincter coordination, and timed voiding strategies. Within 6 to 8 weeks, both children demonstrated marked symptomatic improvement—

 doi:10.20944/preprints202510.1129.v1



reduction in daytime incontinence episodes, improved bladder capacity on follow-up uroflowmetry, and complete resolution of recurrent urinary tract infections. No significant anticholinergic side effects (dry mouth, constipation, or blurred vision) were observed during treatment. The synergistic use of biofeedback and trospium chloride appears to enhance therapeutic response by combining pharmacologic reduction of detrusor hyperactivity with behavioral retraining of pelvic floor control. Similar results have been reported in recent pediatric cohorts, where biofeedback-based urotherapy augmented by selective antimuscarinics significantly improved bladder compliance and reduced infection recurrence rates. These outcomes reinforce the value of a multimodal, function-focused approach in children with refractory functional LUTD before considering invasive procedures.

Trospium chloride offers a distinct pharmacological advantage in pediatric LUTD management due to its limited central nervous system penetration, favorable safety profile, and complementary effect with behavioral retraining via biofeedback. This combination allows functional restoration without the neurocognitive side effects sometimes seen with tertiary amines, making it a preferred option in our institutional protocol for severe or refractory functional LUTD.

*8. Misdiagnosis and Iatrogenic Management Pitfalls*

An important observation from both our experience and the published literature is that children with severe lower urinary tract dysfunction (LUTD) are often misdiagnosed as having structural abnormalities, leading to unnecessary surgical interventions such as endoscopic bulking injections or ureteral reimplantation [28,29]. These procedures are frequently undertaken in response to recurrent urinary tract infections (UTIs) or hydronephrosis, without comprehensive evaluation of underlying voiding dysfunction. When the aetiology is functional bladder outlet obstruction or detrusor overactivity, such surgeries fail to address the primary pathophysiology. Consequently, incontinence, recurrent infection, and high post-void residuals may persist postoperatively, and upper tract deterioration can progress due to sustained high intravesical pressures [28–30].

Several studies have emphasized this diagnostic and management gap (Table 4). For example, Kocvara et al. (2007) and Hoebeke et al. (2010) demonstrated that persistent VUR and febrile UTIs after ureteral reimplantation are often linked to unrecognized dysfunctional voiding or bladder–bowel dysfunction (BBD) rather than surgical failure [16,26]. The International Children's Continence Society (ICCS) guidelines further caution against performing anti-reflux or outlet procedures before comprehensive functional assessment, including uroflowmetry, post-void residual measurement, and urodynamic studies [2,31]. Misinterpreting incontinence or recurrent infections as indicators of anatomical reflux rather than functional dysfunction remains a leading cause of iatrogenic persistence of symptoms and poor long-term outcomes [2].

In our cohort, all three patients were initially assessed elsewhere for suspected reflux or obstruction, and two had been considered for cystoscopy intervention before referral. Correct recognition of non-neurogenic functional LUTD and implementation of urotherapy-based management led to marked symptom improvement or complete resolution, thereby avoiding unnecessary surgical procedures. Case 2 particularly illustrates this pitfall: the patient underwent Cohen cross-trigonal ureteral reimplantation for presumed reflux, yet continued to experience infections and incontinence until a diagnosis of Hinman syndrome was made. Similar outcomes have been described in other reports, where endoscopic bulking, bladder neck incisions, or reimplantation failed to yield durable benefit due to underlying functional voiding disorders [26,28,30]. Collectively, these findings underscore that in children with severe LUTD, surgical correction should be deferred until functional aetiologies are rigorously excluded, and a function-first, multidisciplinary diagnostic paradigm should be adopted to prevent iatrogenic morbidity [1,31,32].

 doi:10.20944/preprints202510.1129.v1



**Table 4.** Common Missteps in the Management of Severe Lower Urinary Tract Dysfunction (LUTD) in Otherwise Healthy Children.

| Common Misstep | Underlying Issue / Reason | Typical Consequence | Evidence-Based Alternative (ICCS & Current Guidelines) |
|---|---|---|---|
| Performing cystoscopic bulking injections for presumed VUR without functional assessment | Misinterpretation of rUTIs or hydronephrosis as anatomical reflux | Persistence or recurrence of UTIs and incontinence; unresolved high bladder pressures; possible upper tract deterioration | Comprehensive LUTD work-up first: uroflowmetry, post-void residual (PVR), and urodynamics; initiate urotherapy ± pharmacotherapy before considering anti-reflux surgery |
| Ureteral reimplantation in children with unrecognized dysfunctional voiding | Reflux secondary to bladder outlet dysfunction misdiagnosed as primary anatomical VUR | Postoperative persistence of reflux/incontinence; recurrent infections despite technically successful surgery | Treat functional outlet dysfunction (biofeedback, timed voiding, bowel management, antimuscarinics/α-blockers); reassess reflux after functional correction |
| Labeling incontinence or retention as behavioral without urodynamic confirmation | Lack of objective testing; underestimation of detrusor overactivity or underactivity | Delayed diagnosis; progression to hydronephrosis or renal scarring | Early non-invasive uroflow/PVR; cystometry when indicated; phenotype-guided therapy |
| Neglecting constipation or bowel dysfunction in LUTD management | Overlooking bladder–bowel interaction | Treatment failure; recurrent UTIs and incontinence | Integrated bowel regimen as part of standard urotherapy; dietary fiber, laxatives, timed toileting |
| Prolonged antibiotic prophylaxis without addressing voiding dysfunction | Treating infection consequence rather than the cause | Persistent bacteriuria and antimicrobial resistance | Functional evaluation and correction; prophylaxis only as temporary adjunct during therapy initiation |
| Proceeding to invasive or surgical intervention before multidisciplinary review | Fragmented care, absence of urodynamic input | Iatrogenic morbidity, continued symptoms | Multidisciplinary team evaluation (urology, nephrology, physiotherapy, psychology); individualized stepwise management |

Key message: A "function-first" diagnostic approach—combining detailed history, bladder diary, non-invasive urodynamics, and bowel assessment—prevents unnecessary surgical procedures and ensures durable symptom resolution.

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*9. Prognosis and Long-Term Outcomes*

With early diagnosis and appropriate multidisciplinary management, functional recovery and renal preservation are achievable. All three patients in our series demonstrated significant clinical improvement and stabilization of the upper urinary tract following conservative therapy. However, recurrence after withdrawal of treatment, as seen in Case 1, highlights the need for long-term follow-up and patient adherence to urotherapy and behavioral measures. In contrast, delayed recognition and inappropriate surgical management, as illustrated by Case 2, may contribute to irreversible renal damage.

*10. Limitations*

Our series is small and retrospective, limiting generalizability. We did not employ validated symptom instruments (e.g., DVSS/Vancouver) at every visit, nor did we include a control group; future prospective studies should incorporate standardized patient-reported outcomes, rigorous UTI definitions, and longer renal follow-up. Imaging and urodynamics were performed at a tertiary center, which may introduce referral bias.

*11. Clinical takeaways*

Severe LUTD in otherwise healthy children is not rare in specialty practice and should be considered when rUTIs persist despite guideline-based infection management. 2) Early urodynamic phenotyping is valuable to direct therapy. 3) Urotherapy plus targeted add-ons (biofeedback, pharmacotherapy, CIC) can meaningfully reduce infections and protect the upper tracts. 4) Consistent use of ICCS terminology facilitates care coordination and research comparability.

The three cases presented reinforce the growing body of evidence that severe lower urinary tract dysfunction (LUTD) can occur in neurologically and anatomically normal children, representing the functional spectrum of disorders historically encompassed by Hinman syndrome. Despite differences in urodynamic profiles—ranging from detrusor overactivity to hypocontractility—all patients exhibited high-pressure bladder dynamics and secondary bladder wall remodeling, which are recognized risk factors for upper urinary tract deterioration and renal scarring if left untreated. Early recognition of this condition, supported by comprehensive urodynamic assessment and endoscopic evaluation, is therefore essential to distinguish functional from anatomical causes of obstruction. Moreover, our experience underscores that timely initiation of conservative, function-oriented management—including pharmacologic modulation, biofeedback retraining, and, when necessary, intermittent catheterization—can lead to substantial functional recovery and preservation of renal integrity. These observations highlight the importance of multidisciplinary follow-up and long-term behavioral reinforcement in achieving sustained remission and preventing irreversible renal damage.

## 5. Conclusions

These cases collectively illustrate the heterogeneity and clinical complexity of severe functional LUTD in neurologically normal children. Recognition of this disorder as part of the Hinman spectrum is essential to avoid unnecessary surgical interventions and to implement early, targeted urodynamic and behavioral therapy. Multidisciplinary evaluation—including pediatric urology, nephrology, physiotherapy, and psychology—is critical to achieve durable functional recovery and prevent long-term renal morbidity.

**Author Contributions:** Conceptualization O.S., M.A.; Investigation O.S., A.M.; Methodology L.B., R.B.; Project administration M.A., Supervision L.B.,R.B.; Validation A.M., M.A.; Writing - original draft O.S.; Writing - review & editing A.M., M.A.

**Funding:** This research received no external funding.

**Informed Consent Statement:** Informed consent was obtained from all subjects involved in the study.

**Data Availability Statement:** De-identified data are available from the corresponding author on reasonable request; public sharing is restricted by patient privacy and institutional policy.

 doi:10.20944/preprints202510.1129.v1



**Acknowledgments:** During the preparation of this manuscript/study, the author(s) used OpenAI's GPT-5 language model (ChatGPT, 2025 version) for the purposes of refining language and generate references. The authors have reviewed and edited the output and take full responsibility for the content of this publication.

**Conflicts of Interest:** The authors declare no conflicts of interest.

## Abbreviations

The following abbreviations are used in this manuscript:

| | |
|---|---|
| LUTD | Lower Urinary Tract Dysfunction |
| UTI | Urinary Tract Infection |
| VCUG | Voiding Cystourethrography |
| ED | Emergency Department |

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