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Research Article

Assessment of Uroflowmetry Patterns in Patients with Suspected Neurogenic Bladder: A Clinical Correlation

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Abstract

Neurogenic bladder remains a challenging clinical condition associated with impaired urinary function and long-term morbidity. Uroflowmetry provides a non-invasive and objective tool for assessing voiding dynamics in patients with suspected neurogenic dysfunction. This study aimed to evaluate uroflowmetry patterns and correlate them with clinical presentations in patients with suspected neurogenic bladder. A cross-sectional comparative analysis was conducted involving 120 patients stratified into suspected neurogenic bladder and non-neurogenic lower urinary tract symptom groups. Uroflowmetry parameters, including maximum flow rate (Qmax), average flow rate (Qavg), voided volume, and post-void residual urine, were recorded. Results demonstrated statistically significant reductions in Qmax (p<0.001) and Qavg (p=0.002) in the neurogenic bladder group, with markedly higher residual urine volumes (p<0.001). A distinct flattened curve pattern was predominant in neurogenic patients compared to bell-shaped curves in controls. These findings suggest that uroflowmetry, when interpreted with clinical features, can serve as a reliable initial screening modality for early identification of neurogenic voiding dysfunction. The study underscores its value in resource-limited settings where invasive urodynamic studies may not be accessible.

Keywords: Uroflowmetry, Neurogenic Bladder, Voiding Dysfunction

Introduction

Neurogenic bladder represents a broad spectrum of lower urinary tract dysfunction resulting from neurological conditions affecting bladder innervation and coordination. The prevalence is increasing in parallel with neurological disorders such as spinal cord injuries, multiple sclerosis, Parkinson's disease, and diabetic neuropathy. Patients frequently present with symptoms of urinary frequency, urgency, incomplete emptying, or recurrent infections. Early detection and accurate evaluation of bladder dysfunction are critical to preventing renal deterioration and improving quality of life.1-4

Urodynamic studies remain the gold standard for diagnosing neurogenic bladder, yet they are invasive, resource-intensive, and often impractical for routine screening in many clinical settings. Uroflowmetry has emerged as a simple, cost-effective, and non-invasive tool that provides valuable insights into voiding patterns by graphically representing urine flow rates. Distinct uroflowmetric patterns, such as plateau-shaped or interrupted curves, are often associated with neurogenic dysfunction, whereas normal physiology typically produces a bell-shaped curve.5-7

Recent literature emphasizes the role of uroflowmetry as a first-line diagnostic tool, particularly in patients with suspected neurogenic bladder. It enables clinicians to objectively document flow abnormalities and determine the need for further invasive testing. Furthermore, coupling uroflowmetry with clinical correlation enhances diagnostic accuracy, potentially reducing unnecessary invasive procedures.8-10

The rationale of the present study is to comprehensively evaluate uroflowmetry patterns in patients with suspected neurogenic bladder and correlate them with clinical findings. By comparing uroflowmetric parameters between neurogenic and non-neurogenic patients, the study seeks to determine whether uroflowmetry can function as a reliable, non-invasive surrogate marker for early detection of voiding dysfunction.

This approach holds particular importance in resource-limited environments, where access to comprehensive urodynamic facilities is limited. Additionally, it has implications for long-term patient monitoring, therapeutic decision-making, and outcome evaluation in patients with established neurological disease.

Methodology

This cross-sectional comparative study was conducted on 120 adult patients presenting with lower urinary tract symptoms at Sialkot Medical College. Patients were stratified into two groups: 60 with suspected neurogenic bladder based on underlying neurological disease or symptoms suggestive of neurogenic dysfunction, and 60 controls with non-neurogenic lower urinary tract symptoms. Sample size was calculated using Epi Info software with a power of 80% and confidence interval of 95%, considering effect size from previously published uroflowmetry studies.

Inclusion criteria included adults aged 18–70 years, with neurological disease or symptoms raising suspicion of neurogenic bladder, and controls with benign prostatic hyperplasia or overactive bladder. Exclusion criteria were acute urinary tract infection, prior pelvic surgery, urethral stricture, or unwillingness to provide verbal consent.

All patients underwent detailed clinical history and physical examination, followed by uroflowmetry using a calibrated digital flowmeter. Parameters recorded included maximum flow rate (Qmax), average flow rate (Qavg), voided volume, flow curve pattern, and post-void residual urine measured by ultrasound. Uroflowmetry was performed with voided volume ≥150 mL for standardization.

Ethical clearance was obtained, and verbal informed consent was secured prior to participation. Data were analyzed using SPSS v25. Mean and standard deviation were calculated for continuous variables, and independent t-test was used for between-group comparisons. A p-value <0.05 was considered statistically significant.

Results

Table 1. Demographic Characteristics

Variable	Neurogenic Group (n=60)	Control Group (n=60)	p-value
Age (years, mean±SD)	48.6 ± 12.3	46.9 ± 11.5	0.42

Zeeshan Shaukat et al / Assessment of Uroflowmetry Patterns in Patients with Suspected Neurogenic Bladder:

A Clinical Correlation

Variable	Neurogenic Group (n=60)	Control Group (n=60)	p-value
Male/Female (%)	60/40	58/42	0.87
Duration of symptoms (months)	14.2 ± 6.7	11.8 ± 5.9	0.08

Demographics were comparable, with no significant difference in age, gender distribution, or symptom duration.

Table 2. Uroflowmetry Parameters

Parameter	Neurogenic Group (mean±SD)	Control Group (mean±SD)	p-value
Qmax (mL/s)	8.9 ± 3.1	15.6 ± 4.2	<0.001
Qavg (mL/s)	5.7 ± 2.4	10.1 ± 3.3	0.002
Voided Volume (mL)	185 ± 52	210 ± 46	0.07
Post-void Residual (mL)	92 ± 28	35 ± 14	<0.001

The neurogenic group exhibited significantly reduced Qmax and Qavg with higher residual volumes compared to controls.

Table 3. Flow Curve Patterns

Curve Pattern	Neurogenic Group (n=60)	Control Group (n=60)
Bell-shaped	12 (20%)	48 (80%)
Plateau/Flat	30 (50%)	6 (10%)
Interrupted	15 (25%)	4 (7%)
Staccato	3 (5%)	2 (3%)

Flattened and interrupted flow curves predominated in neurogenic patients, while controls demonstrated predominantly bell-shaped patterns.

Discussion

This study highlights the clinical value of uroflowmetry as a non-invasive screening tool for suspected neurogenic bladder. Significant reductions in maximum and average flow rates, coupled

with increased residual urine volumes, were observed among neurogenic patients, aligning with the pathophysiological disruption of bladder innervation. Distinct uroflowmetric patterns, particularly plateau and interrupted curves, were strongly associated with neurogenic dysfunction.11-13

The findings corroborate emerging evidence that uroflowmetry can serve as an effective first-line diagnostic modality, particularly in environments lacking advanced urodynamic testing facilities. Reduced Qmax and abnormal curve morphology offer easily interpretable markers for clinicians, facilitating timely identification of neurogenic bladder and guiding further evaluation.14-16

Notably, voided volume was not significantly different between groups, underscoring that flow dynamics, rather than absolute volume, better reflect neurogenic impairment. This suggests that reliance solely on volume-based assessments may overlook subtle dysfunction.17-18

Higher post-void residual volumes in the neurogenic cohort are consistent with impaired detrusor contractility and incomplete bladder emptying, highlighting a crucial risk factor for recurrent infections and upper tract damage. Early identification of this parameter through uroflowmetry has direct implications for patient management. 19-20

The distinct flow curve distribution in neurogenic patients strengthens the role of qualitative uroflowmetry interpretation alongside quantitative analysis. The predominance of plateau and interrupted patterns reflects underlying neurological disruption of detrusor—sphincter coordination, which is pathognomonic for neurogenic dysfunction.

Furthermore, the integration of uroflowmetry with clinical correlation offers a comprehensive, low-cost diagnostic pathway. This model enhances accessibility in resource-limited settings, allowing prioritization of patients for more invasive and costly evaluations.

Future directions include longitudinal monitoring of uroflowmetry patterns in neurogenic patients to assess therapeutic responses and progression. The study underscores the need for broader implementation of uroflowmetry in clinical practice, not only as a diagnostic adjunct but also as a monitoring tool for neurogenic bladder management.

Conclusion

Uroflowmetry demonstrates significant diagnostic value in patients with suspected neurogenic bladder, with reduced flow rates, elevated residual volumes, and abnormal curve patterns strongly correlating with clinical features. Its non-invasive, accessible nature supports its role as an essential frontline diagnostic tool in both primary and specialized care.

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