

Evaluation of Voiding Dysfunction in Patients with Traumatic Brain Injury

Amit Kumar¹ Suryaprakash Vaddi¹ Vijayabhaskar Reddy Gouru¹ Amit Agrawal²

¹Department of Urology and Renal Transplantation, Narayana Medical College, Nellore, Andhra Pradesh, India

²Department of Neurosurgery, Narayana Medical College, Nellore, Andhra Pradesh, India

Address for correspondence Amit Agrawal, MCh, Professor of Neurosurgery, Department of Neurosurgery, Narayana Medical College Hospital, Chinthareddypalem, Nellore 524003, Andhra Pradesh, India (e-mail: dramitagrawal@gmail.com).

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Abstract

Introduction Voiding dysfunction after a traumatic brain injury (TBI) occurs secondary to impaired cognitive or behavioral functioning, brain damage, associated injury to the spinal cord, and direct bladder trauma. In this study, the authors collected data on TBI patients with neurogenic bladder dysfunctions to understand the spectrum of voiding dysfunctions and to investigate the relationships of the severity of TBI with the presentation of urinary tract dysfunctions.

Materials and Methods All consecutive patients with isolated TBI of Glasgow Coma Scale (GCS) (9–15) in the postacute period and at 3 months were included in the study. In patients with previous stroke or any other neurologic disorder or associated spine injury and with urologic conditions such as benign prostatic hyperplasia (BPH), the urethral stricture is excluded. After a successful voiding trial, uroflowmetry to see the flow pattern and ultrasound of the abdomen and pelvis for post-void residual urine is done. A urodynamic study is done in patients with poor urinary flow (< 15 mL/s), high post-void residual urine (> 100 mL), and/or lower urinary tract symptoms.

Results During the study period, a total of 55 patients were enrolled in the study: 43 were males and 12 were females. Out of 55 patients, 47 were treated conservatively and 8 were operated upon for TBI. Out of 55 patients, 26 had a poor stream on uroflowmetry and 19 underwent urodynamic study either due to urinary symptoms or poor stream. Out of the 19 patients, 9 had a urodynamic abnormality. Of the 19 patients who underwent urodynamic study, there was no significant correlation ($p = 0.23$) between the TBI pathology and urodynamic abnormality. There was also no significant correlation seen between site of lesion and urodynamic abnormality.

Conclusion The prevalence of urinary symptoms in mild and moderate TBIs is low (7.3); 47.7% of patients had poor urinary flow. None of the patients had an abnormality in filling phase. Nine patients had an abnormality in voiding phase and were treated with an α -blocker.

Duration During 2012 to 2015.

Keywords

- traumatic brain injury
- voiding dysfunction
- urinary bladder

Introduction

Traumatic brain injury (TBI) can be a serious neurologic event with a residual sequel (may be temporary or permanent).¹ Voiding dysfunction after a TBI occurs secondary to impaired cognitive or behavioral functioning, brain damage, associated

injury to the spinal cord, and direct bladder trauma. There can be a spectrum of voiding dysfunctions following TBI, including urine retention, detrusor over activity, poor urethral sphincter control (urethral sphincter pseudo dyssynergia), and detrusor areflexia. Management of bladder and bowel function is, therefore, an extremely important area to

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be addressed in the rehabilitation of brain-injured patients.²⁻⁶ There are only a few studies that have investigated the urodynamic findings and characteristics of lower urinary tract dysfunction in TBI patients.²⁻⁹ In this study, we collected data on TBI patients with neurogenic bladder dysfunctions to understand the spectrum of voiding dysfunctions and to investigate the relationships of the severity of TBI with the presentation of urinary tract dysfunctions.

Materials and Methods

This is an observational study, during 2012 to 2015, focused on bladder dysfunction in isolated TBI in the postacute phase and at 3 months. Postacute phase means patients have regained their consciousness and are oriented and able to communicate. All consecutive patients with isolated TBI of Glasgow coma scale (GCS) (9–15) are included in the study. In patients with previous stroke or any other neurologic disorder or associated spine injury and with urologic conditions such as benign prostatic hyperplasia (BPH), the urethral stricture is excluded. Patients with GCS (9–15) are enrolled in the study after informed consent. Patients who are conscious are evaluated by the urologist, and a trial without a catheter is planned. After a successful voiding trial, uroflowmetry to see the flow pattern and ultrasound of the abdomen and pelvis for post-void residual urine is done. A urodynamic study is done in patients with a poor urinary flow (< 15 mL/s), high post-void residual urine (> 100 mL), and/or lower urinary tract symptoms (LUTS). All patients voiding with LUTS and patients with abnormal flow pattern and/or significant post-void residual urine were also evaluated by urodynamic study. A detailed cystometrogram with electromyogram was done to study the abnormalities in filling and voiding phases, and the voiding dysfunction is characterized based on the urodynamic findings.

Results

During the study period, total 55 patients were enrolled in the study: 43 were males and 12 were females. The age range was 12 years to 87 years. The mean age was 34.4 years. The mode of injury was fall from height in 6 and road traffic accidents in 49 patients. Nature of injury was extradural hemorrhage (EDH) in 13 out of 55 patients and subdural hemorrhage (SDH) in 10 out of 55 patients. Out of 55 patients, 47 were treated conservatively and 8 were operated upon for TBI. Out of 55 patients, 22 were catheterized in the emergency department. Patients were either referred to our institute with catheter or catheterized due to their low GCS score. In our study, 31 (56.4%) had left-sided lesions and 18 (32.7%) had right-side lesions, and 6 had bilateral injuries. Frontal lesions were seen in 18 (32%), frontotemporal in 6 (10.9%), frontoparietal in 5 (9%), occipital in 3 (5%), fronto-temporo-parietal in 5 (9%), temporoparietal in 3 (5%), temporal in 7 (12%), diffuse cerebral injury in 4 (5%), and basilar and vault fractures in 4 patients. Out of 55 patients, 26 (47%) had a poor stream on uroflowmetry and 19 patients underwent urodynamic study either due to urinary symptoms (4 patients) such as frequency, urgency, and nocturia, or high post-void residue

(> 100 mL) in 5 patients and poor stream. Seven patients with poor flow without other urinary symptoms were observed as they were not willing for urodynamic study. Nine patients had urodynamic abnormality. Eight of these nine patients had high detrusor pressure with poor flow on pressure-flow study suggestive of obstructive voiding. One patient had a hypocontractile bladder. All patients had coordinated sphincter control. On analysis of poor urinary flow group patients (26), most patients with diffuse cerebral lesions (8/10 patients) had poor urinary flow compared with frontal, temporal occipital, and parietal lesion ($p < 0.045$). Of the 19 patients who underwent urodynamic study, there was no significant correlation ($p = 0.23$) between the TBI pathology and urodynamic abnormality. There was also no significant correlation seen between site of lesion and urodynamic abnormality.

Discussion

The storage and voiding of urine depends on complex neural control system that coordinates autonomic and somatic nerve activity to various distal sites, including the detrusor muscle, regions of the bladder, urethra and urethral sphincter, and striated muscles of the pelvic floor. The central connections of the bladder sphincter unit have been functionally grouped together into “circuits”—the so-called Bradley’s loops^{10,11}: Loop I: pathways integrating the cerebral cortex, basal ganglia, thalamus, and cerebellum with the pontomesencephalic reticular formation; loop II: afferents from the bladder stretch receptors traveling via the dorsal roots and posterior column to the pontomesencephalic reticular formation efferent from here to the sacral motor neurons controlling the detrusor and the striated sphincter; loop III: local loops between the bladder stretch receptors, sacral segments, and therefore to the detrusor and sphincters; loop IV: descending pathways from the motor cortex to the sacral anterior horn cells innervating the striated sphincter. All four loops should work in coordinated manner for normal micturition cycle. Disruption at any of the loops can cause urinary dysfunction.

When voiding dysfunction occurs, there may be an initial period of detrusor areflexia. With lesions above the pontine micturition center, involuntary bladder contractions are the most frequent manifestation of chronic lower urinary tract dysfunction. Coordinated sphincter function is the rule. In patients who have more isolated brainstem injuries with involvement below the pontine micturition center, detrusor striated sphincter dyssynergia may occur in addition.

We reviewed the existing literature on voiding dysfunction in TBI patients. There were few studies done on this subject, and majority of them included a small number of patients. Human studies have shown that voluntary voiding is controlled with the connections between the frontal cortex and hypothalamus as well as between the paracentral lobe and pons. Any damage in these areas results in an increase in bladder activity due to the disappearance of inhibitory control.¹² The presence of pathology that affects these systems might cause neurogenic bladder. Brain imaging studies have shown that frontal cortex and anterior cingulate gyrus, the right side of the brain, are responsible for control of micturition.¹³

Traumatic brain injury is more common in males than females with a wide age range. In our study, 43 (78.2%) patients were males and 12 (21.2%) were females. The age range was 12 to 87 years with a mean age of 34.4 years. Chua et al⁷ reported 84 TBI patients with a mean age of 44.7 \pm 17.9 years (range: 18–92 years). The male-to-female ratio was 4:1 (66 males, 18 females). Giannantoni et al⁹ reported 57 patients: 37 men, 20 women. Their mean age was 28.9 years with a range of 13 to 55 years.

The major etiology of TBI is road traffic accidents. In our study, 49 (89%) out of 55 patients had road traffic accidents. In six patients, the etiology was fall from height. Chua et al⁷ reported fall from height was a major cause of TBI accounting for 46 (54.8%) of cases, followed by motor vehicle accidents, recreational injuries, and violence in 33 (39.3%), 3 (3.6%), and 2 (2.4%), respectively. Kulakli et al³ reported that road traffic accident was more common accounting 15 (51.7%), followed by fall, gunshot, and other 9 (31%), 1 (3.4%), 4 (13.8%), respectively. Oostra et al⁶ also reported road traffic accident was the major cause of TBI 23 out of 34 cases accounting 67% followed by fall from height 5 (14%). The second most common cause of TBI is falling from a height. The prevalence of voiding dysfunction depends on the severity of head injury. Chua et al⁷ reported 43 (51%) patients with severe TBI with GCS < 9 and 41 (49%) with moderate head injury, that is, GCS 9–12. Oostra et al⁶ reported that total 34 patients had severe head injuries. As per the study by Singhanian et al,¹² 5 (45%) patients had moderate and 6 (55%) had mild head injury. Moiyadi et al⁵ reported severe TBI in 8 (40%) patients, moderate in 11 (55%), and mild TBI in 1 patient. In our study, 37 (67.3%) patients had a mild head injury and 18 (32.7%) had moderate head injuries.

In our series, 47 (85.5%) patients were treated conservatively and 8 underwent operative management. In our study, 31 (56.4%) patients had left-sided lesions and 18 (32.7%) had right-sided lesions, and 6 had bilateral injuries. Frontal lesions were seen in 18 (32%) patients, frontotemporal in 6 (10.9%), frontoparietal in 5 (9%), occipital in 3 (5%), fronto-temporo-parietal in 5 (9%), temporoparietal in 3 (5%), temporal in 7 (12%), diffuse cerebral injury in 4 (5%), and basilar and vault fractures in 4 patients. Moiyadi et al⁵ reported frontal lesion in 6 patients, temporal in 8, and diffuse cerebral injury in 6 patients in a total of 20. Oostra et al⁶ reported eight out of nine cases were frontal lesions. As per the study by Singhanian et al,¹² frontal, frontoparietal, and fronto-temporo-parietal lesions were seen in 6, 2, and 1 patients, respectively. Most patients had frontal, frontotemporal, and frontoparietal lobe involvement.

In our series, one patient presented as urge urinary incontinence out of four symptomatic patients who had frontotemporal SDH. Three patients had frequency, urgency, and nocturia. The prevalence of urinary symptoms was less in our study compared with other studies. The symptoms and their severity correlated with the severity of TBI in other studies. Most patients with TBI had storage symptoms in all the published literature. As per the study by Giannantoni et al,⁹ out of 57 patients, 49 (85.9%) complained of urinary symptoms; 30 of these (61.2%) had overactive bladder symptoms of urgency and urinary frequency. Seven (14.3%) patients had symptoms of the voiding phase and 12 (24.5%) had both. The

most frequent symptoms of the voiding phase were hesitancy and intermittency (12 patients).

Chua et al⁷ reported that 52 (62%) patients had urinary incontinence and 8.3% had urinary retention. Oostra et al⁶ reported that 50% of patients with severe TBI had incontinence between 3 and 9 months of TBI. Leary et al⁴ reported 50% of patients had urinary incontinence. Kulakli et al³ reported that 3 (21.4%) of 14 hemiparetic patients had storage dysfunction and 4 (28.6%) had emptying dysfunction. Ten of 15 (66.7%) quadri-paretic patients had storage dysfunction and 7 (46.7%) had emptying dysfunction. Urinary symptoms are more common in moderate and severe injuries as compared with mild injuries. Fewer patients with urinary symptoms in our study could be explained by the milder nature of TBI compared with other studies.

In our study, 22 (40%) cases were catheterized in the emergency. All patients voided spontaneously after catheter removal. Three out of these 22 patients had urinary symptoms after removal of the catheter. Seven of them had poor flow (Q_{max} < 15 mL/s) on uroflowmetry. Two patients had LUTS with a poor urinary flow on flowmetry. None of these patients had urinary tract infection or renal impairment. As per the study by Singhanian et al,¹² 10 (90%) patients were catheterized. As per the study by Giannantoni et al,⁹ all the 57 patients were catheterized. After catheter removal, 47 (82%) patients voided spontaneously, 8 (14%) continued to be on indwelling catheter because of severe urinary incontinence, and 2 were on intermittent catheterization. Nine (18.3%) patients reported a history of recurrent urinary tract infections. No patient showed impaired renal function. Chua et al⁷ reported that 6 (7%) of patients had an indwelling catheter and 46 (54.8%) had external collecting devices at the time of admission. At the time of discharge, dependence on external collecting devices decreased significantly from 54.8% (46 patients) to 35.7% (30 patients) on admission and discharge, respectively. In our study, all patients voided at discharge, and in the follow-up at 3 months, all had good urinary flow without any symptoms. The need for indwelling catheters or external collecting devices depends on the severity of brain injury. As all the patients in our study had mild to moderate TBI, catheterization could be avoided in all conscious patients and all patients who were catheterized had a successful trial without a catheter.

Twenty-six (47.7%) patients had poor urinary flow on uroflowmetry. Nineteen patients underwent urodynamic evaluation. Nine out of 19 patients had an abnormality in the urodynamic evaluation. Most of the patients had no urinary symptoms on presentation. On analysis of poor urinary flow group patients (26), most patients with diffuse cerebral lesions (8/10 patients) had poor urinary flow compared with frontal, temporal occipital, and parietal lesion ($p < 0.045$).

Urodynamic study was done in 19 patients. Ten out of 19 patients were found to be normal. In all patients filling phase was normal. No overactivity was seen. In nine patients urodynamic evaluation was abnormal. Eight of these nine patients had high detrusor pressure with poor flow on pressure-flow study suggestive of obstructive voiding. One patient had a hypocontractile bladder. All patients had coordinated sphincter control. Our results are similar to those reported in the study by Krimchansky et al² in which urodynamic study filling phase was

normal in all 17 cases, whereas in other studies a significant percentage of the TBI patients had an abnormal filling phase of the urodynamic study. Ersoz et al⁸ reported that bladder filling sensation was normal in 16 (55.2%) patients, diminished in 11 (37.9%) and was absent in 2 (6.9%). Moiyadi et al⁵ reported 11 patients with abnormal urodynamic study in filling phase that includes overactive detrusor in 8 cases and reduced compliance in 3 cases. Giannantoni et al⁹ reported 12 out of 57 cases of abnormal urodynamic study in which all patients presented with detrusor under activity. Oostra et al⁶ reported detrusor hyperreflexia in 6 out of 9 patients (►Table 1).

In our study, in the 19 patients who underwent urodynamic study, there was no significant correlation ($p = 0.23$)

between the TBI pathology and urodynamic abnormality. There was also no significant correlation seen between the site of lesion and urodynamic abnormality ($p = 0.09$). As per the study by Moiyadi et al,⁵ 7 of 11 patients with abnormal urodynamic had frontal lesions. There was no significant correlation between urodynamic abnormality and catheterization of patients ($p: 0.51$). Five out of eight patients with severe TBI had a urodynamic abnormality. Six out of 12 patients with moderate TBI had a urodynamic abnormality. In our study, 12 out of 18 patients with moderate head injury underwent urodynamic study and 7 had abnormality. Out of nine patients who had urodynamic abnormality, seven had moderate brain injury (►Table 2).

Table 1 Comparison of UDS findings in TBI patients

	Filling phase	Voiding phase
Present series (19)	Normal	High voiding pressure (8) Hypocontractile (1)
Singhania et al ¹² (11)	Hyperreflexia (3)	Normal
Chua et al ⁷ (3)	Hyperreflexia (1)	Hypocontractile (1)
Ersoz et al ⁸ (29)	Delayed sensations (11) Absent sensations (2)	Normal
Giannantoni et al ⁹ (57)	Normal	Hypocontractile (12)
Oostra et al ⁶ (9)	Normal (3) Hyperreflexia (6)	Normal

Abbreviations: TBI, traumatic brain injury; UDS, urodynamic study.

Table 2 Analysis of (26/55) patients with poor stream on uroflowmetry with urodynamic study (UDS)

Sl. No.	Uroflowmetry	Site of injury and nature of pathology—management (conservative/operated)	UDS—filling phase					UDS—voiding phase		EMG
			Capacity	Sensations	Overactivity	Compliance	Leak (DLPP)	Normal	Abnormal Pdet – (cm of water)	
1.	174/9	Left frontotemporal SDH—operated	Normal	Normal	No	Good	No		High pressure Pdet max –115	Synergetic
2.	196/8.9	Left suprasellar SAH—conservative	Normal	Normal	No	Good	No	Yes		Synergetic
3.	196/12.8	Right frontal EDH with right zygomatic and orbital roof fracture—conservative	Normal	Normal	No	Good	No	Yes		Synergetic
4.	314/8	Left zygomatic, ethmoidal, sphenoidal, greater wing of sphenoid fracture with B/L pneumocephalus with SAH—conservative	Normal	Normal	No	Good	No	Yes	High pressure Pdet max –66	Synergetic

(Continued)

Table 2 (Continued) Analysis of (26/55) patients with poor stream on uroflowmetry with urodynamic study (UDS)

Sl. No.	Uroflowmetry	Site of injury and nature of pathology—management (conservative/operated)	UDS—filling phase					UDS—voiding phase		EMG
			Capacity	Sensations	Overactivity	Compliance	Leak (DLPP)	Normal	Abnormal Pdet – (cm of water)	
5.	506/10	EDH in right frontal region and SAH in left parietal region—conservative	Normal	Normal	No	Good	No	Yes		Synergetic
6.	196/4.6	Contusion in left frontal cortex and SDH—conservative	Normal	Normal	No	Good	No	Yes	High pressure Pdet max –62	Synergetic
7.	252/13	Cerebral concussion—conservative	Normal	Normal	No	Good	No	Yes		Synergetic
8.	369/14	Right frontal EDH—conservative	Normal	Normal	No	Good	No	Yes	High pressure Pdet max –62	Synergetic
9.	489/6	Cerebral concussion—conservative	Normal	Normal	No	Good	No	Yes		Synergetic
10.	180/9	Right frontal contusion, SDH in frontotemporal region—conservative	Normal	Normal	No	Good	No	Yes	High pressure Pdet max –92	Synergetic
11.	182/12.4	Left SDH frontoparietal region and EDH on parietal region—operated	Normal	Normal	No	Good	No	Yes		Synergetic
12.	168/11.2	Cerebral concussion—conservative	Normal	Normal	No	Good	No	Yes		Synergetic
13.	214/8.8	EDH in right frontal region and fracture of right roof of orbit—operated	Normal	Normal	No	Good	No	Yes	Hypocontractile Pdet max –15	Synergetic
14.	429/13.9	Left frontal contusion with pneumocephalus—conservative	Normal	Normal	No	Good	No	Yes		Synergetic
15.	260/9	Left temporo-frontal contusion—conservative	Normal	Normal	No	Good	No	Yes	High pressure Pdet max –64	Synergetic
16.	145/12.6	Left temporo-frontal contusion—conservative	Normal	Normal	No	Good	No	Yes	High pressure Pdet max –68	Synergetic

(Continued)

Table 2 (Continued) Analysis of (26/55) patients with poor stream on uroflowmetry with urodynamic study (UDS)

Sl. No.	Uroflowmetry	Site of injury and nature of pathology—management (conservative/operated)	UDS—filling phase					UDS—voiding phase		EMG
			Capacity	Sensations	Overactivity	Compliance	Leak (DLPP)	Normal	Abnormal Pdet – (cm of water)	
17.	719/12.0	Right pneumocephalus with orbital fracture—operated	Normal	Normal	No	Good	No	Yes		Synergetic
18.	303/9.5	Right temporal SDH with fracture of temporal bone—conservative	Normal	Normal	No	Good	No	Yes		Synergetic
19.	143/8.8	Right temporal EDH—conservative	Normal	Normal	No	Good	No	Yes	High pressure Pdet max –55	Synergetic
20.	230/10.8	Cerebral concussion—conservative	Seven patients with the poor flow without symptoms were observed as they were not willing for UDS							
21.	409/13.3	Left frontal contusion with temporal EDH with contusion and SAH—conservative								
22.	163/10.4	Right temporoparietal SDH—conservative								
23.	148/10.8	Right frontal depressed fracture with EDH—conservative								
24.	195/11.0	Left frontal SAH—conservative								
25.	594/11.0	Left frontal SAH—conservative								
26.	184/11.5	Right frontal contusion—conservative								

Abbreviations: B/L, bilateral; DLPP, detrusor leak point pressure; EDH, extradural hemorrhage; EMG, electromyogram; Pdet, detrusor pressure; SAH, subarachnoid hemorrhage; SDH, subdural hemorrhage; UDS, urodynamic study.

Conclusion

The prevalence of urinary symptoms in mild and moderate TBIs is low (7.3); 47.7% of patients had poor urinary flow. None of the patients had an abnormality in filling phase. Nine patients had an abnormality in voiding phase and were treated with an α -blocker. Detailed urologic and urodynamic evaluation should be done in selected patients to diagnose asymptomatic urodynamic abnormality and to treat accordingly.

Conflict of Interest

The authors declare that they have no conflict of interest.

Ethical Standards

All procedures performed in studies involving human participants were in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards.

Informed Consent

Informed consent was obtained from all individual participants included in the study.

Author Contribution

All authors contributed equally to the protocol/project development, data collection or management, data analysis, and manuscript writing/editing.

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