

Profiles of patients with urinary incontinence in the Department of Child Health, Cipto Mangunkusumo Hospital

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ABSTRACT

Background Urinary incontinence (UI) in children is a form of wetting. Early diagnosis and treatment are mandatory to avoid complications such as recurrent urinary tract infections (UTI), vesicoureteral reflux (VUR), or renal damage.

Objective To study the profiles and clinical course of UI in children treated in Cipto Mangunkusumo Hospital.

Methods The study was divided into 2 parts. The first part was a review of patients with UI at Cipto Mangunkusumo Hospital from January 2000 to December 2003. The second was a case series of patients followed up for at least 6 months.

Results There were 35 UI patients aged 3 months to 16 years, mostly between 1 to 5 years old, 16 were males and 19 females. The most prevalent etiology was myelodysplasia (15 cases) followed by posterior urethral valve, and bladder tumor. The most prominent clinical presentation of neurophatic bladder-sphincter dysfunction was wetting, while those of patients with structural incontinence and non-neurophatic bladder-sphincter dysfunction were fever and polakysuria. Most patients had been suffering from renal insufficiency since their first visit. Clean intermittent catheterization (CIC) was the treatment of choice. In a six-month follow-up of 14 patients who received adequate treatment, renal function could be maintained at relatively stable condition in most cases.

Conclusions Myelodysplasia was the most common etiology of UI. Most patients had renal insufficiency or renal failure since their first visit, reflecting an extended period of relapse before patients seek medical help. Renal function can be maintained by adequate treatment in most cases [Paediatr Indones 2005;45:87-92].

Keywords: urinary incontinence, myelodysplasia, renal failure, urinary tract infection

The primary functions of the urinary bladder are adequate storage capacity at low pressure and efficient emptying capability.¹⁻⁵ The International Continence Society (ICS) defines urinary incontinence (UI) as a condition where involuntary loss of urine is a social or hygienic problem and is objectively demonstrable. Others define it as uncontrolled wetting in the form of involuntary loss of small amounts of urine, incomplete voiding that makes the child feel embarrassed and frustrated.⁶

Based on findings of voiding cystourethrography (VCUG) and urodynamic examinations, UI is divided into three etiological classifications i.e., non-neurophatic bladder-sphincter dysfunction (functional incontinence), neurophatic bladder-sphincter dysfunction (neurogenic bladder), and structural or anatomical incontinence.^{7,8} The absence of a uniformed definition of UI leads to problems in epidemiological studies that may influence the prevalence of UI.^{9,10} Data from medical records of the Department of Child Health, Cipto Mangunkusumo Hospital, Jakarta showed that there were 18 cases of UI during an eleven-year period from 1989 to 2000.¹¹

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The diagnosis of UI is based on history-taking, physical examination, urinalysis, ultrasound imaging of kidneys and urinary tracts, VCUG, and urodynamics.^{1,2,4,6,7} The aim of this study was to know the profile and the course of illness of children with UI.

Methods

The study was divided into two parts with two different study designs. The first part was a cross-sectional descriptive study on patients with UI at the Department of Child Health, Cipto Mangunkusumo Hospital between January 2000 and December 2000. The second was a case series report of patients evaluated for 6 months or more of regular follow-up.

The diagnosis of UI was made by pediatric nephrology consultants. Bacteriuria was defined as the presence of organisms >100,000 colony-forming units per ml in a culture of a catheterized urine specimen or a mid-stream urine specimen. Symptomatic UTI was associated with symptoms of dysuria, polakysuria, pyuria, or urgency, with or without fever, and with or without abdominal or costovertebral angle pain. Classification of vesicoureteral reflux was done according to The International Reflux Study Committee, 1981.¹² Renal failure was classified based on GFR as follows: Impaired renal functions (GFR=80-50 ml/minute/1.73 m²), renal insufficiency (GFR=50-30 ml/minute/1.73 m²), chronic renal failure (GFR=30-10 ml/minute/1.73 m²) and end-stage renal failure (GFR=<10 ml/minute/1.73 m²).^{13,14} GFR was determined based on Schwartz's formula.¹⁵ Nutritional status was classified according to the National Centre of Health Statistics standards.¹⁶

Results

During the period of January 2000 to December 2003, 35 patients were eligible for review. Thirty cases were new patients. The patients' age ranged between 3 months to 16 years. Nineteen patients were female and 16 were male. Only 14 patients came back for at least 6 months of regular follow-up. Three patients moved to other hospitals, 10 were lost to follow-up, 3 new patients had not reached 6 months of follow-up at the end of the study period, and 5 died during the study period.

Table 1 shows the various etiologies of UI; myelodysplasia being the most prevalent (15/35).

TABLE 1. THE ETIOLOGY OF URINARY INCONTINENCE

Etiology	Number of patients
• Non-neuropathic bladder-sphincter dysfunction (n=4)	
- Urge incontinence	2
- Lazy bladder syndrome	1
- Hinman's syndrome	1
• Neuropathic bladder-sphincter dysfunction (n=22)	
- Myelodysplasia	15
- Sacrococcygeal tumor	2
- Leukemia infiltration	2
- Neuroblastoma	1
- IDDM with bladder-sphincter dysfunction	1
- Sacral malformation	1
• Structural incontinence (n=9)	
- Posterior urethral valve	4
- Bladder tumor	3
- Ectopic ureter	1
- Urethral stenosis	1

The clinical profile of the patients is shown in **Table 2**. Wetting, urinary tract infection, and polakysuria were the chief complaints of patients with neuropathic bladder-sphincter dysfunction, structural incontinence, and non-neuropathic bladder-sphincter dysfunction, respectively. Renal sonography as an initial examination was done on 33 out of 35 cases. VCUG was performed when there was history of recurrent UTI and suspicion of organic causes. Four patients underwent abdominal CT scan and 2 underwent plain photo of the spine to evaluate the underlying disease of UI.

Renal function deterioration was found in most patients (28/35) i.e., impaired renal function in 11/35, renal insufficiency in 6/35, renal failure in 19/35 and end-stage renal failure in 1/35. Other complications included UTI (31/35), hydronephrosis (21/35), VUR (11/35) and renal scarring (1/35). As for treatment, physiotherapy was performed on 3 patients with non-neuropathic bladder-sphincter dysfunction, clean intermittent catheterization (CIC) was done in 13 patients i.e., 12 with neuropathic bladder-sphincter dysfunction and 1 with non-neuropathic bladder-sphincter dysfunction. Surgical interventions were performed on 5 patients i.e., cystostomy in 3 patients with bladder tumors, bladder augmentation in 1 patient with sacral

TABLE 2. CLINICAL PROFILE OF PATIENTS

Profile	Non-neuropathic bladder-sphincter dysfunction (n=4)	Neuropathic bladder-sphincter dysfunction (n=22)	Structural incontinence (n=9)	Total (n=35)
Age: Range	2 yr 6 mo - 10 yr	1 yr 4 mo – 16 yr	3 mo – 10 yr	3 mo - 6 yr
Mean	4 yr 10 mo	5 yr 7 mo	3 yr 9 mo	
Median	3 yr 5 mo	5 yr 6 mo	3yr 6 mo	
Ratio ♂	1: 3	1: 2.1	8 : 1	1: 1.2
Clinical manifestations	<ul style="list-style-type: none"> • Polakysuria • UTI 	<ul style="list-style-type: none"> • Wetting • Dribbling • UTI 	<ul style="list-style-type: none"> • UTI • Urgency 	
Diagnostic examinations	<ul style="list-style-type: none"> • USG • VCUG 	<ul style="list-style-type: none"> • USG • VCUG 	<ul style="list-style-type: none"> • USG • VCUG 	
Complications				
- UTI	2/4	22/22	7/9	31/35
- Renal function deterioration	2/4	18/22	8/9	28/35
- Hydronephrosis	-	13/22	8/9	21/35
- VUR	-	8/22	3/9	11/35
Treatment	<ul style="list-style-type: none"> • Physiotherapy • CIC • Antibiotics 	<ul style="list-style-type: none"> • CIC • Indwelling catheter • Antibiotics 	<ul style="list-style-type: none"> • Indwelling catheter • Surgery • Antibiotics 	<ul style="list-style-type: none"> • CIC • Indwelling catheter • Antibiotics

malformation and internal urethrotomy in 1 patient with posterior urethral valve.

Evaluation of the 14 patients who completed 6 months of follow-up is shown in **Table 3**. From 35 urine samples, 17 patients had positive culture, but of these, 10 were asymptomatic. *Escherichia coli* was the most common pathogen (9/17) followed by *Pseudomonas sp.* (5/17), *Klebsiella pneumoniae*, *Enterobacter aerogenes* and *Streptococcus anhemolyticus*, respectively. One patient died after 8 months of follow-up due to rhabdomyosarcoma of the bladder.

Discussion

During this study, 30 new UI cases were collected with an average of 7 cases annually. Compared to previous studies, the higher number of patients might be due to the increasing attention on such cases among doctors. The wide age range reflected the various etiologies of UI. Myelodysplasia (15 cases) was most prevalent, especially in patients with neuropathic bladder-sphincter dysfunction. Similar findings were reported by Cass *et al*,¹⁷ in which 78% of neuropathic bladder-sphincter dysfunction were due to myelodysplasia. Since

the incidence of myelodysplasia (1:1000) was high, the incidence of neuropathic bladder-sphincter dysfunction must also be high. Posterior urethral valve (PUV), found in 4 cases, was the second common cause, and the diagnosis was established at the age of 3 months, 1, 3, and 6 years consecutively. Other studies also reported various ages of onset, but it was usually under the age of one year.¹⁸⁻²¹ Clinical manifestations of UI in our study varied due to the various grades of obstruction. Neuropathic bladder-sphincter dysfunctions due to spinal tumors were identified in five cases. Some literature state that micturition disorders were a common symptom found in children with spinal tumors.^{22,23} Bladder dysfunction should be suspected in patients with spinal tumors.

Wetting was the most prominent clinical manifestation of neuropathic bladder-sphincter dysfunction, followed by dribbling and fever due to secondary infection. In non-neuropathic bladder-sphincter dysfunction, the most prominent clinical manifestation was polakysuria.

The majority of patients (28/35) had had renal dysfunction since their first visit. It reflected that most patients came very late for medical help. The presence of vesicoureteral reflux associated with recur-

TABLE 3. CHARACTERISTICS OF 14 PATIENTS WHO COMPLETED 6 MONTHS OF REGULAR FOLLOW-UP

Variable	Patient number													
	1	2	3	4	5	6	7	8	9	10	11	12	13	14
Sex	F	F	M	F	F	M	F	M	F	M	F	M	F	F
Age	3.00	5.06	7.00	5.03	5.02	6.03	4.10	2.06	1.05	3.02	3.07	3.06	3.07	2.00
Diagnosis	HS	SB	SB	SB	SB	PUV	SB	LB	SM	PUV	SB	BT	SB	SB
Treatment	Phys	CIC	CIC	Phys	CIC	IU	CIC	CIC	B.aug	Cath	CIC	Cys	CIC	CIC
Early complication	UTI	UTI CRF	UTI CRI	UTI VUR IRF	UTI VUR	UTI CRI	UTI	UTI CRI	UTI VUR IRF	UTI VUR CRI	UTI VUR CRF	UTI CRF	UTI CRF	UTI CRF
Duration of FU (month)	24	6	8	35	13	25	20	21	29	16	7	8	19	11
Follow up				VUR (-)					VUR (-)					
GFR	↑	↑	↑	↑	↑	↑	↑	↑	↑	↑	↑	↑	↑	↑
Urine culture (positive/ total)*	-/4	1/2	1/2	1/1	1/1	1/5	5/7	2/2	2/3	1/1	2/2	1/2	1/3	1/1
Final or terminal complication(s)	-	UTI	CRF Perineph Absces	-	-	VUR UTI	UTI	UTI	-	-	-	UTI	-	UTI

HS = Hinman syndrome, SB = spina bifida, PUV = posterior urethral valve, LB = lazy bladder, SM = sacrum malformation, BT = bladder tumor, Phys = physiotherapy, CIC = clean intermittent catheterization, IU = internal urethrotomy, B.aug = bladder augmentation, Cath = indwelling catheter, Cys = cystostomy, UTI = urinary tract infection, CRF = chronic renal failure, CRI = chronic renal insufficiency, IRF = impaired renal function, Perineph Absces = perinephric absces

*Positive=bacterial count >100,000 cfu/ml, total = total urine culture.

rent infection potentially increased the risk of renal damage, as mentioned by Van Gool *et al.*²⁴ Both VUR and hydronephrosis are conditions associated with myelodysplasia.²⁵⁻²⁸ In our study, of 15 cases with myelodysplasia, VUR was detected in 7 and hydro-nephrosis found in 11.

Renal scars occurred in one patient, as detected by dimercaptosuccinic acid (DMSA) examination. Cohen *et al.*²⁵ reported 28 renal scars among 180 (15,5%) patients with myelodysplasia. However, we could not report the prevalence of renal scars in our study because only one patient underwent DMSA examination.

Clean intermittent catheterization was applied in 12/22 patients with neuropathic bladder-sphincter dysfunction, indwelling urethral catheters were used in patients with structural incontinence, while physiotherapy was given to non-neuropathic bladder-sphincter dysfunction patients. Cystostomy was done on three patients with bladder tumors (structural incontinence), internal urethrotomy on one patient with posterior urethral valve, and bladder augmentation in one patient with sacrum malformation. Different modes of treatment were given to three categories of UI classification in this study, indicating that the different choices of

treatment were not based on the treatment procedure according to the UI classification. Each case was treated with an individual approach based on the patient's clinical condition and the parents' preference. For instance, although CIC is known as the best choice for neuropathic bladder-sphincter dysfunction, some parents or children, especially boys, refused it because of the inconvenience it caused.

During 6 month follow-up of the 14 patients, increased GFR was found in 12 patients. This phenomenon is known as an "acute on chronic condition". Since most cases were already superimposed with intractable urinary infection, the acute state worsened renal function, but proper treatment of infection could improve renal function.

Bacteriuria occurred frequently following the CIC procedure. Among 22 urine specimens, 9 had no bacterial growth or <100.000 colony-forming units/ml. *Escherichia coli* was the most (6/13) prevalent pathogenic bacteria detected. Ottolini *et al.*²⁹ reported that 207 patients with neuropathic bladder-sphincter dysfunction who used CIC for a mean duration of 6 years, 85% had one or more episodes of untreated asymptomatic bacteriuria, and 35% had one or more febrile episodes associated with positive urine culture results.

The most prevalent pathogen was *E.coli*. Similar findings were reported by Schlager *et al*³⁰ and Szucs *et al*.³¹

No complication of CIC was found in this study, as similarly reported by Cass *et al*.³² But CIC procedure with incorrect technique might lead to difficulty in passing the catheter, resulting in a sore urethra, swelling of the penile, urethral bleeding, and urethral discharge. Urethral false passage as a complication of CIC had been reported by Koleilat *et al*.³³

In conclusion, myelodysplasia was the most prevalent underlying cause of UI. Most patients had renal insufficiency or renal failure since the first visit, reflecting a long time of relapse before the patients seek medical help.

References

1. Atala A, Bauer SB. Bladder dysfunction. In: Barratt TM, Avner ED, Harmon WE, editors. Pediatric Nephrology. 4th ed. Baltimore: Lippincott William &Wilkins; 1999. p. 913-31.
2. Bauer SB. Neurogenic bladder dysfunction. In: Edelmann CM, Bernstein J, Meadow SR, Spitzer A, Travis LB, editors. Pediatric kidney disease. 2nd ed. Boston: Little, Brown and Company; 1992. p. 2085-109.
3. Varlam DE, Dippell J. Non-neurogenic bladder and chronic renal insufficiency in childhood. *Pediatr Nephrol* 1995;9:1-5.
4. Rushton HG. Enuresis. In: Kher KK, Makker SP, editors. Clinical pediatric nephrology. New York: Mc Graw-Hill; 1992. p. 339-419.
5. Mundy AR. The neuropathic bladder. In: Postlethwaite RJ, editor. Clinical paediatric nephrology. 2nd ed. Oxford: Butterworth-Heinemann; 1994. p. 319-33.
6. Van Gool JD. Disorders of micturition. In: Postlethwaite RJ, editor. Clinical paediatric nephrology. 2nd ed. Oxford: Butterworth-Heinemann; 1994. p. 59-74.
7. Van Gool JD, Bloom DA, Buttler RJ, Djurhuus JC, Hjalmas K, De Jong TPVM *et al*. Conservative management in children. In: Abrams P, Khoury S, Wein A, editors. Incontinence. Proceedings of the 1st International Consultation on Incontinence; 1998 June 28- July 1; Monaco. p. 487-549.
8. Homma Y, Batista JE, Bauer SB, Griffiths DJ, Hilton P, Kramer G *et al*. Urodynamic. In: Abrams P, Khoury S, Wein A, editors. Incontinence. Proceedings of the 1st International Consultation on Incontinence; 1998 June 28- July 1; Monaco. p. 351-99.
9. Hunskaar S, Arnold EP, Burgio K, Diokno AC, Herzog AR, Mallett VT. Epidemiology and natural history of urinary incontinence. Incontinence. Proceedings of the 1st International Consultation on Incontinence; 1998 June 28- July 1; Monaco. p. 197-226.
10. Tanago EA. Urinary incontinence. In: Tanago EA, Mc Aninch JW, editors. Smith General Urology. 14th ed. California: Appleton and Lange; 1995. p. 536-51.
11. Tambunan T. Inkontinensia urin pada anak. *Sari Pediatri* 2000;2:163-9.
12. International Reflux Study Committee: Medical versus surgical treatment of primary vesicoureteral reflux: a prospective international reflux study in children. *J Urol* 1981;125:237-8.
13. Sekarwana N, Rachmadi D, Hilmanto D. Gagal ginjal kronik. In: Alatas H, Tambunan T, Trihono PP, Pardede SO, editors. Buku ajar nefrologi anak. 2nd ed. Jakarta: Balai Penerbit Fakultas Kedokteran Universitas Indonesia; 2002. p. 509-30.
14. Ridgen SPA. Chronic renal failure. In: Postlethwaite, editor. Clinical pediatric nephrology. 2nd ed. Oxford: Butterworth Heinemann; 1994. p. 266-81.
15. Schwartz GJ, Haycock GB, Edelman CM, Spitzer A. A simple estimate of glomerular filtration rate in children derived from body length and plasma. *Pediatrics* 1976;58:259-63.
16. Walker WA, Hendricks KM. Pediatric nutrition. Philadelphia: WB Saunders; 1985.
17. Cass AS, Luxenberg M, Johnson CF, Gleich P. Management of the neurogenic bladder in 413 children. *J Urol* 1984;132:521-5.
18. Pereira PL, Espinosa L, Urrutina MJM, Navarro RLM, Jaureguizar E. Posterior urethral valves: prognostic factors. *Int Braz J Urol* 2003;91:687-90.
19. Pereira PL, Urrutina MJM, Espinosa L, Navarro RLM, Jaureguizar E. Bladder dysfunction as a prognostic factor in patient with posterior urethral valves. *Int Braz J Urol* 2002;90:308-11.
20. Onuora VC, Mirza K, Koko AH, Al Turki M, Meabed AH, Al Jawini N. Prognostic factors in Saudi children with posterior urethral valves. *Ped Nephrol* 2000;14:221-3.
21. Connor JP, Burdige KA. Long term urinary continence and renal function in neonates with posterior urethral valves. *J Urol* 1990;144:1209-11.
22. Soler D, Borzyskowski M. Lower urinary tract dysfunction in children with central nervous system tumours. *Arch Dis Child* 1998;79:344-47.

23. Dincer F, Dincer C, Baskaya MK. Results of combined treatment of paediatric intraspinal tumours. *Paraplegia* 1992;30:718-28.
24. Van Gool JD, Hjalmas K, Tamminen-Mobius T, Olbing H. Historical clues to the complex of dysfunctional voiding, urinary tract infection and vesicoureteral reflux - The International Reflux Study in Children. *J Urol* 1992;148:1699-1702.
25. Cohen RA, Rushton HG, Belman AB, Kass EJ, Majd M, Shaer C. Renal scarring and vesicoureteral reflux in children with myelodysplasia. *J Urol* 1990;144:541-4.
26. Kaplan WE, Firlit CF. Management of reflux in the myelodysplastic child. *J Urol* 1983;129:1195-7.
27. Sidi AA, Peng W, Gonzalez R. Vesicoureteral reflux in children with myelodysplasia natural history and results of treatment. *J Urol* 1986;136:329-31.
28. Rushton HG, Majd M. Dimercaptosuccinic acid renal scintigraphy for the evaluation of pyelonephritis and scarring: a review of experimental and clinical studies. *J Urol* 1992;148:1726-32.
29. Ottolini MC, Shaer CM, Rushton HG, Majd M, Gonzalez EC, Patel KM. Relationship of asymptomatic bacteriuria and renal scarring in children with neuropathic bladders who are practicing clean intermittent catheterization. *J Pediatr* 1995;127:368-72.
30. Schlager TA, Dilks S, Trudell J, Whittam TS, Hendley O. Bacteriuria in children with neurogenic bladder treated with intermittent catheterization: Natural history. *J Pediatr* 1995;126:490-6.
31. Szucs K, O'Neil KM, Faden H. Urinary findings in asymptomatic subjects with spina bifida treated with intermittent catheterization. *Pediatr J Infect Dis* 2001;20:638-9.
32. Cass AS, Luxenberg M, Gleich P, Johnson CF, Hagen S. Clean intermittent catheterization in the management of the neurogenic bladder in children. *J Urol* 1984;132:526-8.
33. Koleilat N, Sidi AA, Gonzalez R. Urethral false passage as a complication of intermittent catheterization. *J Urol* 1989;142:1216-7.