



Assessment of Cases of Congenital Obstructive Uropathy in Children

Harshindar Kaur^{1*}, Sukhmani Kaur², Sanjana Saholi³, Pehal Goyal⁴

¹Associate Professor,
Email: drharshpatiala@yahoo.com,
Orcid ID: 0000-0002-0913-7068

²PCMS 1,
Email: sukhmani866@gmail.com,
Orcid ID: 0000-0002-2418-0830

³PCMS 1, Junior Resident,
Email: doc.sanjana.singla@gmail.com,
Orcid ID: 0000-0002-9737-5129

⁴Senior Resident,
Email: pehalgoel@gmail.com,
Orcid ID: 0000-0002-4421-8728

*Corresponding author

Received: 12 January 2022
Revised: 13 February 2022
Accepted: 24 February 2022
Published: 22 April 2022

Abstract

Background: The aim is to assess cases of congenital obstructive uropathy in children. **Material & Methods:** Sixty-five children in age ranged 5-12 years of either gender with congenital obstructive uropathy were recruited for the study. In all cases, ultrasonography was done in all cases. Growth of the child was monitored and height standard deviation score (Ht-SDS) was calculated annually. Glomerular filtration rate (ml/min/1.73 m²) was calculated. **Results:** Out of 65 patients, males were 40 and females were 25. Etiology found to be ureteropelvic junction obstruction (PUJO) in 12, posterior urethral valve (PUV) in 14, PUJO + PUV in 30 and obstructive megaureter in 9 cases. Symptoms were fever in 34, pain abdomen in 20, burning micturition in 45, poor urinary stream in 21 and flank pain in 15 cases. Treatment given was pyeloplasty in 14, nephrectomy in 20, PUV fulguration in 11, ureterocele decompression in 12 and ureteric reimplantation in 8 cases. **Conclusions:** Most common etiologies for congenital obstructive uropathy in children were congenital uretero-pelvic junction obstruction and posterior urethral valve. Male preponderance was seen with burning micturition.

Keywords:- Congenital Obstructive Uropathy, Children, Posterior Urethral Valve.

INTRODUCTION

Among various causes of chronic kidney disease (CKD) in children, congenital obstructive uropathy is important one. The occurrence of urinary tract dilatation and obstructive lesion is indicative of obstructive uropathy.^[1,2] It is evident that 75% of cases of CKD in children are due to obstructive uropathy. Structural anomalies of the urinary tract and steroid resistant nephrotic syndrome constitute the major portion of patients with CKD.^[3]

Urinary tract obstruction may be due to congenital (anatomic) lesion or is caused by

acquired such as trauma, neoplasia, calculi, inflammation or surgical procedures.^[4] It is seen that maximum childhood obstructive lesions are congenital. Obstructive lesion can occur at any region from the calyces to the tip of urethra.^[5] Ureter can be obstructed by external compression or kinks at any site along its course in the retroperitoneum pelvi-ureteric & uterovesical junction & where it crosses the iliac artery. Similarly, the urethra is vulnerable to obstructive pathology at the internal and external meatus.^[6] Various imaging modalities are available that helps in diagnosis of congenital obstructive uropathy. Radionuclide procedures also contribute in diagnosis.^[7]

Ultrasonography, radiocontrast studies, magnetic resonance (MR) imaging and MR urography and radionuclide scintigraphy are widely used imaging modalities. Endoscopy can be diagnostic as well therapeutic.^[8,9] Considering this, we selected present study with the aim to assess congenital obstructive uropathy in children.

MATERIAL AND METHODS

A total of sixty- five children in age ranged 5-12 years of either gender with congenital obstructive uropathy were recruited for the study. The study was approved from ethical clearance and review board. Parental consent was obtained in every case. Children with other major systemic malformations like congenital heart disease, gastrointestinal malformations, syndromic associations were excluded.

A thorough clinical and physical examination was carried. Laboratory investigations such as serum creatinine and urinalysis were performed at admission and every follow- up visit. Urine culture and sensitivity was done in

the presence of pyuria. Proteinuria was calculated with urine protein was $\geq 1+$. In all cases, ultrasonography was done in all cases. Growth of the child was monitored and height standard deviation score (Ht-SDS) was calculated annually. Interval dimercaptosuccinic acid (DMSA) scan was done in recurrent urinary tract infections (UTI). Glomerular filtration rate (ml/min/1.73 m²) was calculated. Results of the study was analysed using Mann Whitney U test. The level of significance was set below 0.05.

RESULTS

Out of 65 patients, males were 40 and females were 25. Etiology found to be ureteropelvic junction obstruction (PUJO) in 12, posterior urethral valve (PUV) in 14, PUJO + PUV in 30 and obstructive megaureter in 9 cases. Symptoms were fever in 34, pain abdomen in 20, burning micturition in 45, poor urinary stream in 21 and flank pain in 15 cases. A significant difference was found ($P < 0.05$) [Table 1, Figure 1].

Table 1: Baseline characteristics.

Parameters	Variables	Number	P value
Gender	Male	40	<0.05
	Female	25	
Etiology	Ureteropelvic junction obstruction (PUJO)	12	<0.05
	Posterior urethral valve (PUV)	14	
	PUJO + PUV	30	
	Obstructive megaureter	9	
Symptoms	Fever	34	<0.05
	Pain abdomen	20	
	Burning micturition	45	
	Poor urinary stream	21	
	Flank pain	15	

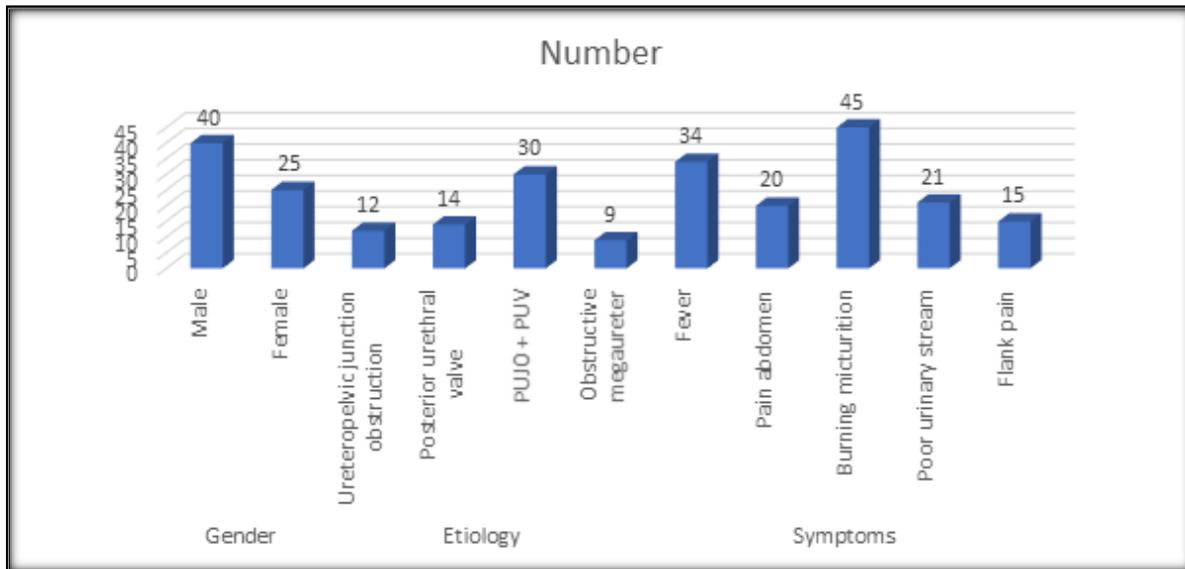


Figure 1

Table 2: Treatment given in children

Treatment	Percentage	P value
Pyeloplasty	14	>0.05
Nephrectomy	20	
PUV fulguration	11	
Ureterocele decompression	12	
Ureteric reimplantation	8	

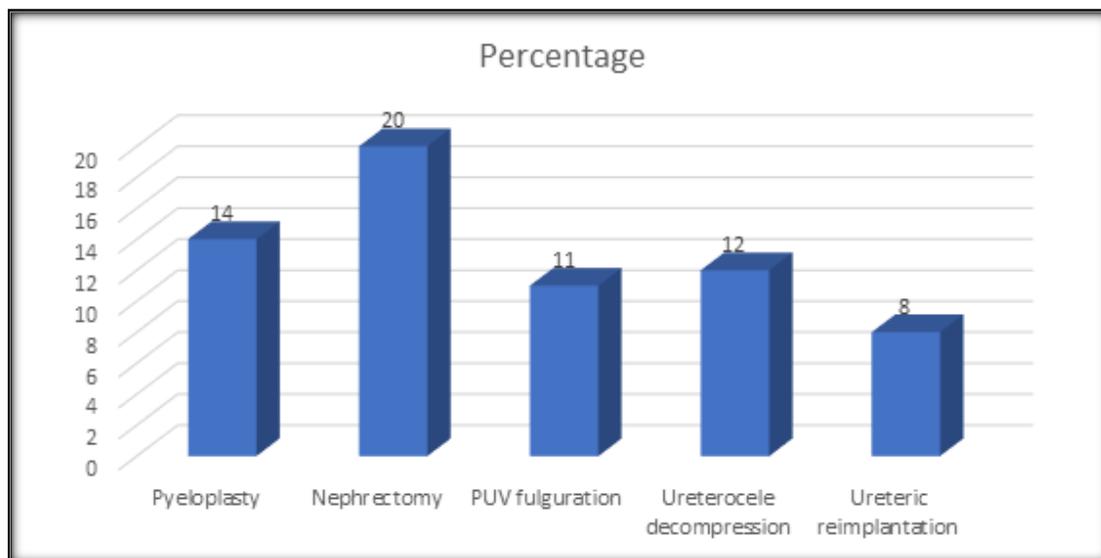


Figure 2

Table 3: Renal parameters

Parameters	Variables	At diagnosis	At follow up		
			Improved	Maintained	Faltered
Ht. SDS at diagnosis	0 to +1SD	30	2	28	-
	0 to -1SD	15	-	13	2
	-1 SD to -2 SD	12	2	8	2
	< -2SD	8	3	4	1
Hypertension eGFR (ml/min/1.73m ²)	>90	6	38		
	60-89	8	15		
	45-59	11	8		
	30-44	14	2		
	15-29	26	1		
	<15	-	1		

Treatment given was pyeloplasty in 14, nephrectomy in 20, PUV fulguration in 11, ureterocele decompression in 12 and ureteric reimplantation in 8 cases. The difference was non-significance ($P > 0.05$) [Table 2, Figure 2].

Ht. SDS at diagnosis between 0 to +1SD was seen among 30 which was maintained in 28 at followed up. 0 to -1SD was seen in 15 which was maintained in 13 at follow up. Hypertension with eGFR >90 was seen in 6 and improvement was seen in 38 at follow up [Table 3].

DISCUSSION

In children, pyelonephritis can occur due to urinary stasis. An upper urinary tract stone can cause abdominal or flank pain and hematuria.^[10,11] With bladder outlet obstruction, the urinary stream may be weak, dribbling of urine, straining, retention and palpable bladder; urinary tract infection is common.^[12] Flank pain, nausea, vomiting may occur due to acute obstruction. Chronic obstruction can be silent or can cause vague abdominal pain with increased fluid intake.^[13] In young infant features of sepsis may be there due to pyelonephritis. Obstructive

renal insufficiency can manifest itself by failure to thrive, vomiting, diarrhoea or other non-specific signs and symptoms.^[14,15] We selected present study with the aim to assess congenital obstructive uropathy in children.

Our study comprised of 65 patients. Males were 40 and females were 25. Etiology found to be ureteropelvic junction obstruction (PUJO) in 12, posterior urethral valve (PUV) in 14, PUJO + PUV in 30 and obstructive megaureter in 9 cases. Symptoms were fever in 34, pain abdomen in 20, burning micturition in 45, poor urinary stream in 21 and flank pain in 15 cases. Tabgirala et al,^[16] conducted a study on 60 children with congenital obstructive uropathy. It was found that the most common etiologies was congenital uretero-pelvic junction obstruction followed by posterior urethral valve. In 88.3% males were involved and in 36.6%, poor urinary stream was the most common complaint. 40% patients had elevated creatinine level in urine. 15% were hypertensive and 25% had growth failure at diagnosis. However, there was a reduction in the number of children with poor estimated glomerular filtration rate (eGFR), hypertension and growth

faltering during follow-up. Among the risk factors, hypertension at diagnosis and p value < 60 ml/min/1.73m²) had more height faltering and hypertension at follow-up.

We observed that treatment given was pyeloplasty in 14, nephrectomy in 20, PUV fulguration in 11, ureterocele decompression in 12 and ureteric reimplantation in 8 cases. Bomalaski et al,^[17] revealed that the prognosis was better in children with PUV when presented before 1 years of age. Tapia et al,^[18] in their study observed that improvement in renal function was significantly more following pyeloplasty performed before 1 year of age and not in older children. Uthup et al,^[19] had demonstrated a statistically significant association between intervention beyond 28 days of life in children with PUV and poor eGFR at follow-up.

We found that Ht. SDS at diagnosis between 0 to +1SD was seen among 30 which was maintained in 28 at followed up. 0 to -1SD was

seen in 15 which was maintained in 13 at follow up. Hypertension with eGFR >90 was seen in 6 and improvement was seen in 38 at follow up. Pelviureteric junction (PUJ) obstruction is due to functional obstruction of junction between the renal pelvis and ureter. 10 It is the most common cause of hydronephrosis with an incidence of 1 in 2000 children,¹² with a male female ratio 3:1 & bilateral in 20-25% of cases. This could be due to intrinsic abnormality, muscular abnormalities of the ureter, ureteral polyps, ureteral folds, crossing vessels & rarely secondary to VUR. Children may present with abdominal pain (dull constant pain or severe spasmodic pain), lump or UTI.^[20]

CONCLUSIONS

Results of the study showed that most common etiologies for congenital obstructive uropathy in children were congenital uretero-pelvic junction obstruction and posterior urethral valve. Male preponderance was seen with burning micturition.

REFERENCES

1. Mayor G, Genton N, Torrado A, Guignard J-P. Renal function in obstructive nephropathy: long-term effect of reconstructive surgery. *Pediatrics*. 1975;56:740.
2. Schwartz GJ, Feld LG, Langford DJ. A simple estimate of glomerular filtration rate in full-term infants during the first year of life. *J Pediatr*. 1984;104:849-54.
3. Mirshemirani A, Khaleghnejad A, Rouzrokh M, Sadeghi A, Mohajezadeh L, Sharifian M. Posterior urethral valves; a single center experience. *Iran J Pediatr*. 2013;23:531-5.
4. Drozd D, Drozd M, Gretz N, Möhring K, Mehls O, Schäfer K. Progression to end-stage renal disease in children with posterior urethral valves. *Pediatr Nephrol*. 1998;12(8):630-6. doi: 10.1007/s004670050517.
5. Betts PR, Magrath G. Growth pattern and dietary intake of children with chronic renal insufficiency. *Br Med J*. 1974;2(5912):189-93. doi: 10.1136/bmj.2.5912.189.
6. Ylinen E, Ala-Houhala M, Wikström S. Prognostic factors of posterior urethral valves and the role of antenatal detection. *Pediatr Nephrol*. 2004;19(8):874-9. doi: 10.1007/s00467-004-1474-4.
7. Parkhouse HF, Barratt TM, Dillon MJ, Duffy PG, Fay J, Ransley PG, Woodhouse CR, Williams DI. Long-term outcome of boys with posterior urethral valves. *Br J Urol*. 1988;62(1):59-62. doi: 10.1111/j.1464-410x.1988.tb04267.x.
8. Gulati S, Mittal S, Sharma RK, Gupta A. Etiology and outcome of chronic renal failure in Indian children.



- Pediatr Nephrol. 1999;13(7):594-6. doi: 10.1007/s004670050750.
9. Warady BA, Chadha V. Chronic kidney disease in children: the global perspective. *Pediatr Nephrol.* 2007;22(12):1999-2009. doi: 10.1007/s00467-006-0410-1.
 10. Kanitkar M. Chronic Kidney Disease in Children: An Indian Perspective. *Med J Armed Forces India.* 2009;65(1):45-49. doi:10.1016/S0377-1237(09)80055-5
 11. Hari P, Singla IK, Mantan M, Kanitkar M, Batra B, Bagga A. Chronic renal failure in children. *Indian Pediatr.* 2003;40:1035-42.
 12. Hogg RJ, Furth S, Lemley KV, Portman R, Schwartz GJ, Coresh J, et al; National Kidney Foundation's Kidney Disease Outcomes Quality Initiative. National Kidney Foundation's Kidney Disease Outcomes Quality Initiative clinical practice guidelines for chronic kidney disease in children and adolescents: evaluation, classification, and stratification. *Pediatrics.* 2003 Jun;111(6 Pt 1):1416-21. doi: 10.1542/peds.111.6.1416.
 13. Woolf AS, Thiruchelvam N. Congenital obstructive uropathy: its origin and contribution to end-stage renal disease in children. *Adv Ren Replace Ther.* 2001;8(3):157-63. doi: 10.1053/jarr.2001.26348.
 14. Roth KS, Carter WH Jr, Chan JC. Obstructive nephropathy in children: long-term progression after relief of posterior urethral valve. *Pediatrics.* 2001;107(5):1004-10. doi: 10.1542/peds.107.5.1004.
 15. Prem AR. Common paediatric problems. *BMJ.* 2006;333(7566):486-489. doi:10.1136/bmj.333.7566.486
 16. Tangirala S, Bhaskaranand N, Kini PG, Konda KC, Gajjala ST. Clinical Profile and Outcome of Children with Congenital Obstructive Uropathy. *Indian J Pediatr.* 2019;86(4):354-359. doi: 10.1007/s12098-019-02876-w.
 17. Bomalaski MD, Anema JG, Coplen DE, Koo HP, Rozanski T, Bloom DA. Delayed presentation of posterior urethral valves: a not so benign condition. *J Urol.* 1999;162(6):2130-2. doi: 10.1016/s0022-5347(05)68140-2.
 18. Uthup S, Binitha R, Geetha S, Hema R, Kailas L. A follow-up study of children with posterior urethral valve. *Indian J Nephrol.* 2010;20(2):72-5. doi: 10.4103/0971-4065.65298.
 19. Tapia J, Gonzalez R. Pyeloplasty improves renal function and somatic growth in children with ureteropelvic junction obstruction. *J Urol.* 1995;154(1):218-22.
 20. Makino Y, Kobayashi H, Kyono K, Oshima K, Kawarabayashi T. Clinical results of fetal obstructive uropathy treated by vesicoamniotic shunting. *Urology.* 2000;55(1):118-22. doi: 10.1016/s0090-4295(99)00403-3.
- Source of Support: Nil, Conflict of Interest: None declared