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ORIGINAL ARTICLE

Cases of Congenital Obstructive Nephropathy in Children- A Clinical Study

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***ABSTRACT*:**
Background: The present study recorded the cases of congenital obstructive nephropathy in children. **Materials & Methods:** The present study was conducted on 46 children age ranged 1- 10 years of both genders. Etiology and clinical features were recorded in all patients. **Results:** Out of 46, boys were 26 and girls were 20. Congenital uretero-pelvic junction obstruction was etiologic agent in 32 and posterior urethral valve in 14. The difference was significant (P< 0.05). Hypertension (23) and UTI (14) were predictors for development of chronic kidney disease in children. **Conclusion:** Authors found that hypertension and frequent UTI were observed to be strongly associated with progression of CKD.
Key words: Congenital obstructive nephropathy, Hypertension, UTI

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INTRODUCTION

Maldevelopment of the collecting system resulting in urinary tract obstruction (UTO) is the leading identifiable cause of CKD in children. Specific etiologies are unknown; most cases are suspected by discovering hydronephrosis on prenatal ultrasonography.[1] Congenital UTO can reduce nephron number and cause bladder dysfunction, which contribute to ongoing injury. Severe UTO can impair kidney growth in utero, and animal models of unilateral ureteral obstruction show that ischemia and oxidative stress cause proximal tubular cell death, with later development of interstitial fibrosis.[2] Congenital obstructive nephropathy therefore results from combined developmental and obstructive renal injury. Due to inadequacy of available biomarkers, criteria for surgical correction of upper tract obstruction are poorly established. Lower tract obstruction requires fetal or immediate postnatal intervention, and the rate of progression of CKD is highly variable.[3] Obstruction of the urinary tract generally causes hydronephrosis. Antenatal ultrasonography detects hydronephrosis in about 1 in 100 fetuses, the prevalence ranging from 0.6 to 5.4 percent depending on diagnostic criteria. The condition is bilateral in 17 to 54 percent and additional abnormalities are occasionally associated. In early development, chronic urinary tract obstruction impairs renal growth & development.[4]

Urinary tract obstruction can result from congenital (anatomic) lesion or can be caused by acquired such as trauma, neoplasia, calculi, inflammation or surgical procedures, although most childhood obstructive lesions are congenital. Obstructive lesion can occur at any point from the calyces to the tip of urethra. Ureter can be obstructed by external compression or kinks at any site along its course in the retroperitoneum.[5] The present study recorded the cases of congenital obstructive nephropathy in children.

MATERIALS & METHODS

The present study was conducted in the Pediatrics department. It comprised of 46 children age ranged 1- 10 years of both genders. The study was approved from ethical committee. Parents were informed regarding the study and written consent was obtained.

Data such as name, age, gender etc. was recorded. Etiology and clinical features were recorded in all. Results were subjected to statistical analysis. P value less than 0.05 was considered significant.

RESULTS

Table I Distribution of patients

Total- 46		
Gender	**Boys**	**Girls**
Number	26	20

Table I shows that out of 46, boys were 26 and girls were 20.

Table II Etiology in patients

Etiology	Number	P value
Congenital uretero-pelvic junction obstruction	32	0.01
Posterior urethral valve	14	

Table II, graph I shows that congenital uretero-pelvic junction obstruction was etiologic agent in 32 and posterior urethral valve in 14. The difference was significant (P< 0.05).

Graph I Etiology in patients

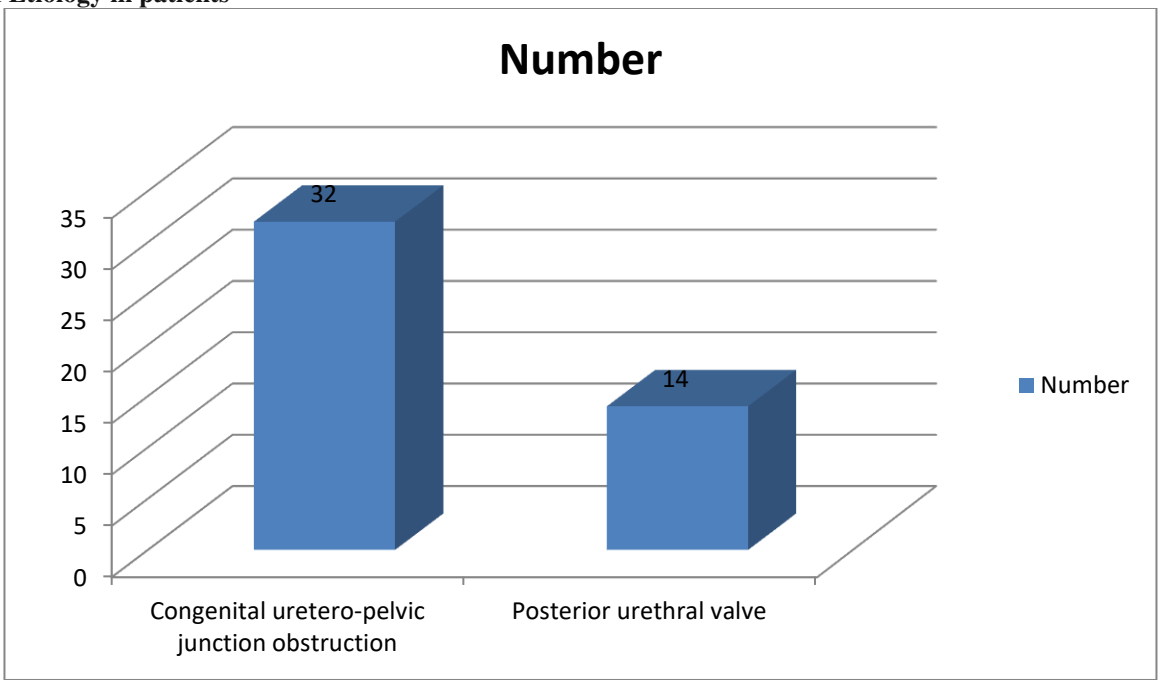

Table III Predictors for development of chronic kidney disease

Predictor	Number	P value
Hypertension	23	0.04
UTI	14	

Table III, graph II shows that hypertension (23) and UTI (14) were predictors for development of chronic kidney disease in children.

DISCUSSION

The effects of obstruction anywhere in the urinary tract are predictable. Obstruction in the urethra, the most distal part of urinary tract, results in dilatation of the proximal urethra.[6] The bladder in an attempt to overcome the obstruction undergoes hypertrophy manifested by thickening of bladder wall, trabeculation, sacculation and hypertrophy of bladder neck. The intravesical pressure increases causing decreased emptying of ureter into bladder and reflux of urine into the ureter. The resultant effect is dilatation of ureter, which also undergoes hypertrophy.[7] The intraureteric pressure rises & back pressure lead to hydronephrosis and thinning of renal parenchyma.

Stasis of urine in the dilated tract results in repeated infections. The combination of stasis, repeated infections and high intraluminal pressure results in renal parenchymal scarring and progressive chronic kidney disease, suggested by proteinuria and hypertension. The resulting impairment of renal function is detrimental to normal growth and development, and may be life threatening.[8] The present study recorded the cases of Congenital obstructive nephropathy in children.

In present study, out of 46, boys were 26 and girls were 20. Congenital uretero-pelvic junction obstruction was etiologic agent in 32 and posterior urethral valve in 14. The difference was significant (P< 0.05). Becker et al[9] studied the etiology and clinical profile of congenital obstructive uropathy in children, renal status and growth at diagnosis and at follow-up and to determine the predictors for development of chronic kidney disease (CKD). Congenital

uretero-pelvic junction obstruction followed by Posterior urethral valve were the most common etiologies identified. Male preponderance (88.3%) was observed with poor urinary stream being the most common presentation (36.6%). Forty percent of the population had elevated creatinine. Fifteen percent 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 [O.R-12.8 (2.21-74.22) and p value <0.05] and frequent urinary tract infection (UTI) [O.R-14.06 (2.32-85.42) and p value <0.05] were the most important factors for CKD progression. Children with low eGFR (< 60 ml/min/1.73m2) had more height faltering and hypertension at follow-up (p value <0.05).

We found that hypertension (23) and UTI (14) were predictors for development of chronic kidney disease in children. The clinical feature in most of the patient are due to consequences of the obstruction. Obstruction of the urinary tract generally causes hydronephrosis, which is typically asymptomatic in its early phase. An obstructed kidney secondary to aureteropelvic junction or ureterovesical junction obstruction can manifest as mass or cause upper abdominal or flank pain on affected side. Pyelonephritis can occur because of urinary stasis. An upper urinary tract stone can cause abdominal or flank pain and hematuria. With bladder outlet obstruction, the urinary stream may be weak, dribbling of urine, straining, retention and palpable bladder; urinary tract infection is common. An acute obstruction may cause flank pain, nausea, vomiting. Chronic obstruction can be silent or can cause vague abdominal pain with increased fluid intake. 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.[10]

CONCLUSION

Authors found that hypertension and frequent UTI were observed to be strongly associated with progression of CKD.

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