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Sonographic assessment of the effect of vesicoureteral reflux and urinary tract infections on growth of the pediatric solitary kidney

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Summary

Introduction—Perusal of recent guidelines relating to proper evaluation of infants and children with urinary tract infection (UTI) suggests that the occurrence of vesicoureteral reflux (VUR) may not have the clinical import previously ascribed to this anatomic abnormality. Patients with a solitary kidney uniquely allow investigation of the effects of both vesicoureteral reflux (VUR) and urinary tract infection (UTI) on renal growth unencumbered with the inevitable questions of laterality that confound analysis in patients with two kidneys. Several previous studies with conflicting results have addressed whether vesicoureteral reflux (VUR) impacts ultimate renal size in children with a solitary kidney. Few published studies have considered the occurrence of both urinary tract infection (UTI) and VUR on the degree of compensatory hypertrophy. This is the largest series to date investigating the effect of both UTI and VUR on the degree of compensatory hypertrophy with time.

Objective—Our objective was to analyze sonographically determined renal growth in patients with a solitary kidney, stratifying for both the occurrence and severity of UTIs and the occurrence and severity of VUR.

Study design—We retrospectively reviewed the clinical history (including bladder and bowel dysfunction (BBD)) and radiology reports of 145 patients identified as having either a congenital or acquired solitary kidney in our pediatric urology practice from the prior 10 years. UTIs were tabulated by severity, where possible, and the grade of VUR was recorded based on the initial cystogram. Sonographically determined renal length was tabulated for all ultrasounds obtained

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Conflict of interest

None.

throughout the study. Based on a mixed-effects model, we investigated the influence of UTI and VUR on renal growth.

Results—Of the 145 patients analyzed, 105 had no VUR and 39 had VUR (16 = Gr I&II, 11 = GIII, 12 = GIV&V). Comparison showed that there was no difference in the occurrence of UTI between those without VUR (27/105 with UTI) and those with VUR (15/39 with UTI; $p = 0.14$). There was no difference in the occurrence of BBD in patients with VUR (15/39) and those without VUR (36/106, $p = 0.62$). While neither VUR nor UTI alone affected renal growth in the solitary kidney, the three-way interaction term among age, VUR, and UTI was significant ($p = 0.016$). The growth of the kidneys in the various patient groups is depicted in the table. From the analysis, a refluxing solitary kidney with UTI showed a significantly lower growth rate than the other groups ($p < 0.001$).

Discussion—This study is limited by the inherent selection bias of retrospective studies. Additionally, the variability of sonographic renal measurement is well recognized. Lastly, our sample size did not allow us to incorporate the severity of the UTIs and the grades of VUR in our final regression model. Nevertheless, the overall patterns suggest that when both VUR and UTI are present, the solitary kidney demonstrates less renal growth with time. Study of larger cohorts of patients with solitary kidneys will be necessary to confirm our observations and discern what, if any, are the consequences of high-grade VUR and upper tract UTI in these patients.

Conclusion—In the largest series to date we were able to discern no independent effect of either VUR or UTI on sonographically determined renal growth in patients with a solitary kidney. However, UTI and VUR together result in kidneys that are smaller than other solitary kidneys not so affected. Follow-up studies of larger cohorts seem warranted to confirm these findings and discern the clinical import of these smaller kidneys.

Keywords

Solitary kidney; Vesicoureteral reflux; Urinary tract infection; Renal compensatory hypertrophy

Introduction

Group	n	Renal growth, cm/year (SE)
–VUR, –UTI	79	0.48 (0.02)
+VUR, –UTI	24	0.47 (0.04)
–VUR, +UTI	27	0.45 (0.03)
+VUR, +UTI	15	0.27 (0.04)

The relationship between urinary tract infections (UTIs), renal scarring, and vesicoureteral reflux (VUR) has been investigated in multiple studies over numerous decades [1–5]. Even though renal failure secondary to the interaction of VUR and UTI is decidedly rare, the relationship is of interest. Mainstream pediatric thought currently minimizes the importance of VUR and UTI in the development of renal scarring as evidenced by the recent guidelines for UTI published by the AAP [6]. With the widespread use of prenatal imaging, an increased number of solitary kidneys are being diagnosed neonatally and most are further evaluated with cystography. This single kidney “model” lacks the inevitable questions of

laterality regarding both infection and VUR which arise in patients with two kidneys. The solitary kidney allows direct observation of the effect of VUR and UTI on renal growth. In the largest series to date, we evaluated serial ultrasound renal length measurements in a cohort of pediatric patients with a solitary kidney in an attempt to better understand the long-term effect, if any, of VUR and UTI on renal growth and compensatory hypertrophy.

Materials and methods

After institutional review board approval was verified, records were obtained based on medical billing codes for 226 patients with the diagnosis of solitary kidney seen between 2001 and 2011 in our pediatric urology practice. Patients with spina bifida, posterior urethral valves, triad syndrome, ureterocele, unknown VUR status, incorrect diagnostic code, and no renal ultrasound data were excluded from the study. Patients with prior nephrectomy were included in the study. One-hundred and forty-five patients were identified for review. Serum creatinine values were tabulated when available and when obtained beyond the neonatal period. Age at sonographic assessment and ultrasound renal length measurements were tabulated based on reports from radiology. UTIs were tabulated if verified by positive urine culture using standard criteria. UTIs were considered upper tract if patient had documented fever with flank pain or showed evidence of systemic involvement with leukocytosis, elevated c-reactive protein, etc. Lower tract infections lacked these signs, symptoms, and laboratory findings. Poorly characterized infections were those culture positive UTIs noted in the record with no supporting data to verify or refute upper tract involvement. VUR status and grade of the solitary kidney was based on first available voiding cystourethrogram (VCUG) according to established criteria. Patients with VUR were initially placed on prophylactic antibiotics. The VUR group was further subdivided into low (grade 1 and 2), moderate (grade 3), and high (grade 4 and 5) grades for ease of statistical assessment. No patient had surgical intervention for VUR. Other clinical characteristics recorded included diagnostic circumstances of the solitary kidney and the occurrence of clinically determined bowel and bladder dysfunction. Characteristics of 145 patients were summarized by descriptive statistics (frequencies and percentages for categorical variables, and means and standard deviations for continuous variable), and were compared between patients with VUR and without VUR: Fisher's exact tests for categorical variables and Wilcoxon rank-sum tests for continuous variables. As the renal measures were repeated over time, we used a linear mixed-effects model for multivariable analysis. The model contains the fixed effects resulting from variation from main factors and the random effects from correlations of repeated measures within a patient. The renal growth rate for solitary kidneys with, versus without VUR and UTI was evaluated, adjusting for age, gender, and laterality. To investigate the joint effect of VUR and UTI in renal growth, a three-way interaction among age, VUR, and UTI status was also included in the model. All data analyses were performed in SAS 9.3 (Cary, NC, 2011). A two-tailed *p* value of less than 0.05 was regarded as statistically significant.

Results

Demographic and clinical characteristics of the 145 patients can be found in Table 1. Of the solitary kidneys, 89 (61.4%) were diagnosed prenatally and only five (3.5%) secondary to

UTI. Prior nephrectomy was present in seven (4.8%). The most common etiology of solitary kidney status was contralateral multicystic dysplastic kidney (MCDK) (60%) followed by congenital absence (34.5%) of the contralateral kidney. The median follow-up for the entire group was 3.6 years and the interquartile range (IQR) was 4.6 years. Based on initial VCUG, low-, moderate-, and high-grade VUR were diagnosed in 16 (11.0%), 11 (7.6%), and 12 (8.3%) patients, respectively. A serum creatinine (obtained beyond the newborn period) was available for 47 of the 106 patients with a solitary kidney and no VUR (mean value 0.53 mg/dl). Of the 39 patients with a solitary kidney and VUR, 21 had a serum creatinine available for review (mean value = 0.55 mg/dl). Of the 12 patients with high-grade VUR (grades 4 and 5), nine had an available creatinine (mean value 0.43 mg/dl).

Comparing the VUR and non-VUR subgroups (Table 2), the number of renal ultrasounds was significantly higher in patients with VUR (mean (SD) 4.7 (2.8) for no VUR, and 6.8 (3.2) for VUR; $p < 0.001$). Although we tabulated the occurrence of UTI only with documented culture positivity, many of the UTIs, with or without VUR, were poorly characterized as to symptoms, not allowing analysis based on symptoms and signs at the time of UTI. The distribution of patients with UTIs characterized by symptoms and stratified by VUR status is demonstrated in Table 3. Among patients with both UTI and VUR, 10 had moderate- or high-grade VUR, and 10 had upper tract/febrile UTI, with seven having both. Three of the 16 patients with low-grade VUR had upper tract/febrile UTI. There was no association between VUR and UTI ($p = 0.14$). Of the 27 patients with no VUR but UTI, 17 had BBD. Of the 15 patients with VUR and UTI, 11 had BBD. There was no statistical difference in the occurrence of bladder/bowel dysfunction (BBD) comparing patients with or without VUR ($p = 0.62$).

Growth curves for various randomly selected index patients are represented in Fig. 1. With regards to renal growth, correcting for age and gender, the left solitary kidney tended to be larger than the right ($p = 0.042$). At the average age of the study sample (51.0 months), the average length of a left solitary kidney was estimated at 8.82 cm (SE 0.13), compared with 8.47 cm (SE 0.13) on the right. The three-way interaction term among age, VUR, and UTI was significant ($p = 0.016$). Adjusting for age, gender, and laterality, and considering the three-way interaction among age, reflux, and infection status, a non-refluxing solitary kidney without UTI showed the highest growth rate at 0.48 cm/year (SE 0.02). A refluxing solitary kidney without UTI showed the second highest growth rate at 0.47 cm/year (SE 0.04), followed by a non-refluxing solitary kidney with UTI at 0.45 cm/year (SE 0.03). These small differences were not statistically significant ($p > 0.05$) (Fig. 2). A refluxing solitary kidney with UTI showed significantly lower rate at 0.27 cm/year (SE 0.04) than the rate of a non-refluxing kidney without UTI, a non-refluxing kidney with UTI, and a refluxing kidney without UTI ($p < 0.001$).

Discussion

Since Hodson reported on the likelihood of VUR in children with pyelonephritis and renal scarring in 1960 [1], several studies have associated decreased renal size and growth with the presence of VUR in patients with two functional renal units [7,8]. Historically, some workers reported increased growth after VUR correction procedures [9,10]. Others report

that in children with persistent VUR renal growth is likely to be normal if the kidneys are unscarred and there are no recurrent UTIs [2,3]. There are relatively few publications that touch on this process in children with solitary kidneys. Controversy exists as to whether VUR impacts renal size and growth in this patient population where the kidney is expected to demonstrate compensatory hypertrophy. In an analysis of 48 patients with a MCDK, Zerin et al. found that the kidneys of nine children with VUR to the functionally solitary kidneys were significantly smaller than those in patients without VUR [11], although they did not specifically consider UTIs. In 2001 an Italian study was published that showed significant decreased renal length at birth and at 2 years of age in patients with VUR contralateral to a MCDK compared with those without VUR [12]. Although some of these children had surgery to correct VUR, UTI occurrence was not tabulated. More equivocal are the findings of Abidari et al., who showed that three out of five patients with VUR to a solitary kidney had inadequate growth, while the other two patients, including one with grade 5 VUR, had normal growth [13] (again UTI was not considered). Miller et al. investigated the effects of VUR on 19 kidneys contralateral to a MCDK and found that VUR, with or without UTI, was not a significant factor on the growth of the solitary renal unit in the first few years of life [14]. Our study is the largest to date and we find that neither VUR nor UTI alone significantly affect the size of a solitary kidney. Together, VUR and UTI do result in significantly smaller kidneys with less compensatory hypertrophy. Although the clinical import of this observation is uncertain, it seems likely that a larger kidney is a healthier kidney [15].

In general, a solitary kidney implies normal longevity, but a recent publication by Cherchi [16] suggests that there is a subpopulation of patients with a solitary kidney and unanticipated renal failure at an early age independent of the presence of VUR. In their work they speculated that perhaps some individuals with CAKUT (Congenital Abnormalities of Kidneys and Urinary Tract) and a solitary kidney have reduced number of glomerulae, and therefore are subject to the consequences of hyperfiltration with attendant focal segmental glomerulosclerosis (FSGS). Although these authors did account for VUR, the fog of time did not allow assessment of the occurrence of UTI. Perhaps chronic kidney disease in such patients derives not from hyperfiltration, but rather from the combination of VUR and poorly tabulated and forgotten UTI on the solitary kidney.

Our sonographically determined renal size is subject to criticism. However, in 1984, in the most comprehensive nomogram at the time, Rosenbaum showed that sonography was a reliable renal imaging modality with good correlation to urographic studies [17]. Since that time multiple growth curves and nomograms have been created and employed based on sonographic renal assessment [18–21].

Some authors have suggested superiority of height [19] and total body surface area-based growth curves, but age-based renal nomograms have generally been preferred because of their ease of use. Others have recommended renal parenchymal area or volume rather than length measurement [21]. Inter- and intra-observer variation has been cited as one downside of renal length measurement using ultrasonography. In fact, Ferrer et al. extrapolated from a study of 18 piglets and postulated that error in ultrasound measured kidney length can equal 1 or more years of growth in a child [22]. These authors strongly suggest that

sonographically determined growth is not accurate. However, our sonographic observations were obtained over several years, decreasing the impact of variability in personnel, techniques, and equipment. Although there are concerns about sonographically determined renal size, ultrasound length measurement remains the most widely accepted, highly utilized, cost-effective, and safe method for evaluating renal size, growth, and well-being. In 2009, Geelhoed et al. demonstrated good reproducibility of renal ultrasound measurements [23]. Another drawback of our study is that the renal length measurement does not account for focal renal scarring, which may be important for renal well-being. Another potential limiting factor in our study is the lack of imaging standardization as noted above. Although most imaging was done at our institution, some studies were obtained at different imaging centers and interpreted by outside radiologists. Still, the variability of the measurement does not negate our findings. The variability of measurement causes the loss of statistical significance for comparisons that may be true. However, despite the variability of measurement our findings remain true with statistical significance. As to the etiology of renal hypotrophy, some have suggested that decreased renal size and renal impairment in high-grade VUR may be a result of congenital dysplasia or may occur early in the natural history of the disease [24–26]. Sonographically occult renal dysplasia cannot not be excluded in our cohort because renal biopsies were not performed; however, our observed size discrepancy developed over time and the effect of dysplasia should be apparent in infancy. Another explanation of the growth retardation could be disruption of the still poorly understood process of compensatory hypertrophy. Regardless of etiology, our finding that solitary kidneys start out similar in size but those with VUR and UTI become relatively smaller over time, suggests an ongoing combined effect of VUR and UTI on renal growth.

Even though this is the largest series of solitary kidneys studied, the sample size still did not allow us to incorporate the types of UTI and/or the grades of VUR in the final regression model. Although the patterns of impact seem to be similar across the UTI types and VUR grades, larger confirmatory follow-up studies will be needed to fully evaluate the impact of these important factors.

The retrospective nature of our study also limits the conclusions we are able to draw. Additionally, VCUGs and ultrasounds were obtained at different time intervals. Follow-up VCUGs were not routinely obtained for all patients in our study, and change in VUR status is therefore not reported. Lastly, despite meticulous review of the patient medical records UTI details were often lacking, forcing us to tabulate many of the UTIs as poorly characterized. Still, although the charts lacked the clinical details of the UTIs, their occurrence was associated with decreased renal growth when VUR was present. Given the vagaries of the medical history, it is also likely that some patients were treated for UTI by other providers without our knowledge.

Despite some of the aforementioned limitations, our study remains the largest analysis of solitary kidney growth. Our findings are supported by previous studies and may aid in the management of this specific, relatively vulnerable population. The clinical import of less compensatory hypertrophy in patients with both VUR and UTI is not known, but the smaller renal size is concerning in light of Cherchi's work suggesting poor renal outcome in some patients with a solitary kidney. As noted above, current paradigms tend to minimize the

importance of VUR and UTI in the development of renal damage, but the findings of this single kidney “model” of VUR and UTI suggest a combined effect on renal well-being manifested by less growth and less compensatory hypertrophy.

Conclusion

In the largest series to date, no independent effect of either VUR or UTI on the development of compensatory hypertrophy in patients with a solitary kidney was noted. However, UTI and VUR together result in kidneys that are smaller than other solitary kidneys not so affected. The clinical import of these smaller than expected kidneys remains unclear and close long-term follow-up of larger cohorts of solitary kidneys seems warranted.

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Figure 1.

Growth curves of four representative patients (Patient A and B: No VUR and No UTI (Male and Female), C: VUR and no UTI, D: VUR and UTI).

Figure 2. Growth rate estimates of solitary kidneys (cm/year) for four subgroups including standard errors based on a linear mixed-effects model.

Table 1

Demographic and clinical characteristics of 145 children.

	No. of patients (%)
Gender	
Female	65 (44.8)
Male	80 (55.2)
Side of solitary kidney	
Left	74 (51.0)
Right	71 (49.0)
VUR	
Low grade (1,2)	16 (11.0)
Moderate grade (3)	11 (7.6)
High grade (4,5)	12 (8.3)
No VUR	106 (73.1)
Clinical presentation	
Prenatal	89 (61.4)
Incidental	15 (10.3)
Surgical	7 (4.8)
Infection	5 (3.5)
Unknown	29 (20.0)
Etiology of solitary kidney	
Contralateral MCDK	87 (60.0)
Congenital absence of contralateral kidney	50 (34.5)
Contralateral nephrectomy	7 (4.8)
Unknown	1 (0.7)
Bowel/bladder dysfunction	51 (35.2)
VATER	9 (6.3)
Patients with UTI during follow-up	42 (29.2)

Table 2

Comparison of children with and without VUR.

	No VUR <i>n</i> = 106	VUR <i>n</i> = 39	<i>p</i>
	Mean (SD)	Mean (SD)	
Number of ultrasounds	4.7 (2.8)	6.8 (3.2)	<0.001
Age in month of first ultrasound	33.1 (50.5)	15.3 (23.7)	0.13
	<i>n</i> (%)	<i>n</i> (%)	
Gender			0.57
Male	60 (56.6)	20 (51.3)	
Female	46 (43.4)	19 (48.7)	
Side of solitary kidney			0.74
Left	55 (51.9)	19 (48.7)	
Right	51 (48.1)	20 (51.3)	
Clinical presentation			0.09
Prenatal	61 (57.6)	28 (71.8)	
Incidental	13 (12.3)	2 (5.1)	
Infection	2 (1.9)	3 (7.7)	
Surgical	7 (6.6)	0 (0.0)	
Unknown	23 (21.7)	6 (15.4)	
Bowel/bladder dysfunction	36 (34.0)	15 (38.5)	0.62
VATER	5 (4.8)	4 (10.3)	0.26
UTI	27 (25.7)	15 (38.5)	0.14

Table 3

Distribution of children with respect to VUR grades and UTI types based on symptoms.

UTI	VUR			
	No VUR	Low grade (1–2)	Moderate grade (3)	High grade (4–5)
	<i>n</i> (%)	<i>n</i> (%)	<i>n</i> (%)	<i>n</i> (%)
Upper Tract UTI	10 (9.5)	3 (18.8)	3 (27.3)	4 (33.3)
Lower/asymptomatic UTI	8 (7.6)	0 (0.0)	0 (0.0)	2 (16.7)
Poorly characterized UTI	9 (8.6)	2 (12.5)	1 (9.1)	0 (0.0)
No UTI	79 (74.3)	11 (68.7)	7 (63.6)	6 (50.0)
Subtotal	106	16	11	12