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The prevalence of congenital anomalies of kidney and urinary tract in children with spina bifida: A cross-sectional study

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Abstract

Objectives: The aim of this study was to investigate the prevalence of renal anomalies in a large cohort of children with spina bifida and review the information in the literature.

Patients and methods: Between January 2005 and February 2025, a total of 1,039 children (499 males, 540 females; mean age: 4.7±3.1 years; range, 4 days and 17 years) with the diagnosis of spina bifida and spina bifida occulta who were under follow-up within the last 20 years were included in the study. These patients were evaluated for the presence of congenital anomalies of kidney and urinary tract (CAKUT). Age, sex, any urinary anomaly detected with ultrasonography and scintigraphy were noted. English literature was also reviewed the studies reporting the association of spina bifida and CAKUT.

Results: Of all patients, 17 had renal rotational anomaly and 13 had horse-shoe kidney. Nine patients had renal agenesis. Three patients had cross-renal ectopia, while one patient had ureterocele and another patient had ureteropelvic junction obstruction. The total number of patients with congenital renal anomalies associating with spina bifida was found to be 44 with a prevalence of 4.23% in this cohort.

Conclusion: This cohort seems to have the largest patient population reported in a single center on this subject. The prevalence of CAKUT is increased in patients with spina bifida compared to general population. The cause of this increase may be the close embryological background of two systems. Awareness of such a clinical entity may promote the renal protective approach in patients with spina bifida.

Keywords: Children, congenital, congenital anomalies of kidney and urinary tract, renal anomalies, spina bifida.

Spina bifida is a congenital spinal anomaly characterized by defective closure of neural tube during embryogenesis. This complex malformation is associated not only with neurogenic bladder dysfunction (NBD), but also with a broad spectrum of congenital anomalies of kidney and urinary tract (CAKUT). Vertebral bodies start to develop at third weeks of gestation with the formation of notochord. These somites further develop to form vertebral bony structures. During this period paraxial mesoderm and intermediate mesoderm lie next to the notochord. Paraxial mesoderm is responsible for the formation of the vertebrae, as well as the dermis of the skin, striated skeletal muscle, muscles of the head and connective tissue. Renal development starts with pronephros which is formed from intermediate mesoderm.[1,2] Disruptions during the critical period of neural tube closure (Weeks 3 to 4 of gestation) can disturb the intricate process of mesodermal differentiation, leading to concurrent anomalies in both systems.[1] Typically, CAKUT include renal dysplasia, unilateral renal agenesis, ectopic kidneys, collecting system duplications, ureteral malformations, and even structural bladder abnormalities.[3,4] The occurrence of these malformations highlights the shared embryological origins of the spinal cord and the urinary tract.

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Congenital renal anomalies, even without NBD due to spina bifida, are clinically important as they comprise 30 to 40% of all children with chronic kidney disease worldwide.[5] Therefore, the association of both anomalies clearly increases the potential lifelong risk of renal impairment in these patients. The exact prevalence of congenital renal anomalies associating with spina bifida is currently unknown. There are conflicting data in the literature regarding a prevalence rate of between 2 and 17.8%.[3,6-9] In the present study, we aimed to investigate the prevalence of congenital renal anomalies associating with spina bifida, which, to the best of our knowledge, is the largest single-center pediatric case series in the literature.

PATIENTS AND METHODS

This single-center, cross-sectional, observational, retrospective study was conducted at Demiroğlu Bilim University Faculty of Medicine, Department of Pediatric Surgery between January 2005 and February 2025. A total of 1,039 children (499 males, 540 females; mean age: 4.7±3.1 years; range, 4 days and 17 years) with the diagnosis of spina bifida and spina bifida occulta who were under follow-up within the last 20 years were included in the study. Medical data were retrieved from the hospital records. Inclusion criteria were as follows: having a diagnosis of either occult or apert spina bifida, any renal anomaly detected with either urinary ultrasonography or static scintigraphy including rotation, fusion, developmental and positional anomalies. The imaging methods used in the follow up of our patients were ultrasonography, voiding cystourethrography (VCUG), when needed, and dimercaptosuccinic acid (DMSA) scintigraphy. Routine radiological follow up of the patients was yearly ultrasonographic screening and VCUG, when needed, and at least two DMSA scintigraphy scans in the first five years of life. If a consistent diagnosis of any congenital renal anomaly in all these imaging methods was seen, then, the patient was accepted to have this condition. Patients with the diagnosis vesicoureteral reflux (VUR) and hydronephrosis not associated with the congenital renal anomaly were excluded, as these pathologies might not be regarded as congenital but secondary to the effects of NBD. Genital anomalies were also excluded, as these malformations have different developmental origin than mesodermal defects. These data were evaluated with the past information in the literature. Age and sex of the patients were also recorded. A comprehensive literature search was carried out. The databases searched during the study were PubMed, Scopus, Embase and Cochrane Library with the keywords of spina bifida, spinal dysraphism, renal, kidney, renal failure, VUR, reflux, urinary bladder, urodynamics, urology, congenital anomaly, malformation, abnormality, spine. All the English references were collected from these databases. All the reports, case series or case reports were searched to identify the association of congenital renal anomalies with spina bifida. Data were gathered and summarized. Frequencies and percentages of the anomalies were calculated, including the side of the renal anomaly and sex difference in each anomaly. Written informed consent was obtained from the parents and/or legal guardians of the patients. The study protocol was approved by the İstanbul Medeniyet University Göztepe Training and Research Hospital Clinical Research Ethics Committee (Date: 02.12.2020, No: 2020/0618). The study was conducted in accordance with the principles of the Declaration of Helsinki.

Statistical analysis

The data were expressed and given in case numbers, frequency and percentages. The mean ages were given in mean ± standard deviation (SD).

RESULTS

Of a total of 1,039 patients, 127 had unilateral hydronephrosis and 40 had bilateral hydronephrosis. There were 135 right-sided and 72 left-sided hydronephrosis in terms of the side of the pathology.

Seventeen patients had some form of rotational anomaly: 10 on the right side and seven on the left. Of these, 12 were male and five were female. Thirteen patients had the diagnosis of horse-shoe kidney (six boys, seven girls). Nine patients had renal agenesis: four had right-sided and five had left-sided agenesis. Of these, five were male and four were female. Three patients had cross-renal ectopia and one had ureterocele and one had ureteropelvic junction obstruction. The total number of patients with congenital renal anomalies associating with spina bifida was found to be 44 with a prevalence of 4.23% in this cohort.

TABLE 1
Literature review of congenital anomalies of kidney and urinary tract

Author	Year of publication	Type of publication	Duration of the study	Total number of patients	Renal anomalies: number of patients	Type of renal anomalies	n
Roberts[6]	1961	Retrospective	NS	140	28	• Bilateral renal agenesis • Unilateral renal agenesis • Hypoplasia • Double kidney • Horseshoe kidney • Crossed renal ectopia • Bilateral polycystic kidney • Unilateral polycystic kidney • Stricture of distal ureter • Pelviureteric stricture • Bilateral ectopic ureter • Bladder anomalies	2 2 1 1 9 2 4 2 1 1 1 2
Smith[10]	1965	NS	NS	100	5	• Duplicated urinary collecting system • Horseshoe kidney • Exstrophy of the bladder	3 1 1
Tori and Dickson[11]	1980	Retrospective	1960-1977	160	7	• Agenesis of one kidney • Horseshoes kidney • Duplication of upper collection system • Duplication of bladder	3 2 1 1
Fernbach and Davis[8]	1986	Cohort	NS	68	42	• Anomalies of the renal axis • Horseshoe kideney • Other NS	38 19 4
Whitaker and Hunt[12]	1987	Retrospective	NS	190	17	• Renal agenesis • Horseshoe kidneys • Ureteral duplications • Ureterocele	3 5 8 1
Alston et al.[13]	1989	Case report		1	1	• Ectopic immature renal tissue	
Bamforth and Baird[7]	1989	Retrospective	1952-1986	479	10	• Unilateral renal aplasia • Horseshoe kidney • Pelvic kidney • Crossed fussed ectopia • Duplex ureters • Polycystic kidney	3 3 1 1 1 1
Hulton et al.[14]	1990	Retrospective	1971-1987	163	17	• Renal agenesis • Ureteral duplication • Horseshoe kidney • Crossed ectopia • Pelvic kidney • Other NS	4 3 2 2 4 2
Mandell et al.[15]	1996	Retrospective	NS	189	21	• Horseshoe kidneys • Solitary kidneys • Duplications • Cross fused ectopia	13 4 3 1
Johnston and Borzyskowski[16]	1998	Retrospective	1976-1995	61	40	• Dilated upper renal tracts • Residual volume postmicturition • Thick walled bladders • Unilateral small kidney • Renal scars • Other NS	14 6 6 2 1 11
Nallegowda et al.[17]	2003	Case report	-	1	1	• Ectopic kidney	
Uzum et al.[18]	2005	Case report	-	1	1	• Horseshoe kidney	
Patel et al.[19]	2007	Retrospective	NS	140	6	• Horseshoe kidney • Ectopic kidney • Crossed fused ectopia	2 3 1
Baradaran et al.[20]	2008	Retrospective	2001-2007	17	4	• Bladder exstrophy • Horseshoe kidney • Dysplastic kidneys	2 1 1
Kari et al.[21]	2009	Retrospective	1997-2006	33	-	• Neurogenic bladder • Vesico-uretral reflux • Renal agenezi	30 26 1

TABLE 1
Continued

Author	Year of publication	Type of publication	Duration of the study	Total number of patients	Renal anomalies: number of patients	Type of renal anomalies	n
Thakur et al.[22]	2010	Case report	-	1	1	• Kidney malrotation (reverse rotation of left kidney with hydronephrotic changes on both the sides	
Torre et al.[23]	2011	Retrospective	25 years	502	N.s	• Renal agenezi	17
Patiatil et al.[24]	2012	Case report	-	1	1	• Bilateral simple renal ectopia	
Steelman et al.[25]	2012	Case report	-	1	1	• Unilateral renal agenesis	
Calleja Aguayo et al.[26]	2012	Case report	-	1	1	• Horseshoe kidney	
Parker et al.[3]	2013	Retrospective	1976-2011	1,170	42	• Renal agenesis (unilateral) • Horseshoe kidney • Double collecting system • Ectopic kidney • Multicystic kidney type 2 • Kidney agenesis • Kidney dysplasia (unilateral) • Renal agenesis (bilateral) • Polycystic kidneys • Accessory kidney • Absent ureter • Absent bladder/urethra • Prune belly syndrome • Kidney dysplasia (bilateral)	14 7 5 4 3 2 1 1 1 1 1 1 1 1
Aydin et al.[27]	2015	Case report	NS	2	2	• Unilateral renal agenesis • Unilateral renal hypoplasia	1 1
Bozdogan et al.[28]	2016	Case report	-	1	1	• Reverse u-shaped horseshoe kidney	
Parmar et al.[29]	2016	Case report	-	1	1	• Ectopic kidney	
Özgönenel et al.[30]	2017	Retrospective	NS	100	NS	• Unilateral renal agenesis • Horseshoe kidneys • Atrophic kidney with function loss • Scarred kidneys • Ectopic kidneys • Hydronephrosis • Pelvic ectasia • Bladder diverticulosis	3 3 4
Maeda et al.[31]	2018	Case report	-	1	1	• Unilateral renal agenesis	
Kaur et al.[32]	2019	Retrospective	2008-2017	164	6	• Renal agenesis • Polycystic kidney • Horseshoe kidney	2 3 2
Ozturk et al.[9]	2019	Case report	-	4	4	• Unilateral renal agenesis • Unilateral renal dysplasia	
Puvabanditsin et al.[33]	2020	Case report	-	1	1	• Multicystic dysplastic kidney and hydronephrosis	
Mazzone et al.[34]	2020	Cohort	7 years	82	5	• Posterior urethral valves • Hypodysplastic kidneys • Distal hypospadias	1 3 1
Hong et al.[35]	2021	Retrospective	2013-2018	190	23	• Solitary kidney • Renal dysplasia • Renal ureteral duplication • Horseshoe fusion kidney • Ectopic kidney • Bladder duplication • Bladder ectropion	5 5 4 4 3 1 1
Current study	2025	Retrospective	2005-2025	1,039	44	• Rotation anomaly • Horseshoe kidney • Renal agenesis • Cross renal ectopia • Ureterocele • Ureteropelvic junction obstruction	17 13 9 3 1 1

NS: Not specified.

The patients reported in the English literature to date, combined with this study findings, are summarized in Table 1.

DISCUSSION

The embryogenesis of the urinary tract is intricately linked to the development of the vertebral column. Vertebral somites develop from paraxial mesoderm and pronephros develop from intermediate mesoderm. These two mesodermal tissues lie adjacent to each other during embryogenesis.[1] During early gestation, the formation of the pronephros, mesonephros, and ultimately the metanephros, which becomes the definitive kidney, occurs in a tightly regulated sequence. Disruptions in these processes, which may accompany neural tube defects, can result in anomalies such as renal dysplasia or unilateral renal agenesis.[2] Aberrations in the formation or branching of the ureteric bud may lead to duplications or ectopic positioning of the collecting system, further complicating the clinical picture. These anomalies not only affect renal function, but also influence the dynamics of the lower urinary tract, setting the stage for secondary complications such as VUR and recurrent urinary tract infections (UTIs). Early identification is crucial, as congenital anomalies, if unrecognized, can predispose patients to long-term complications including VUR, UTIs, and progressive renal scarring, which may eventually compromise renal function.[5]

Bladder is embryologically, urogenital sinus in origin and its functions are in neurological control. This control is compromised in patients with spina bifida due to the primary and secondary neurological injury. Loss of synergistic activity of detrusor and urinary sphincter causes urinary tract deterioration in as much as 71% of newborns within the first three years of life.[36] The presence of congenital urinary anomalies in spina bifida patients carries additional significant clinical ramifications. Renal dysplasia and unilateral renal agenesis may reduce the overall renal reserve, rendering patients more vulnerable to the effects of NBD. In a retrospective analysis of 312 children with spina bifida, 72 of these patients (23%) were found to have renal scarring in their follow up. Additionally, late referral, female sex, NBD with detrusor overactivity and detrusor sphincter dyssynergia were observed to significantly affect renal impairment in this study.[37] Therefore, early and accurate diagnosis is paramount in mitigating the progression toward chronic kidney disease in these patients.

Currently, the prevalence of CAKUT associating with spina bifida is largely unknown. According to our literature search, this ratio seems to be between 2 and 17.8%.[3,6-9] In this current study, among 1,039 children with spina bifida, 44 of them were found to have a form of CAKUT with a prevalence rate of 4.23%. The only study that comprised 1,170 patients was from a multi-national, multi-centric, long-term database study and this prevalence was 3.59% in the same patient group. Therefore, our study seems to have the largest single center case series in the literature on this subject. Still, when we consider the general incidence of CAKUT of 4 to 60/10,000 live births, it is plausible to speculate that this ratio is extremely high in cases of spina bifida. In a clinical study including 231 patients with congenital scoliosis, the incidence of urological anomalies was found to be 18%.[38] This raises the possibility of a genetic background for renal anomalies in patients with congenital spinal pathologies. However, although the role of genetics in organ development is well established, the interplay between genetic and environmental factors is still accepted to be responsible for CAKUT.[4] Among these environmental factors, maternal obesity, diabetes mellitus and folic acid deficiency were also accused for the development of CAKUT.[4,39] This issue is critical, as these factors are also accepted to be responsible for the development of neural tube defects. In their study, Hernandez-Diaz et al.[39] showed that folic acid antagonists taken during pregnancy increased the risk of not only neural tube defects, but also cardiovascular defects, oral clefts and urinary tract defects. This point may give us a reasonable explanation of the association with spina bifida and CAKUT which needs further clarification with detailed clinical studies.

Given the progressive nature of many congenital urinary anomalies, long-term follow-up is essential. Regular monitoring through renal ultrasonography, VCUG, and nuclear imaging modalities such as DMSA scintigraphy allows for the early detection of changes in renal structure and function. Such surveillance is critical in identifying evolving complications such as worsening reflux or increasing

renal scarring. Ongoing follow-up also provides an opportunity for timely intervention, which is crucial in preserving renal function and maintaining quality of life. Furthermore, the integration of emerging biomarkers and molecular diagnostics may, in the future, refine risk stratification and guide more personalized therapeutic approaches.

Nonetheless, this study has some limitations. First, it has a single-center, cross-sectional, observational, retrospective design and there is no control group to test the hypotheses for the association of these two groups of anomalies. However, it seems to be the largest single-center case series of spina bifida patients in the literature and the literature review of all the case series may serve as a reference for further studies on this subject. Second, the diagnosis of renal anomalies was made based on ultrasonography and DMSA scintigraphy which may have potential for diagnostic bias; i.e., some anomalies might have been missed with imaging studies. Third, we did not include the patients with VUR to the patient group, as it is difficult to understand if the reflux is primary or secondary under the circumstances of NBD. The numbers of patients with hydroureteronephrosis may show the high association of VUR with spina bifida and also the bladder dysfunction related with spina bifida by its own. However, we did not use these criteria for the impossibility of differentiating the congenital or acquired forms of VUR in these patients. Further multi-center, large-scale, prospective studies are warranted to confirm these findings.

In conclusion, CAKUT in spina bifida represent a multifaceted clinical challenge that extends beyond the primary neural tube defect. The prevalence of CAKUT seems to be increased in patients with spina bifida compared to general population. The interplay between embryological disruption, genetic predisposition, and other factors may result in a spectrum of urinary tract malformations that has potential impact on renal function and quality of life. Early detection via advanced prenatal imaging, combined with a multidisciplinary management strategy, is critical to mitigate long-term renal damage and improve patient outcomes. Continued research into the molecular mechanisms underlying these anomalies may hold promise for the development of innovative prenatal therapies and more personalized treatment protocols. Ultimately, vigilant post-natal monitoring and comprehensive care remain essential to safeguard renal function and enhance the quality of life for spina bifida patients associated with CAKUT.

Data Sharing Statement: The data that support the findings of this study are available from the corresponding author upon reasonable request.

Author Contributions: Idea/concept, control/supervision, data collection and/or processing: Ş.K.Ö., İ.A.; Design, writing the article, materials: Ş.K.Ö.; Literature review, references and funding: Ş.K.Ö., L.A.; Critical review: İ.A., L.A.

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