Case Report

BILATERAL BIFID URETER WITH ACCESSORY RENAL ARTERY: A CASE REPORT

Vasudha Kulkarni[1], Ramesh B.R[2], Prakash.B.S[3]

ABSTRACT

Context: Most common type of congenital abnormality of urinary system is the abnormality of renal collecting system. One such variation is presence of bifid ureter.

Objective: To describe and analyze the clinical and embryological significance of bilateral bifid ureter with accessory renal artery.

Design: The presence of bilateral bifid ureters was seen as an incidental finding during routine educational dissection. The etiology of this variant is fission of the ureteric bud during embryogenesis. In the same cadaver, bilateral accessory renal artery was seen, arising from abdominal aorta opposite to inferior poles of kidneys.

Outcome: The knowledge of relationship of renal arteries and collecting system is necessary for effective endo-urological applications and intra-renal surgeries.

Conclusion: Bifid ureter with accessory renal artery forms one of the common differential diagnoses for ureteric calculi. Such anatomical variants may simulate pathological obstruction of urinary tract and must be considered while performing endo-urological procedures.

Keywords: Bifid ureter, accessory renal artery, bifid pelvis.

INTRODUCTION

Nearly 10% of all human beings are born with a congenital abnormality of the urogenital system[1]. 40% of renal pathologies are due to its variations. The principal variation of ureter is complete or incomplete division into two[2]. Duplication of the upper urinary tract accounts for one of the common variants occurring in 1 in 160 individuals[1]. Incomplete duplication is known as bifid ureter. Presence of bifid ureter is often associated with various congenital anomalies and clinical complications[1].

CASE REPORT

During routine educational dissection of an adult male cadaver aged 58 years, we found incomplete duplication of upper ureter bilaterally. The bifid ureter rejoined to continue as a single ureter, opposite to uretero-pelvic junction, seven centimeters distal to corresponding renal hilum. Right kidney was contracted and granular. Incidentally, accessory renal artery, a branch of abdominal aorta was found to supply inferior pole of corresponding kidneys.

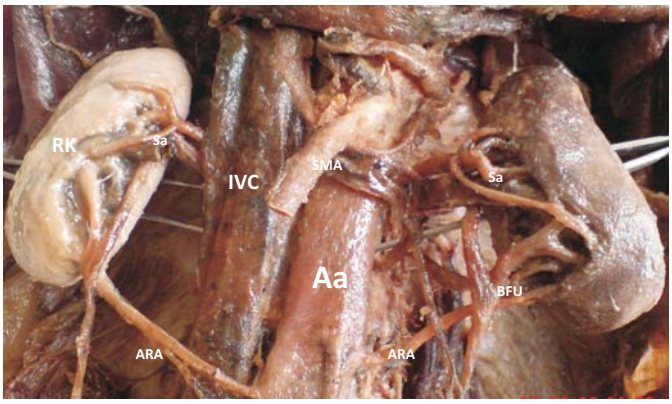
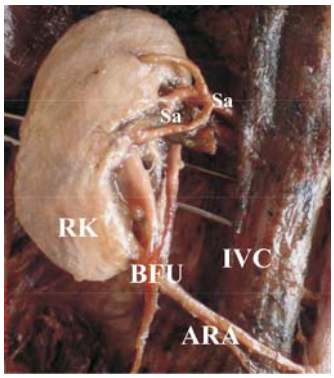


Fig 1: Anterior view of both the kidneys showing presence of bilateral bifid ureters and accessory renal artery. RK- right kidney, LK- left kidney, BFU - bifid ureter, ARA - accessory renal artery, Aa - Abdominal aorta, SMA- superior mesenteric artery, IVC- inferior venacava, Sa - segmental branches of renal artery.



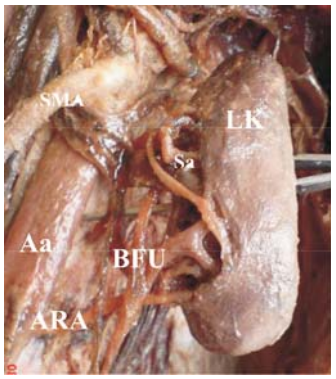


Fig 2: Anterior view of right kidney, LK- left kidney, BFU- bifid ureter, ARA- accessory renal artery, Aa- Abdominal aorta, SMA- superior mesenteric artery, IVC- inferior venacava, Sa- segmental branches of renal artery.

Fig 3: Anterior view of left kidney .RK-right kidney, BFU- bifid ureter, ARA- accessory renal artery, Aa- Abdominal aorta, SMA- superior mesenteric artery ,IVC- inferior venacava, Sa- segmental branches of renal artery.

DISCUSSION

Duplication in the ureter is either complete or incomplete. As a rule, the duplicated ureters unite a little above the bladder, so that there is only one vesical orifice. Bifid ureter occurs in an incidence of 0.5%[2]. The incidence ranges from 0.5% to 3.0%[3]. It is two to five times more common in females[4]. Incomplete duplication is three times more common than complete duplication[5]. Duplication of ureter is more common on right side[6]. In a study, the incidence of bifid ureter was found to be 18 out of 4215 autopsies; 2 were bilaterally bifid, 7 were unilaterally bifid, and 8 were completely duplicated ureters. On an average, 3% of excretory urograms show ureteral duplication on routine examination. Bifid renal pelvis occurs in 10% of cases[5]. The incidence for incomplete ureter duplication and a bifid renal pelvis (duplication of pelvis of ureter) is 4% in the North American population. It is common in Caucasian race[2].

Bifid ureter may remain asymptomatic and create academic interest only lest it may cause complications like ureteric stenosis, urinary lithiasis and pyelonephritis. Patients present with fever, chills, nocturia, dysuria or gross hematuria. Intravenous pyelography is diagnostic[4]. 40% of pathological conditions of urogenital system are due to congenital abnormalities[2].The risk of infection in children with bifid ureter increases twenty fold[1]. A bifid urinary system is prone to urinary tract infection either from obstruction of ectopic ureter, calculi or vesicoureteric reflux[5]. It can also result in ureteroureteric reflux or ureteropelvic obstruction at the lower pole of the kidney. Familiarity of such abnormalities is essential for differential diagnosis of filling defects in excretory urography or cystography. Patients with bifid ureters have increased risk of hydronephrosis. Segmental nephrogram shows persistence of nephrogenic density in parts of kidney in case of obstruction of one limb of a bifid renal pelvis[6].

Vesicoureteric reflux occurs in 69% of cases of complete duplex systems and 22% case of partial duplex systems[4]. Segmental nephrogram shows persistence of nephrogenic density in cases of vescicoureteric reflux with bifid ureter[6].Association of bifid ureter must be anticipated as a differential diagnosis for ureteric calculous[6].

Table showing Vesicoureteric reflux with duplicated systems 4

Sl no	Author	Percentage of vesicoureteric reflux
1.	Ambrose and Nicolson	50%
2.	Bisset	69%
3.	Fehrenbaker	72%

Presence of accessory renal artery accounts for incidence in the range of 9-76%. Median incidence is 30%. They are essential tissue sustaining end arteries. When associated with bifid ureter, it may cause compression of the latter resulting in hydronephrosis[7].

Embryological basis:

Ureter development starts at 5th week of gestation. The ureteral bud (metanephric blastema) arises as a diverticulum from the wolffian duct and invades nephrogenic blastema. The ureteral bud divides and branches to form the renal collecting system. [3] After its emergence from the metanephric duct, the ureteric bud can split or bifid. The bifid ureter can eventually reunite as a common ureter[8].

Incomplete double ureter is caused due to early division of the renal bud extending beyond the renal pelvis into the ureter segment. Each ureter is incorporated into posterior wall of urogenital sinus at 7th week of intrauterine life[3].

The additional ureter may be derived from an additional renal bud which arises from the wolffian duct[3]. The interaction of the ureteric bud with the metanephric blastema is critical to the correct ontogeny of the ureter and collecting system of future kidney[9]. Bone morphogenic factor 4 (required for urinary tract organogenesis) and Glial cell derived neurotropic factor (responsible for urinary bud growth and elongation) are responsive for abortive attempts at ureteral duplication[10]. c-ret, a receptor for GDNF, is responsible for branching and growth of the ureteric bud. Lack of the receptors causes bifid ureters and renal agenesis[10]. PAX2, a member of paired box family of genes plays a critical role in development of kidney and ureter. PAX2 mutation is a cause of bifid system as well as familial vesicoureteric reflux[11].

Thus ureteric bud is a highly plastic structure which can branch in a variety of complex patterns including bifid (bifurcation), terminal bifid and lateral branching[12].

Embryologically, a capillary network known as "rete arteriosum urogenitale", gives rise to all definitive renal arteries. The segmental splanchnic arteries branch from the aorta and form the proximal portion of this network. Persistence or enlargement of these vessels gives rise to variations in the renal arteries. Other factors such as genetic background, oxygenation and hemodynamic changes also account for the presence of accessory renal artery. Inferior polar artery is considered as segmental vessel with an unusual origin because of persistence of foetal vessels[7].

Bifid ureters are associated with other congenital anomalies such as Goltz's Syndrome [5], high cephalad kidney and duplication of pelvis[5], ureterocele, malrotation, ureteropelvic obstruction[5], bladder diverticulum [5], unilateral pulmonary hypoplasia [5], complete duplication of contralateral ureter [5], branchio –oto–ureteral syndrome (BOU)[13] . The inheritance of bifid ureter is by an autosomal dominant gene[1].

Anomalous differentiation of the kidney and upper urinary tract is common (88.8%) in gonadal dysgenesis[14].

Duplication of pelvicalyceal system is among the common anomalies of urinary tract, majority is unilateral. It results from division of metanephric diverticulum. It may be associated with cephalad renal ectopia and patent ductus arteriosis. The constellation of genitourinary, skeletal and cardiovascular anomalies is due to common mesodermal origin of these systems[15].

The awareness of variations in the origin of renal arteries is of importance for the urologists while performing nephron preserving surgery, kidney transplantation, and management of renal vascular hypertension. The association of polar renal vessels with ureteropelvic junction obstruction is of particular concern with minimally invasive techniques, because crossing vessels compressing or distorting the ureteropelvic junction (UPJ) may be the sole cause of ureteral outflow obstruction. The knowledge of renal vascular variations also provides guidelines for endovascular procedures like therapeutic embolization and angioplasties[15].

CONCLUSION

Bifid ureter is the most common congenital anomaly accounting for 1% of incidence. In association with accessory inferior polar artery, it enhances risk of renal complications. To avoid injury to accessory renal artery, such variations have to be kept in mind. Recognition of such variants prior to renal surgeries can decrease complications, improve surgical approach and its outcome.

Acknowledgements

We extend our sincere thanks to our nonteaching staff who helped us and supported during the writing of this manuscript. We also express our thanks to our institution, Dr. B. R. Ambedkar medical college and all the faculty members for helping in presentation of this manuscript. I "had full access to all the data in the study and I take responsibility for the integrity of the data and the accuracy of the data analysis."

REFERENCES

1. J. D. Atwell, P. L. Cook, C. J. Howell. Familial incidence of bifid and double ureters. Archives of Disease in Childhood. 1974; 49: 390-393.

2. Adel K. Afifi, Ryosuke Miyauchi, Ronald A. Bergman; Illustrated Encyclopedia of Human Anatomic Variation: Opus IV: Organ Systems: Urinary System Kidneys, Ureters, Bladder, and Urethra; www.anatomyatlases.org . Accessed on Nov 2010.
3. Setsuko Tohno, Cho Azuma, Yoshiyki Tohno. A case of double renal pelvis and ureters associated with double Superior venacavae. J Nara Med Assoc. 2008; Vol 59, Issue 5: 183-187.
4. George S Bisset III, Janet L Strife .The Duplex collecting system in girls with urinary tract infection ; Prevalence and Significance. AJR. March 1987; 148: 497-500.
5. Das S, Dhar P, Mehra R.D. Unilateral Isolated Bifid Ureter: A Case Report. J Anat. Soc. India. 2001; 50(1): 43-44.
6. Ray Dyer, Stephen Miller, Bonnie L Anderson, James E Drake, John S Shaffer. The Segmental Nephrogram. AJR. August 1985; 145: 321-322.
7. Khin Pa Pa Hlaing, Srigit Das , Israa Maatoq Sulaiman, Azian Latiff, Norzana Abd Ghafar, Farihah Haji Suhaimi. Accessory renal vessels at the upper and lower pole of the kidney: A cadaveric study with clinical implications; Morphological study. Bratisl lek listy. 2010; 111(5): 308-310.
8. M. Y. Mubarak, A. R .Zainun, M. Rohaya. Ureter Triplication with Contra-Lateral Partial Duplex System; case report. Med J Malaysia. September 2009; Vol 64; No 3:236 – 237.
9. Walsh, Retik, Vaughan, Wein. Campbell Urology. 2002; 8th Edition, Vol 3: 2007-2008.
10. Frank Costalini. Renal branching morphogenesis: concepts, questions, and recent advances; International Society of Differentiation. 2006; 74: 402–421.
11. K. L. Choi, L. A. McNoe, M C French et al., Absence of PAX2 gene mutations in patients with primary familial vesicoureteric reflux. J Med Genet. 1998; 35: 338-339.
12. Bergman B, Hanssan G, Nilson.A.E. Duplication of the renal pelvis and blind – ending bifid ureter in twins. Urol Int. 1977; 32 (1): 49- 55.
13. Wellington Hung, Joseph M, Lo Presti M. Urinary Tract Anomalies in Gonadal Dysgenesis. American Journal of Roentgenology. October 1965; Vol. 95; No. I: 439-441.
14. Haider M Al Attia ; Cephalad Renal Ectopia, Duplication of Pelvicalyceal System and Patent Ductus Arteriosus in an Adult Female . Scand J Urol Nephrol. 1999; 33: 257–259.
15. Satheesha Nayak B; Presence of accessory renal artery and kinking of aorta due to abnormal origin of renal arteries. The Internet Journal of Biological Anthropology. 2008; Vol 1, NO 2.

1. **Vasudha Kulkarni**
Assistant Professor of Anatomy
DR B R Ambedkar Medical College
K G Halli, Bengaluru -560045
Karnataka, India

2. **Ramesh B.R**
Professor & HOD
Department of Anatomy
DR B R Ambedkar Medical College
K G Halli, Bengaluru -560045
Karnataka, India

3. **Prakash.B.S**
Associate Professor
Department of Anatomy
DR B R Ambedkar Medical College
K G Halli, Bengaluru -560045
Karnataka, India