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Original Research Article

Bifid Ureter: A Cadaveric Study

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Abstract:

Bifid Ureter is a condition where upper end of the ureter is bifid. In the lower part of the course two ureters join together to open by a common orifice into the urinary bladder. It occurs due to premature division of ureteric bud.

Aim: To study any anomalies of ureter in routine anatomical dissection.

Methods: 50 cadavers of both sexes that had been fixed in formalin were used in the current study. During the dissection, the ureter's anatomy was the main point of attention. From the renal pelvis to the uretrovesical junction, which opens into the urine bladder, the ureter was traced. One of them yielded a cadaver in which two ureters were independently draining a single kidney into the UB and were unrelated to any other congenital abnormality. The left kidney and ureter were both perfectly healthy.

Conclusion: Patients with bifid ureters may be accompanied by other ureteral anomalies such as ectopic ureter and have an increased risk of developing urinary tract infection, hydronephrosis and stone formation. The knowledge of abnormality in renal collecting system is necessary for effective endo-urological applications and intrarenal surgeries. The present study adds on to the literature and will be helpful and interesting for the radiologists and urologists as it describes the ureteric anomaly.

Keywords: Bifid Ureter, Kidney Ureter Bladder Region, Mesonephric Duct, Ureteral Duplication, Ureteric Bud.

Introduction

The ureter measures 25 cm. At the pelviureteric junction, where it crosses the pelvic brim, and when it enters the bladder wall, it is very narrow at these spots. The gonadal vessels themselves are crossed anteriorly by it as it descends on the psoas major and crosses in front of the genitofemoral nerve. Lower down, it is anteriorly crossed by the right colic and ileocolic veins as well as by the root of the mesentery, while the top portion is behind the third part of the duodenum on the right. It is lateral to the inferior mesenteric vessels on the left, is anteriorly crossed by the left colic vessels, and is apex of the sigmoid mesocolon at the pelvic brim. It exits the psoas muscle at the common iliac artery bifurcation, travels over the sacroiliac joint, and enters the pelvis. It typically crosses the external iliac artery and vein before passing in front of the internal iliac artery on the side wall of the pelvis (and behind the ovary). It passes over the obturator nerve, destroyed umbilical artery (superior vesical), obturator artery, and obturator vein from above downward.[1]

The vas deferens in males crosses medially above the ureter before running medially to the ureter. Normally, the upper end of the seminal vesicle is located immediately below where the ureter joins the bladder wall. The ureter is located at the base of the wide ligament in females, where the uterine artery crosses it. Under the broad ligament the ureter penetrates the condensed tissue that forms the lateral cervical ligament, traversing the lateral vaginal fornix 1-2 cm from the cervix before entering the bladder in front of the fornix. [2] The ureter's upper end is bifid in those with bifid ureter. In lower third of course two ureters merge and open by a shared orifice into the urine bladder. The

ureteric bud's early division is the cause of this. [3,4]

Ureter

The kidneys and ureters are located in the rear abdominal wall, retroperitoneally. With the creation of the uretric bud, the collecting ducts of the permanent kidney begin to develop. The metanephric blastema, which acts as a cap and is formed over its distal end, gets penetrated by it. This end then dilates to form the primitive pelvis, separates into the cranial and caudal portions, and develops more large calyces. In the antero-posterior meaning, the topographic arrangement of the hilar structures is known as its veinarterypelvis in classical literature. One of the uncommon anomalies of the collecting system is unilateral bifid ureter, which is characterised by renal pelvis that is outside the renal parenchyma, linked with changes of renal arteries, and disposition of hilar structures. Other abnormalities like bifid kidney, ectopic kidney, horseshoe kidney, and renal dysplasia may be linked to this variance. They may develop hydronephrosis as a result of embryological causes. Knowing about these variances could be a helpful tool for vascular surgeons, urologists, and radiologists. It can aid in avoiding surgical complications and diagnostic blunders. Double renal pelvis with bifid ureters at the hilum that descend as individual renal pelvises and fuse together at the lower pole of the kidney subsequently displayed their typical anatomy.

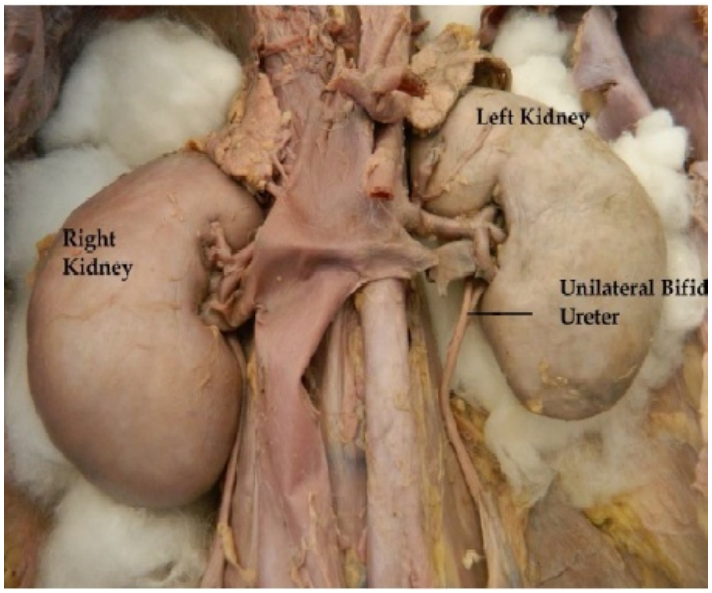

Figure1 Showing Unilateral bifid Ureter on left kidney.

Complications

Neverthless some complications have been reported such as:

- Frequent urinary tract infection.
- Uretero ureteric reflux.
- Ureteric stenosis.
- Urinary lithiasis.
- Pyelonephritis.
- Non-functioning of kidney units.

Congenital Abnormalities of the Ureter

The pelvic surgeon must have some familiarity with these because they are many and diverse. The Wolffian duct's posterior side, just before it enters the cloaca, is where the ureter develops as a bud. This bud contacts the primitive nephrogenic mass that is currently located in the female pelvis and will later create the renal pelvis and the ureter proper. The calyces are formed after the ureteric bud separates. By the eighth week of foetal life, the primitive kidney has ascended from its pelvic location and has arrived at its adult subcostal location. It rotates medially as it ascends, causing the hilum to open medially as opposed to ventrally as it did initially. The reason why two ureters are typically crossed is because of this rotational action. The kidney obtains its blood supply en route from the numerous major vessels that it may come into contact with as it ascends from the pelvis. The kidney may rest at the pelvic brim or may stay in the pelvis permanently. As a result, its blood supply will be abnormal—something to keep in mind if such a kidney needs to be removed. The opening of extra ureters can take place in the urethra, the bladder, or even the vagina. The ureter may only be partially double and bifid in some circumstances. The potential of having two or more ureters should be kept in mind since the surgeon may decide to save the ureter that opens in the vagina while surgically removing the better ureter. Congenital abnormalities in one kidney may be accompanied with a rudimentary or aplastic kidney on the opposite side; this is particularly true of the ectopic pelvic kidney. Such a kidney may not only be

single, but it may also be linked to Mueller's duct developmental flaws including the absence of the vagina.

Terminology

There is currently no agreed-upon terminology for blind-ending bifid ureters, and a number of seemingly unrelated words are used. These include an unfinished bifid ureter with a blind-ending pouch, an unfinished bifid ureteral diverticulum, and an unfinished collecting system duplication (ref).

Epidemiology

Present in ~5% (range 1-10%) of the population [1,2].

Gross anatomy

A bifid ureter is present when there is a duplex kidney (separate pelvicalyceal collecting systems) draining into separate ureters, but the ureters unite before draining into the bladder at a single ureteric orifice[1].

Rarely, one of the branches of a bifid ureter will be blind-ending and will not unite[2].

Differential Diagnosis

- Double ureter: persistent of ureters to drain at separate ureteric orifices in the bladder

Ureteric Compression and Obstruction from Extraneous Sources

Unfortunately, few gynaecologists are aware that a variety of disorders in the female pelvis are connected to mild ureteric obstruction:

(a) Uterine Prolapse. The main supporting element of the uterus, Mackenrodt's ligament, is significantly lengthened in complete procidentia, and when it descends with the uterus, a loop of the ureter is dragged down and rests outside the vaginal entrance. It is not surprising that this mechanism results in acute ureteral angulation and causes hydro-ureter and hydro-nephrosis. As the uterus descends, the uterine arteries get longer and may potentially compress the ureter. These patients frequently have recurrent urine infections, which when combined with ureteric blockage may severely impair their renal function and make them high-risk candidates for surgery.

(b) Pelvic Tumours. These can compress and clog the ureter, especially in the case of a myoma that fits tightly and is deeply buried in the pelvis. The similar appearance is produced by ovarian cysts, both benign and malignant, and even pelvic inflammatory disease. Before surgery, these patients should undergo a complete urological examination because about half of them would exhibit some ureteric blockage, which could very well be the cause of a post-operative urine infection in gynaecological patients. In 70% of cases, removing these tumours will return the urinary tract to normal. The cases where a section of the ureter has developed a permanent stricture as a result of pelvic inflammatory illness are the worst offenders.

Related Works

About 25% of people have bifid ureters, which are typically unilateral [5]. It results from the uretric bud's premature division close to its terminations. The kidney and the ureter grow from intermediate mesoderm in terms of their development. The metanephric kidney, which consists of two parts—collecting and secretory—began to mature in the fourth week of intrauterine life. Urine, major, and minor calyces are all part of the collecting portion. The ureteric bud's cranial end continues into the intermediate mesoderm, where it repeatedly splits and eventually gives rise to the kidney's pelvis, major and minor calyces, and collecting tubules. In this instance, a single normal ureteric bud was longitudinally split, resulting in a bifid ureter [6]. more marked. The major calices may continue to descend past the hilum for a distance before uniting to form the ureter without clearly expanding. The pelvis is absent under these circumstances; but, if the calices dilate, one or two pelvises may be present [7].

The duplex collecting system was described by Siomou E et al. [8] and Inamoto K et al. [9], however the bifid ureter and its connections to the surrounding structures highlighted in this case make it more exceptional. Such anomalies could lead to genitourinary tract anomalies such an ectopic ureter or a higher risk of discomfort, hydronephrosis, urinary tract infections, and stone formation. Results from the current study were compared with those from earlier research on the patterns of hilar structures, as can be seen. In the investigation carried out by Joao et al., [10] it was found that the renal artery's main trunk had split into three segmental branches before it reached the renal tissue. On the right side, Arora et al. noted the presence of two segmental arteries [11]. The main trunk of the right renal artery in this investigation, however, had split into four segmental branches, and the main trunk of the left renal artery had split into three segmental branches before entering the renal tissue. Such differences in the irregular pattern of the renal artery divisions in the hilar area are typically linked to renal abnormalities in the developing kidney [12].

The polar or auxiliary renal arteries were included in the 25% of numerous renal arteries presented in 2008 by Kaneko et al. This element, according to certain writers [13,14], may be a misinterpretation factor for the actual number of renal arterial divisions. 40% of people have accessory renal

arteries, which often originate from the aorta above or below the primary renal artery. The difference in the number of arteries is caused by the persistence of the lateral splanchnic arteries [15,16] or by the persistence of blood supply from a lower level than normal during the ascent of the kidney from the pelvic to the upper body. The common iliac artery branches supply the kidneys when they are located in the pelvic cavity. The kidneys' arterial supply switches from the common iliac artery to the abdominal aorta as they climb to the lumbar area. The fact that supplementary renal arteries are end arteries must be understood because if one is injured, the kidney tissue it supplies is likely to become ischaemic [17].

The right kidney's inferior suprarenal artery and the left kidney's auxiliary renal artery both

It is a rare variation when the intermediate and inferior suprarenal arteries directly emerge from the right renal artery. Such changes have not yet been recorded. According to Joao A et alanalysis .'s of the distribution of the extra parenchymal renal vein, 2.6% of the kidneys had multiple renal veins, and 7% had bifurcated renal veins. We found two divisions of the renal veins in our investigation, one in front and one behind the kidney's hilum, which was consistent with Satyapal's [18] findings from his South African study. The prevalence of the different patterns of renal veins was more prevalent on both kidneys in this study, according to a frequent observation. There are very few examples of extra renal veins, which are less common than arteries. The embryological foundation may be to blame for this. Such renal vein variations may affect the technical viability of the operation, even though they have not been mentioned in current medical literature [19].

The abnormal growth of the venous channels, which is related to the emergence of the interaction between the renal hilar structures may vary as a result of renal veins [20]. The relationship between the renal vein, renal artery, and pelvis is described in traditional anatomy texts as being antero-posterior and above downward at the hilum of each kidney. According to reports, the incidence in the classic position of the renal vein anterior to the renal artery is 65%, whereas the incidence in the position of the artery anterior to the vein is 35%.

There have been reports of variations in the arrangement of the ureter, pelvis, and renal calices. According to traditional studies, there are three possible configurations for the renal calices, pelvis, and ureter. The kidney is left in the form of a single ureter in a type IIIb birth defect where the pelvis is divided into two separate pieces. An incidence of 1% has been reported for the current case. Consequently, in this scenario, the ureter's usual physical relationship to the renal artery, renal vein, and the ureter from anterior to posterior was not observed [21].

The renal pelvis, which has a funnel form, is considered to gradually convert into the tubular proximal ureter in the UPJ [23]. According to some writers [23], it is a physiological sphincter with significant luminal folds and enhanced muscle thickness that can produce a high-pressure zone to control urine flow. The UPJ is made up of three primary layers: the inner urothelium, middle smooth muscle, and outer adventitia, just like the nearby renal pelvis and ureter. Urine is propelled from the renal pelvis to the bladder by smooth muscle contraction, which is controlled by the autonomic innervations of the body and a variety of neurotransmitters, including acetylcholine, noradrenaline, substance P, neuropeptide Y, neurokinin A, vasoactive intestinal peptide, and nitric oxide (NO).

The distal tubules of the kidney are formed from metanephric mesoderm that extends from the nephron. The ureteric bud is the embryological origin of the collecting duct and all subsequent structures, such as the major and minor calyces, renal pelvis, and ureter [26, 27]. Thus, the UPJ is only generated from the ureteric bud and does not represent an embryological fusion site.

Initial formation of the pronephros includes a nephrogenic duct that leads into the cloaca. The mesonephric duct develops from the nephrogenic duct by the fourth week of pregnancy. On the dorsomedial surface, the ureteric bud results from this. By the sixth week, the ureteric bud has grown caudally and has separated into the ureteric and Wolffian ducts at its confluence with the mesonephric duct [28].

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The Chawalla membrane, a thin band of epithelium that spans the ureteral opening, has also been observed to temporarily clog the ureter during the sixth week. Ureteral valves and ureteral stenosis have been linked to luminal obliteration or Chawalla membrane persistence.

The ureteric bud emerges from the mesonephric duct during the fifth week of gestation and is made up of a simple epithelial layer that extends into loose mesenchyme [31]. The transitional epithelium is then created as a result of epithelial cell differentiation and proliferation. Beginning in the 12th week of human pregnancy, epithelial paracrine and mesenchymal autocrine signals induce the production of smooth muscle cells from mesenchyme [32,33]. Numerous signalling molecules have been linked to the proliferation, aggregation, differentiation, and direction of smooth muscle cells as they ring the urothelial tube in mouse models. In postnatal mice (similar to the second trimester of pregnancy in humans), a second wave of smooth muscle differentiation takes place, which is controlled by the hormones [34,35].

An undifferentiated network of smooth muscle cells surrounds the unicellular epithelial tube that represents the freshly created ureter (SMC). According to research on first-trimester foetal autopsy specimens, the immature ureter is patent between gestation weeks 5 and 7 before becoming intermittently blocked by epithelial cells and going through a series of recanalization cycles as the organ extends during the elongation phase of development. By week 12 of pregnancy, the recanalization process is finished, and the normal ureter continues to develop as a hollow tube.

Although the function of this cycle of obstruction and recanalization in the development of the human foetal ureter is unknown, it does appear that it is specific to ureter morphogenesis because it has not been shown to occur in other ureteric bud-derived structures, such as the renal collecting ducts and the pelvicalyceal system. One hypothesis is that this process is necessary to change the embryonic ureter's lumen from a flat layer of cells to a stratified urothelium. The young ureter epithelium changes from a monolayer to a pseudostratified epithelium between weeks 12 and 15. This pseudostratified epithelium is characterised by the development of apical proteins termed uroplakin that impart water impermeability.

By week 22, the entire ureter is supposed to be coated in smooth muscle. SMC differentiation is believed to start in the area around the proximal ureter and renal pelvis and continue in a proximal to distal way. The spiral bundles of smooth muscle fibres that surround the growing ureter are constructed from differentiated smooth muscle cells. A second inner layer of smooth muscle is produced by week 17 and is put together into longitudinal bundles. The distal two-thirds of the ureter and the area around them are the only places where longitudinal smooth muscle fibres can be seen. They also extend into the tunnel the ureter creates as it passes through the dorsal bladder wall. The active process of urine flow from the kidney to the bladder is mediated by the ureter's peristaltic activity.

Luminal obliteration results from the proliferation and differentiation of epithelial cells to generate transitional epithelium. By physiologic recanalization of the ureter, it is repaired at the conclusion of the embryonic stage. The proliferation and differentiation of the mesenchyme into smooth muscle cells (SMC), which cluster and orient to ring the epithelial tube, are stimulated by epithelial paracrine and mesenchymal autocrine signalling [36].

It is possible to picture a signalling pathway with TSHZ3 coming after bone morphogenetic protein 4 but before myocardin and smooth muscle cell contractile protein synthesis, starting with the secretion of sonic hedgehog by the developing ureteric urothelium and ending with the differentiation of ureteric smooth muscle cells. The phenotype of TSHZ3 mutant mice is similar to that of congenital constriction of the pelviureteric junction in humans, and these people may have mutations in the genes encoding molecules involved in the Tshz3-mediated differentiation pathway. In the mouse, a regularly used experimental model, the metanephros, the precursor to the mature kidney, begins to develop between 10 and 11 days (d), which is physically similar to 24 to 30 days (d) of gestation in humans.

The ureter develops from the mesonephric duct as an epithelial protrusion, and this ureteric bud (UB) causes certain intermediate mesodermal cells to differentiate into nephrons. Following the development of the ureter's basic form, the epithelia of the ureter mature and the epithelial tube develops a layer of smooth muscle cells. Uroplakin (Upk) proteins, which form heterodimers on the apical epithelial surface, enable UB stalk epithelia to develop into a watertight barrier. In mice with null mutations of UpkIIIA or UpkII, the ureters are deformed and either reflux or become occluded by epithelial overgrowth. The ureteric lumen obstructs between days 28 and 35 of a typical human pregnancy, which is physically equal to days 10.5 to 12 in mice. Patients with anomalies like VUR, hydronephrosis, and multicystic dysplastic kidney—the latter of which is connected to a ureter without a patent lumen—have been described to have human UpkIIIA mutations.

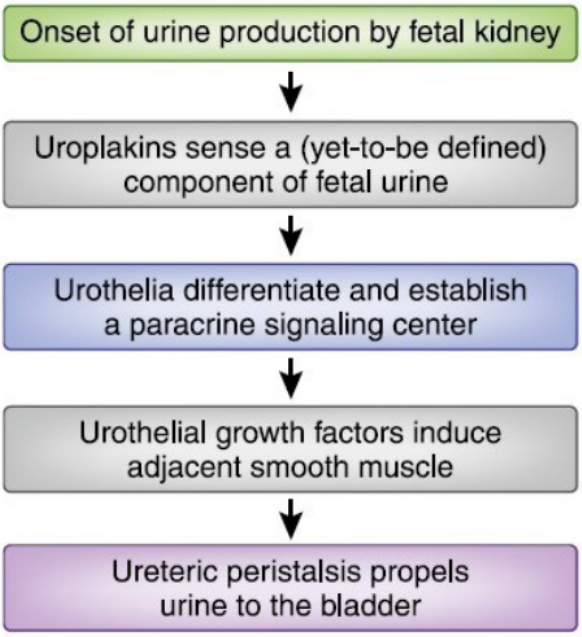

Figure 2. Harmonized development of the kidney and ureter. Woolf et al [37]

It takes synchronised smooth muscle contraction and relaxation for the mature ureter to actively push tubular fluid from the renal pelvis to the bladder (peristalsis). Along the mature ureter's length, there are various SM bundle arrangements. SM development in mice begins at embryonic day 15 in the proximal ureter closest to the kidney and progresses distally; it is characterised by an upregulation of the distinctive muscle proteins known as alpha-smooth muscle actin (SMAA). Similar to animals, spiral SM bundles are first seen in the proximal ureter of humans around 12 weeks of gestation, where muscularization also starts. Longitudinal bundles can be observed in the distal ureter wall starting at week 17, and at week 22, a muscular coat grows around the part of the ureter that crosses the bladder wall (figure 4).

An image of a mouse's late-gestation proximal ureter is shown in cross section on the left side of the image (anatomically equivalent to a human foetus the end of the first trimester). Between these two layers are stromal cells. The urothelium (blue cells) has apical Upk protein (brown line), and the mesenchyme cells that surround it have gathered around the epithelium and developed into SMCs (green) (red). In the normal state, stromal cells and SMCs both express Tshz3, while nascent urothelia express Shh and Upk. The urothelium has likewise reached maturity and begun to express Upk in the Tshz3 mutant ureter, which is seen on the right. SMCs are unable to differentiate despite the presence of mesenchymal cells (light brown) that have gathered around the urotheliu and some stromal cells that can be seen close to it.

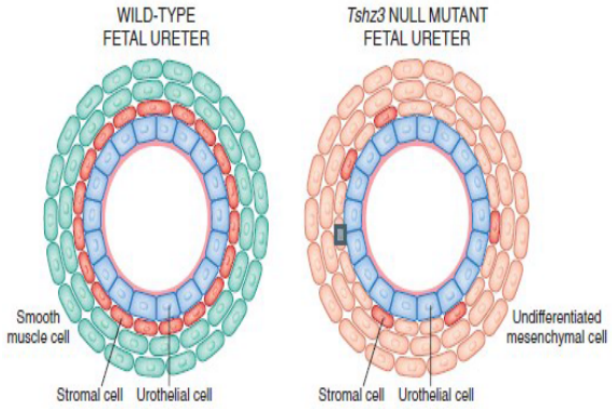

Figure 3. Cell differentiation in the proximal ureter. Adapted from Lye [38]

Sites of activity that start peristaltic waves can be found in the renal pelvis and proximal ureter; these pacemakers may be "atypical" SMCs (similar to Cajal's gut interstitial cells), which show scant immunoreactivity to SMAA and few contractile filaments. Because antibodies to Kit disrupt embryonic ureteric peristalsis, cells expressing the Kit receptor tyrosine kinase, assumed to originate from a periaortic area, surround the developing ureter and may be involved in pacemaker activity and/or wave propagation. [39] However, in vivo sensory afferents are hypothesised to influence peristalsis. It is interesting to note that embryonic ureteric proximal to distal peristalsis occurs when isolated organs are maintained in organ culture. [40]

Materials and Methods

The anatomical study of ureteric anomaly was conducted in 50 properly embalmed and formalin-fixed cadavers of both sexes to teach 1st-year MBBS students in the Department of Anatomy, from 2018 to 2022. Out of 50 cadavers, 32 were males and 18 were females. Using conventional dissection techniques, following Cunningham's practical manual, the abdomen and pelvis region of all 50 cadavers were dissected. Skin incision was followed by superficial fascia, muscles of anterior abdominal wall and peritoneum to reach up to abdominal cavity. Intra-abdominal organs were removed as the kidneys are located in the posterior abdominal wall behind the peritoneum to expose kidney-ureter-bladder (KUB) region and the dissection of kidney and ureter was carried out. Each cadaver was examined on both (right and left) sides for the presence of any anomalies in the kidney and ureter and the one showing variation was described and photographed.

Results

In the present study, as shown in Table 1, out of 100 specimens, bifid ureter was found in one cadaver (1%) only. The cadaver had unilateral bifid ureter on the right side [Figures 1-2]. The left kidney was drained by single ureter and opened into the Urinary Bladder at a single orifice. There were no other major abnormalities in the ureters. In all 100 specimens, kidneys of both sides were normal in relation to size and shape.

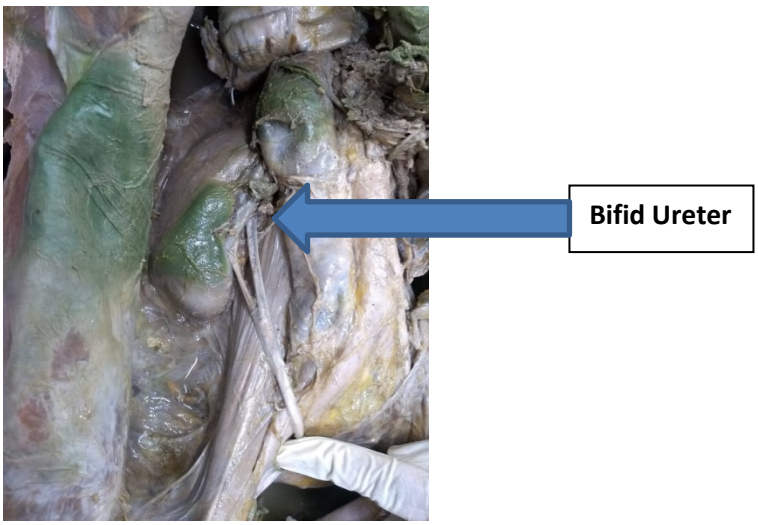

Figure 4: Right side Bifid Ureter

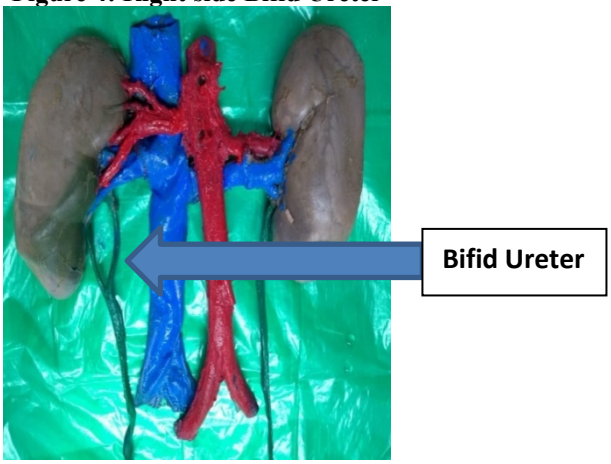

Figure 5: Right side Bifid Ureter as seen in cadaver.

Discussion

Metanephroi is the primodia of permanent kidneys. It begins to develop early in the fifth week and start to function about 4 weeks later. It has 2 components: - the metanephric diverticulum (ureteric bud) and the metanephric mass of intermediate mesoderm (metanephrogenicblastema). The ureteric bud is an outgrowth from the mesonephric duct near its entrance into the cloaca. The metanephric diverticulum (ureteric bud) is the primodium of the ureter, renal pelvis, calices and collecting tubules. As it elongates it penetrates the metanephric mass of intermediate mesoderm. The stalk of the metanephric diverticulum becomes the ureter and its expanded cranial end forms the renal pelvis. [41]

It may be discovered in childhood, less frequently in later life or may be occult and discovered at autopsy. Complications like frequent urinary tract infections; ureteric stenosis, urinary lithiasis and non-functioning of kidney units are associated with bifid ureter. The study conducted by Kulkarni et al. [42] revealed that duplication in the ureter occurs in an incidence of 0.5% and ranges from 0.5% to 3.0% and it is two to five times more common in females, common in Caucasian race. Literature also suggested that, the incidence of duplicated ureter is more common on the right side, which is just opposite in our case. In 1989, Asakawa et al. [43] reported five cases of double pelvis and ureter among 340 cadavers (1.47%, 1.8% R, 0.3% L). In our study, the prevalence of left-sided complete bifid ureter was 0.64% which was almost similar.

Marcelo Abidu-Figueiredoet all (2016) [44] reported a case report in a male cadaver fixed with a 10% formalin solution. Cadaver was dissected for teaching purposes when accidentally they found the presence of a duplex kidney collecting system - a bifid ureter on both sides. Monalisa Roy et al (2020)(45)conducted a study in the Department of Anatomy on 156 formalin-fixed cadavers of both sex and focus on the ureteric anomaly in routine dissection method. The ureter was followed from the renal pelvis to the uretrovesical junction, an opening into the urinary bladder (UB). Out of them, they got in one cadaver (0.64%) that two ureters were draining a single kidney into the UB separately and were not associated with any other congenital anomaly. There was complete duplication of ureter on unilateral side (left). Right kidney and its ureter were entirely normal.

A cadaveric study by Deka and Saikia [46] revealed out of 60 specimens, 56 (93.3%) cadaver with normal ureter and renal pelvis, whereas 4 (6.7%) specimens presented with variations of the renal pelvis and ureter. Out of these, 2 (3.3%) specimens presented with unilateral variations of ureter. As per the study of Moore and Persuad, the duplicated ureters may join together before reaching the bladder or remain separate while entering the bladder at two distinct points. [47] The variation we found in one cadaver is the complete duplication of ureter till their separate openings into the bladder.

Duplex ureters if undiagnosed pre or peroperatively can get injured during gynaecological surgeries. Alexander et al [48] has reported a case of duplex ureter which got damaged during laparoscopic hysterectomy and was diagnosed postoperatively. Surgeons performing surgeries in pelvic region should be well aware of such anomalies as congenital anomalies in genitourinary region has an incidence of about 10% [49] and duplication is one of the commonest anomalies of upper urinary tract.

Conclusion

Symptomatic patients usually have complete ureteric duplication, in which the ureters are prone to develop obstruction, reflux, and infection. [50] Duplications may be discovered in childhood less frequently, in later life often accompanied by various complications, or they may be occult and discovered at autopsy.[51-52] It is vital to pay attention to the symptoms (although it can be asymptomatic) and to diagnose other associated malformations.

References

1. Chummy S. Sinnatamby. Last's Anatomy Regional and Applied 12th edition.
2. Datta AK. Essentials of Human Anatomy (Thorax and Abdomen). 9th ed. Kolkata: Current Books International; 2014.
3. Sadler TW. Langman's Medical Embryology. 9th ed. Baltimore, Maryland, USA: Lippincott Williams & Wilkins; 2004.
4. Vishram Singh. Textbook of Clinical Embryology : Elsevier 2013 reprinted.
5. Lee McGregor's Synopsis of Surgical Anatomy 12th Ed., India, K.M.Varghese Company. 1995.
6. Anson BJ. In: Morris's Human Anatomy (12th edn.). McGraw Hill, New York, 1996; 1478.
7. Bergman RA, Thomson SA, Afifi AK, Saadeh FA. Compendium of Human Anatomic Variation.
8. Baltimore, Urban & Schwarzenberg. 1988; 81–92,171–172. [4]. Siomou E et al. Duplex collecting system diagnosed during the first 6 years of life after a first urinary tract infection: a study of 63 children, Journal of Urology. 2006;175:678-81.
9. Inamoto K, Tanaka S, Takemura K, Ikoma F. Duplication of the renal pelvis and ureter: associated anomalies and pathological conditions. Radiat Med. 1983;1:55-64.

10. Pereira-Correia JA, Valentim LS, Castro KF, Gasque GP, Celina AF, Rosário CA, Prinz RA. Analysis of renal hilum extra parenchymal structures in Brazilian adult human cadavers. Euro J. Anat. 2009;13(3):145-53.
11. Anterpreet K, Verma Poonam, Lalit Manika, et al., Variant segmental renal arteriers in the right kidney – Clinical correlations, Anat Physiol. 2012;2:3
12. Bayramoglu A, Demiryurek D, Erbilk M. Additional renal arteries and an additional renal vein associated with unrotated kidneys. Sauudi. Med. Journal. 2003;24:535-37.
13. Samul E, Miller DDDDR. Variations in the renal vascular pedicle (an anatomical and radiological study with interference to renal transplantation)Br J. Urology. 1961;33:4798-85.
14. LH JR, Flye M W, Seigler H F , Incedence of Anatomical variations in renal vasculature in the presence of normal renal function. Ann Surg 1978;188: 89.
15. Willam P, Lawrence HB, Martin MB et al. In: Gray's Anatomy (38th edn.). Churchill Livingstone, 1995; 1826.
16. Madhyastha S, Suresh R, Rao R. Multiple variations of renal vessels and ureter. Indian J Urol, 2001;17:164-5.
17. Nayak BS (2008) Multiple variations of the right renal vessels. Singapore Med J. 2008; 49: e153-e155.
18. Satyapal KS. Classification of the drainage patterns of the renal veins. J Anat 1995;186: 329-33.
19. Satyapal KS, Kalideen JM, Haffejee AA, Singh B, Robbs JV. Left renal vein variations. Surg Radiol Anat. 1999;21:77-81.
20. Trivedi S, Athavale S, Kotgiriwar S. Normal and variant anatomy of renal hilar structures and its clinical significance. Int. J. Morphol. 2011 Dec 1;29(4):1379-83.
21. Das S. Paul et al., Variation of renal hilar structures: A cadaveric case, Eur J Anat, 2006; 10(1):41.
22. M.D. Stringer, S. Yassaie, Is the pelviureteric junction an anatomical entity?, J. Pediatr. Urol. 9 (2013) 123–8.
23. Shafik, A. Al-Sherif, Ureteropelvic junction: A study of its anatomical structure and function. Ureteropelvic junction sphincter?, Eur. Urol. 1999;36: 150–6; discussion 156-7.
24. P. Santicioli, C.A. Maggi, Myogenic and neurogenic factors in the control of pyeloureteral motility and ureteral peristalsis., Pharmacol. Rev. 1998;50 683–722.
25. L. Nemeth, D.S. O'Briain, P. Puri, Demonstration of neuronal networks in the human upper urinary tract using confocal laser scanning microscopy., J. Urol. 2001;166: 255–8.
26. L. Jackson, M. Woodward, R.J. Coward, The molecular biology of pelvi-ureteric junction obstruction., Pediatr. Nephrol. 2018;33: 553–571.
27. M. Takasato, M.H. Little, The origin of the mammalian kidney: implications for recreating the kidney in vitro., Development. 2015;142: 1937–47.
28. R.G. Notley, The structural basis for normal and abnormal ureteric motility. The innervation and musculature of the human ureter., Ann. R. Coll. Surg. Engl. 1971;49: 250–67.
29. L.A. Baker, R.A. Gomez, Embryonic development of the ureter and bladder: acquisition of smooth muscle., J. Urol. 1998;160: 545–50.
30. Alcaraz, F. Vinaixa, A. Tejedo-Mateu, M.M. Forés, V. Gotzens, C.A. Mestres, J. Oliveira, P. Carretero, Obstruction and recanalization of the ureter during embryonic development., J. Urol. 1991;145: 410–6.
31. E.S. Crelin, Normal and abnormal development of ureter., Urology. 1978;12: 2–7. http://www.ncbi.nlm.nih.gov/pubmed/685004.
32. T. Matsuno, S. Tokunaka, T. Koyanagi, Muscular development in the urinary tract., J. Urol. 1984;132: 148–52.
33. C.P. Chang, B.W. McDill, J.R. Neilson, H.E. Joist, J.A. Epstein, G.R. Crabtree, F. Chen, Calcineurin is required in urinary tract mesenchyme for the development of the pyeloureteral peristaltic machinery., J. Clin. Invest. 2004;113:1051–8.
34. Y. Miyazaki, S. Tsuchida, H. Nishimura, J.C. Pope, R.C. Harris, J.M. McKanna, T. Inagami, B.L. Hogan, A. Fogo, I. Ichikawa, Angiotensin induces the urinary peristaltic machinery during the perinatal period., J. Clin. Invest. 1998;102: 1489–97.
35. A.S. Woolf, J.A. Davies, Cell Biology of Ureter Development, J. Am. Soc. Nephrol. 2013;24: 19–25.
36. Z.X. Mahoney, B. Sammut, R.J. Xavier, J. Cunningham, G. Go, K.L. Brim, T.S. Stappenbeck, J.H. Miner, W. Swat, Discs-large homolog 1 regulates smooth muscle orientation in the mouse ureter, Proc. Natl. Acad. Sci. 2006;103: 19872–19877.
37. R. Viana, E. Batourina, H. Huang, G.R. Dressler, A. Kobayashi, R.R. Behringer, E. Shapiro, T. Hensle, S. Lambert, C. Mendelsohn, The development of the bladder trigone, the center of the anti-reflux mechanism, Development. 2007;134: 3763–3769.
38. S.G. David, C. Cebrian, E.D. Vaughan, D. Herzlinger, c-kit and ureteral peristalsis., J. Urol. 2005;173: 292–5.

39. X. Caubit, C.M. Lye, E. Martin, N. Core, D.A. Long, C. Vola, D. Jenkins, A.N. Garratt, H. Skaer, A.S. Woolf, L. Fasano, Teashirt 3 is necessary for ureteral smooth muscle differentiation downstream of SHH and BMP4, Development. 2008;135: 3301–3310.
40. R. Hurtado, G. Bub, D. Herzlinger, The pelvis–kidney junction contains HCN3, a hyperpolarization-activated cation channel that triggers ureter peristalsis, Kidney Int. 2010; 77:500–508.
41. Moore Persaud. The Developing Embryo. Clinically Oriented Embryology.7th Edition.
42. Kulkarni V, Ramesh BR, Prakash BS. Bilateral bifid ureter with accessory renal artery: A case report. Int J Basic Med Sci 2012;3:67- 70.
43. Asakawa M, Kubodera T, Okamura Y, Habara K, Ito H. Five cases of the double renal pelvis and ureter. Kaibogaku Zasshi 1989;64:206-9.
44. Marcelo Abidu-Figueiredo, Noemi S. Barbuto, Lucas AS. Pires, Márcio A. Bilateral bifid ureter: Case report and clinical discussion International Journal of Medical and Health Research. October 2016; 2(10): 12-14
45. Roy M, Singh BR, Gajbe UL, Thute P. Anatomical variations of ureter in central India: A cadaveric study. J DattaMegheInst Med SciUniv. 2017;12:277-9.
46. 46.Deka B, Saikia R. A study of human cadaveric ureter by simple dissection method.Int J Anat Res. 2016;4:3005-8.
47. Moore KL, Persuad TV. The Developing Human Clinically Oriented Embryology. 8th ed. Saunders, Elsevier; 2009.
48. Alexander P. B. Dalzell, Richard G. Robinson, Kirsten M. Crooke(2) Pelviperineology. Duplex ureter damaged during laparoscopic hysterectomy. 2010; 29:
49. 49.Burkland, C. E. The significance of genetic and environmental factors in urogenital disease. Journal of Urology, 1958;79:532.
50. Sailaja TK, Rajasree TK. A rare anomaly of ureter and its developmental aspect – A case report. MRIMS J Health Sci. 2014;2:102-4.
51. Amitai M, Hertz M, Jonas P, Apter S, Heyman Z. Ectopic ureterocele in adults with a comparison of the anomaly in children. UrolRadiol. 1992;13:181-6.
52. Cronan JJ, Amis ES, Zeman RK, Dorfman GS. Obstruction of the upper-pole moiety in renal duplication in adults: CT evaluation. Radiology. 1986;161:17-21.