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Case Report

A unique confluence of renal anatomical variations found during cadaveric dissection: A case report

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Abstract

Anatomical variations in the renal and urinary systems, though often asymptomatic, hold significant clinical and surgical relevance. Variants such as bifid ureter, aberrant renal vessels, and polycystic kidneys may complicate diagnostic and operative procedures if unrecognized.

During a routine cadaveric dissection conducted in the Department of Rachana Sharir, anatomical variations were noted in a formalin-fixed 72 yrs old male cadaver of Indian origin. Following reflection of the posterior peritoneum and removal of overlying fascia and fat, the kidneys, ureters, renal vessels, and surrounding structures were carefully examined and documented. The cadaver presented with four notable renal anatomical variations: (1) Incomplete Bifid Ureter (Left Side, Unilateral): a unilateral left-sided incomplete bifid ureter, with two ureteric branches fusing 3.5 cm from the renal pelvis; (2) Multiple Tributaries of the Left Renal Vein: three distinct tributaries—designated as LRV1, LRV2, and LRV3—were observed draining the left kidney. These tributaries converged to form a single venous trunk, which subsequently drained into the inferior vena cava. (3) Early Division of the Left Renal Artery: the left renal artery was noted to bifurcate into segmental branches prior to its entry into the renal hilum, indicating an early prehilar division and (4) Bilateral polycystic kidneys, each containing 10–15 cysts, varying from 0.5 to 3.5 cm in depth, with dark, viscous fluid content. The coexistence of multiple renal anatomical variations in a single individual is rare and underscores the importance of awareness in clinical and surgical settings. Thorough anatomical knowledge is crucial in preventing iatrogenic complications during renal surgeries, transplants, or radiological interventions. Cadaveric studies remain invaluable for understanding such variations.

Keywords: Bifid ureter, Renal vein variation, Early artery division, Polycystic kidney, Cadaveric dissection

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Introduction

Anatomical variations of the renal and urinary systems are not uncommon and have significant clinical and surgical relevance. These variations, often asymptomatic during life, are frequently encountered incidentally during radiological investigations or cadaveric dissections. Among the notable variations are duplications of the ureter, aberrant renal vessels, and morphological abnormalities of the kidneys. Embryologically, the development of the renal and urinary systems involves intricate interactions between the metanephric blastema and the ureteric bud, which arise from the intermediate mesoderm. Incomplete fusion or duplication of the ureteric buds can lead to bifid or duplicated ureters. Early branching of the renal artery reflects aberrant angiogenesis during the formation of the renal vascular tree, while the development of polycystic kidneys results from mutations affecting tubular epithelial differentiation and morphogenesis.

A bifid ureter, arising from the incomplete fusion of ureteric buds during embryonic development, represents a relatively common congenital anomaly. It may occur unilaterally or bilaterally and is characterized by varying degrees of ureteral duplication, ranging from partial bifurcation to complete duplication. While often clinically silent, it may predispose individuals to urinary tract infections or obstruction.

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Variations in renal venous anatomy—particularly on the left side—are well-documented and attributed to the more intricate embryological development of the left renal vein. These variations may include multiple tributaries, retroaortic or circumaortic courses, and atypical drainage patterns into the inferior vena cava. The presence of multiple tributaries emerging from the kidney and converging into a single renal vein can pose challenges during retroperitoneal surgeries or renal transplantation.

Polycystic kidney disease (PKD) is a hereditary and progressive disorder characterized by the presence of multiple fluid-filled cysts in both kidneys. Over time, the increasing number and size of these cysts can compromise normal renal architecture and function, potentially leading to renal insufficiency or end-stage renal disease. Although it is more commonly diagnosed clinically or radiologically in living patients, its recognition during dissection offers valuable insights into the disease's gross pathology.

An early division of the renal artery, wherein the vessel branches into segmental arteries before reaching the renal hilum, is of notable clinical significance. This anatomical variation is particularly important during renal surgeries, transplantations, and angiographic procedures, as it increases the risk of inadvertent ligation, injury, or incomplete perfusion of renal segments if not properly identified and managed preoperatively.

Cadaveric studies provide a valuable opportunity to identify and document such variations, contributing to the anatomical knowledge base and offering insight into their potential clinical implications. This case report presents a unique confluence of these renal anatomical variations—bifid ureter, multiple venous tributaries draining into a single left renal vein, bilateral polycystic kidneys, and an early division of left renal artery—identified during routine cadaveric dissection. The coexistence of such variations in a single individual underscores the importance of awareness and documentation of anatomical diversity in medical education and clinical practice.

1. Materials and Methods

During a routine dissection conducted at the Department of Rachana Sharir a confluence of Renal anatomical variations were observed. The cadaver, donated to the department, belonged to 72 yr old aged Indian male. Following the dissection of the anterior abdominal wall and the division of the root of the mesentery, all intraperitoneal abdominal organs were systematically removed. Subsequently, the posterior parietal peritoneum was carefully reflected, and the overlying fat and fascia on the anterior surfaces of both kidneys were meticulously dissected and cleared to facilitate detailed anatomical observation. Subsequently, the renal veins, renal arteries, course of ureter from each kidney were carefully traced and examined.

2.1. Observed variations

During routine dissection of this 72 yr old formalin-fixed male cadaver we found a confluence of variations mentioned as.

1. **Variation found in ureter:** A Unilateral Incomplete bifurcation of Ureter was observed in this cadaver. The left ureter was partially split into two (**Figure 1 a**). The ureter on the left side displayed a duplication up to a length of 3.5 cm, after which the two ureteric branches fused to form a single ureter. This single ureter then opens into the urinary bladder through its left lateral trigonal angle. The total length of the ureter, including the renal pelvis, measures 25 cm, with the split region accounting for 3.5 cm. In this anatomical variation, two distinct ureteric pathways are observed emerging from the left renal pelvis, following separate courses initially before fusing distally. The right renal pelvis and ureter appear normal (**Figure 1 b**).

2. **Variation found in renal vein:** In this cadaver we found unilateral Left sided multiple renal veins arising from the Kidney mentioned in figure as LRV1, LRV2, LRV3 in which LRV 2 &3 joined to form a single vein which later joins with LRV1 and form a common Left Renal Vein which drains into IVC as shown in (**Figure 2**).

3. **Variation found in renal artery:** In this cadaver we found unilateral left sided earlier division of LRA (Left Renal Artery) into segmental arteries as mentioned in (**Figure 3** & **Figure 4**) as ALRA (Accessory left renal artery) and LRA (Left renal artery).

4. **Pathology found in kidney:** In this cadaver, during dissection we found bilateral Polycystic Kidneys with multiple cyst as shown in (**Figure 5** & **Figure 6**). The depth of largest cyst was found to be 3.5 cm. All the cysts were greyish-black in color, with the fluid inside them being dark in color and viscous in consistency. This unique appearance and consistency of the cysts may suggest a pathological variation, potentially indicative of a more severe or advanced form of cystic kidney disease. There were almost 12-15 cysts on Left Kidney and 10-12 cysts on Right Kidney. The depth of cysts in Left and Right Kidneys varies from 0.5 cm to 3.5 cm.

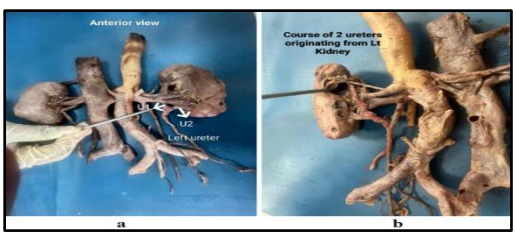

Figure 1: a & b left ureter was partially split into two

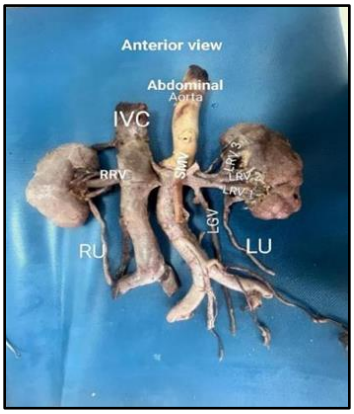

Figure 2: Common left renal vein which drains into IVC

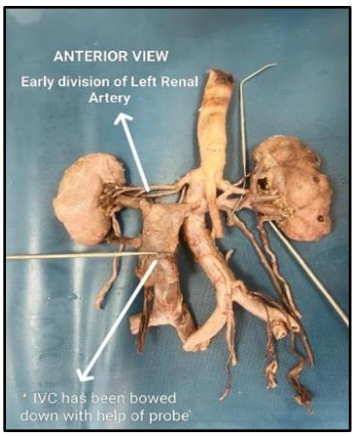

Figure 3: ALRA (Accessory left renal artery)

Figure 4: LRA (Left renal artery)

Figure 5: Cyst found in anterior view of polycytic kidney

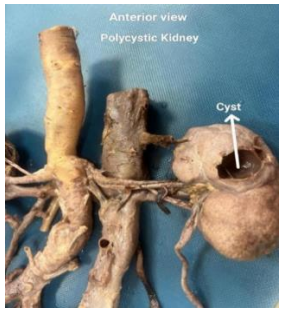

Figure 6: Cyst found in anterior view of polycytic kidney

2. Discussion

The discussion for the above variations have been discussed under individual category of variations found as-

3.1. Variation in ureter

The incidence of unilateral complete double ureter, bifid ureter, and megaureter has been reported as 0.62%, 0.62%, and 1.25%, respectively.[1] The ureter begins to develop during the fifth week of intrauterine life, originating as a diverticulum from the dorsomedial aspect of the mesonephric duct, known as the ureteric bud. This bud extends cranially toward the metanephric mesenchyme. Upon contact with the metanephric blastema, the ureteric bud undergoes bifurcation, a process regulated by glial cell line-derived neurotrophic factor (GDNF). Further branching of the ureteric bud, along with interactions with the surrounding mesenchyme, gives rise to the structural components of the kidney, including the major and minor calyces. In early development, the ureteric wall is highly permeable, and its lumen initially becomes obliterated before undergoing recanalization. This process starts in the mid-portion of the ureter and progresses both cranially and caudally. Additionally, two fusiform dilatations emerge at the lumbar and pelvic regions of the developing ureter (Chawla, Gupta, Singh et al., 2014). A deficiency of glial-derived neurotrophic factor (GDNF) or premature branching of the ureteric bud before it reaches the nephrogenic tissue can lead to the development of an incomplete or bifid ureter. In most cases of duplication, the ureters are incompletely duplicated, joining above their origin in the bladder. Such anomalies are often associated with ureteroureteral reflux (WU) (SNOW and Taylor, 1986). A duplex urinary tract is prone to complications such as infection, typically due to obstruction associated with an ectopic ureter or vesicoureteric reflux.[2] These conditions usually diagnosed in adults can compromise normal urinary flow and increase susceptibility to hydronephrosis and urinary tract infectis (onUTIs).[3]

3.2. Variation in renal vein

The overall prevalence of multiple renal veins is 16.7% (ranging from 14.3% to 19.2%), with a significantly higher occurrence on the right side (16.6%, ranging from 14.2% to 19.1%) compared to the left side (2.1%, ranging from 1.3% to 3.2%).[4] The occurrence of congenital variations in the

renal veins can be attributed to the complexity of their embryological development, which is closely linked to the formation of the inferior vena cava (IVC). This developmental process begins around the 4th week of gestation and is typically completed by the 8th week. During this period, a network of three paired, longitudinally arranged veins—the posterior cardinal, subcardinal, and supracardinal veins—develop in parallel and are interconnected. Initially, the venous system is bilaterally symmetrical. However, through a series of selective regressions and anastomoses, it transforms into a predominantly right-sided structure, forming the definitive IVC. In the early stages, each side of the embryo possesses two renal veins—one ventral and one dorsal. These typically converge to form a single renal vein. If both the ventral and dorsal veins persist instead of one regressing, accessory or multiple renal veins may result. The definitive renal veins are formed from the anastomoses between the subcardinal and supracardinal veins. Normally, the dorsal renal vein regresses while the ventral vein persists to become the mature renal vein. Variations arise when this pattern of regression and persistence deviates from the typical course.[5]

3.3. Variation in renal arteries

An early division of the renal artery was observed in 13% of individuals. Of these, 5% had early division on the right side, 7% on the left side, and 1% exhibited early division on both sides. A similar study showed that the early division of the renal artery into segmental arteries was observed in 8% of individuals.[6] A communication delay between the mesenchyme of the blood vessel and the mesenchyme of the metanephros, possibly influenced by factors such as glial-derived neurotrophic factor (GDNF) and hepatocyte growth factor (HGF), may contribute to the early division of the renal artery.[7] When comes to the early division of renal artery the current study correlating with the Budhiraja et al (2010) observed pre-hilar branching pattern in 11% of cases which were duplicated and triplicate.[8]

3. Discussion on Polycystic Kidney

The majority of patients with ADPKD have few or no symptoms at the time of diagnosis. When symptoms do occur, they typically begin between 30 to 50 years of age, and most commonly include acute abdominal or flank pain.[9] Polycystic Kidney Disease (PKD) encompasses a class of disorders presenting with bilateral cyst formation in the kidney. PKD can be inherited as a dominant (ADPKD) or a recessive (ARPKD) trait, due to mutations into multiple genes, the most frequent being *PKD1*, *PKD2* and *PKHD1*. The protein products of these genes (polycystin-1, polycystin-2 and fibrocystin, respectively) have been shown reside within the primary cilium or to be important for the maturation and trafficking of proteins to the primary cilium. The primary cilium is an organelle protruding from the apical surfaces of renal epithelial cells that functions to sense extracellular signals and translate them into intracellular biochemical information. PKD represents the most common monogenic disorder affecting the kidney and the most common manifestation of human ciliopathies.[10]

4. Conclusion

Anatomical variations like duplex or bifid ureters and renal vascular anomalies hold crucial clinical importance, especially in surgeries like transplantation, nephrectomy, or ureteral preimplantation. These can lead to complications such as obstruction, infections, or altered hilar anatomy,[11] necessitating precise preoperative imaging. Duplex ureters are more common in females; finding one on the left in a male cadaver is rare. In conditions like ADPKD, such anomalies can worsen outcomes and complicate treatment. Early identification helps guide interventions and reduce iatrogenic risks. Understanding these variants ensures safer and more effective clinical decision.

5. Source of Funding

None.

6. Conflict of Interest

None.

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