

## A study of congenital renal anomalies in adult cadavers

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### Abstract

**Introduction:** Congenital abnormalities of the kidneys and urinary tract play a major role in the morbidity and mortality. Many of these renal anomalies predispose to obstruction which may lead to renal failure. We had in our study observed the different malformations in human kidneys among the adult human cadavers.

**Materials and Method:** 50 cadavers who died of renal failure and were scheduled for post mortem were included in the study. The position of the suprarenal gland and the upper poles of the kidneys, the size, shape and the arrangement of the attached structures such as the hilum, ureter, bladder abdominal aorta and the inferior vena cava were noted and recorded.

**Results:** Out of the 50 cadavers that were included into the study, 5 of them had congenital renal anomaly accounting for 10% of the deaths due to renal failure in adults. All the patients were between 40–60 years of age. There were two cases of lobulated kidney, one horse – shoe shaped kidney, one case of congenital hypoplasia and one 7 shaped left kidney.

**Conclusion:** Renal anomalies are one of the common congenital anomalies which may remain unnoticed till adulthood. Of them, renal agenesis, horseshoe kidneys, renal hypoplasia and lobulated kidneys are relatively predominant.

**Keywords:** Congenital renal anomalies, Cadavers, renal hypoplasia, Lobulated kidney, Horseshoe kidney.

### Introduction

Congenital abnormalities of the kidneys and urinary tract play a major role in the morbidity and mortality. It accounts for approximately 3.3-11.1% incidence in the general population and about 50% of all the congenital abnormalities.<sup>(1)</sup> Congenital kidney abnormalities are the leading causes of end stage renal disease in children and subsequent problems in adulthood. Many of these renal anomalies predispose to obstruction which may lead to renal failure.<sup>(2)</sup> Many of the anomalies range from mild, asymptomatic malformations such as double ureters or minimum renal pelvic obstructions to severe pathologies such as renal agenesis, renal dysplasia, horse shoe shaped kidneys etc., which are many times fatal.<sup>(2)</sup>

Congenital anomalies of the kidney is usually found associated with the urinary tract (CAKUT). This comprises of a broad spectrum of renal and urinary malformations which can range from complete renal agenesis to hypodysplasia, multicystic kidney dysplasia, duplex renal collecting system, ureteropelvic junction obstruction (UPJO), megaureter etc.<sup>(3,4)</sup>

Most of the time, these abnormalities are associated with each other and take up a familial pattern, with complete and variable penetrance resulting in different anatomical pattern.<sup>(5)</sup> It is therefore estimated that there is a common pathological mechanism and genetic cause for all the anomalies of the renal system.

One of the most common renal fusion anomalies observed is the horseshoe kidney which is estimated to occur in 1 per 400 people and was seen more common in male than in females.<sup>(6-8)</sup> Male preponderance is seen in renal agenesis also. Unilateral renal agenesis commonly affects approximately 1 in 500 live births, while bilateral is more rare.<sup>(9)</sup>

Hypoplasia usually occurs due to inadequate ureteral bud branching and results in a small kidney with histologically normal nephrons, though few in number. In case of oligomeganephronia, these nephrons are highly enlarged.<sup>(10)</sup>

We had in our study observed the different malformations in human kidneys among the adult human cadavers.

### Materials and Method

This study was conducted by the Department of Anatomy at Medicity institute of medical sciences and Gandhi Medical College during the period of two years. 50 cadavers who died of renal failure and were scheduled for post mortem were included in the study. The postmortem was conducted by the Department of Forensic Medicine and the relevant information was collected and noted from them.

All the cadavers were properly numbered and labeled and checked for any gross external abnormalities. The age, sex and height of the specimens were noted. If any external anomaly was observed, the cadavers were excluded from the study.

They were all then embalmed and kept in the tank solution for dissection, which was performed after 4-5 days.

The dissection was performed with a midline incision from supra-sternal notch to symphysis pubis and two transverse incision from the umbilicus laterally as far as possible to expose the abdominal and the thoracic cavities completely. The position of the suprarenal gland and the upper poles of the kidneys were noted and recorded. The size, shape and the kidneys were also recorded. The arrangement of the attached structures such as the hilum, ureter, bladder

abdominal aorta and the inferior vena cava were also noted and recorded.

The cause of the renal failure was estimated. It the cause was not due to a congenital anomaly, the cadaver was discarded.

## Results

Out of the 50 cadavers that were included into the study, 5 of them had congenital renal anomaly accounting for 10% of the deaths due to renal failure in adults. All the patients were between 40 – 60 years of age.

There were two cases of lobulated kidney, one horse – shoe shaped kidney, one case of congenital hypoplasia and one 7 shaped left kidney.

The patient with horse shoe kidney was 55 years old. There were no external anomalies, but the corresponding renal arteries were aberrant. The upper poles of the kidneys were wide apart from each other and were fixed in the midline of the lower plane by a flat broad isthmus, with inferior mesenteric vessels running in front of it. The renal arteries were found arising from the right and left common iliac arteries, and the renal veins were opening into the common iliac veins.

One patient had congenital hypoplasia of the left kidney with a narrow ureter. The size and shape of the right kidney was normal and placed between T12-L3, while the left kidney was placed at the level of T11-L1. The size of the left kidney was small measuring 7 x 4 x 2 cm in comparison to a normal kidney of 12 x 5 x 2cm. The length of the ureter was normal of 32cms but the girth or the diameter was very narrow. It opened into the bladder at left lateral trigone of the bladder through a narrow orifice (Fig. 1).



**Fig. 1: Congenital hypoplasia with narrow ureter**

A 40 year old male had a 7 or inverted L shaped left kidney, measuring 15 x 5 x 4cm and occupying the left lumbar region and the epigastric region, due to its large size. The right kidney and its associated structures were normal in size and presentation. The left ureter was slightly larger than the right ureter, though its length was normal (Fig. 2).



**Fig. 2: 7- shaped kidney**

2 cadavers had lobulated kidneys, a 60 year old male had left kidney lobulated while the other one was a 50 year old female who had a right lobulated kidney.

The left kidney in the first case showed 4 lobes measuring 16x3x2cms at the level of T11-L3 vertebrae, with vascular anomalies. The two accessory renal arteries were seen arising from the abdominal aorta, one above and one below the renal artery. The upper artery entered the kidney in its upper pole, while the lower entered the kidney through the lower pole. The right renal artery entering through the middle of the hilum was posterior to the renal vein

In the second case, there were no external abnormalities. Internally, there were three lobes on the right kidney of the female cadaver. There were two accessory renal arteries arising from the abdominal aorta. As in the earlier case, the upper artery entered the kidney in its upper pole, while the lower entered the kidney through the lower pole. The right renal artery entering through the middle of the hilum was posterior to the renal vein (Fig. 3).



**Fig. 3: Lobulated kidney**

## Discussion

Renal malformation is the set of aberrations which develop during the fetal stage and form major structural and anatomical anomalies.<sup>(1)</sup> The most serious of the malformations was absence of the kidney or agenesis.

In our study out of 10 cadavers, 5 of them had renal anomalies, accounting for 10% of the cases. Of

them, two were lobulated kidneys, one was horse shoe kidney and one was congenital hypoplasia.

Renal agenesis is a non-development of the kidney. It may be unilateral or bilateral.<sup>(12)</sup> Bilateral agenesis is normally a part of a syndrome of oligohydramnios, pulmonary hypoplasia, and extremity and facial anomalies. Normally this presentation is fatal.

Unilateral agenesis is comparatively common accounting for 5% of the total renal anomalies. Many cases result from complete involution in utero of a multicystic dysplastic kidney. It usually is accompanied by ureteral agenesis with absence of the ipsilateral trigone and ureteral orifice.<sup>(13)</sup>

When renal agenesis is an isolated anomaly, it is normally asymptomatic. Our study showed no incidence of renal agenesis.

Anin Barket et al stated that anomalies of the urinary tract rank 3<sup>rd</sup> to 4<sup>th</sup> among the congenital anomalies and occurs in 10% of the population. Anomalies were generally due to the interaction between environmental factors such as maternal illness and exposure to toxic agents. It was observed that 15% of the urogenital anomalies were due to chromosomal aberrations 10% multifactorial, 9% due to teratogenic factors. According to them, the critical period of urinary tract is 15 – 94 days.<sup>(1)</sup>

In another study by Pankau et al, renal abnormalities in Williams Beuren Syndrome was 17.7% high in comparison to 1.5% in normal population. The spectrum of these abnormalities ranged from minor anomalies such as bladder diverticula to more serious malformations such as renal aplasia or hypoplasia, duplicated kidney and renal agenesis.<sup>(14)</sup>

Accessory renal arteries are known to be common and usually arise from the aorta above or below the renal artery and follow till the renal hilum and are regarded as persistent embryonic lateral splanchnic arteries.<sup>(15)</sup> In our study, one renal artery and two accessory arteries were seen entering the kidneys in the lobulated kidney cases which was similar to a study by Vikram Rao et al, who observed 28% of the cadavers had accessory arteries and 12% of the cases a double renal artery was seen supplying the kidneys.<sup>(15)</sup> This was similar to another study by Khamanarong et al where 17% of the patients had double arteries.<sup>(16)</sup> In other cases, an incidence of extrarenal arteries were observed in 9-76% of the cases among cadavers.<sup>(17)</sup>

Renal hypoplasia is a mal development of the kidney that effects its size, shape or structure. Normal renal development is initiated by penetration of metanephric blastema. True hypoplasia is restricted to describe those small kidneys that have less than the normal number of calyces and nephrons but are not embryonic.

In our study we found only one case of renal hypoplasia among 50 cadavers. Rubenstein et al found an incidence of 2.5% of true hypoplasia.<sup>(18)</sup>

Horseshoe kidney is the most common fusion abnormalities in the kidney and is estimated to occur in 1 in 400 people with a predominance in males.<sup>(6-8)</sup> Horseshoe kidneys may be a result of teratogenic factors, which are responsible for the increase in the incidence of related congenital anomalies and nephroblastoma<sup>(19)</sup> and is often associated with hydronephrosis and renal calculi.<sup>(20,21)</sup>

Although sometimes horseshoe kidneys are associated with other anomalies, they occur as isolated malformations also. In our study, we observed one case of horseshoe kidney in a male. Horseshoe shaped kidney was observed by Ongetti et al in their study but this was associated with a rare bilateral ureteral duplication.<sup>(22)</sup>

The kidneys develop in several lobules that fuse as they develop and grow. Incomplete fusion of these renal lobules can persist postnatally and may be observed in adults as lobulated kidneys. Normally though this condition may be apparent in new born, as the baby grows and new cells are formed, these lobules disappear. In very rare cases they may persist, forming abnormalities. These are fairly symmetrical, limited to lower and middle part of the kidney. These are sharply angulated surface indentations (notches) between the calyces rather than opposite a calyx.

In our study we had observed 2 cases (4%) of cases with lobulated kidneys. More et al in their study observed lobulation in 5% of the right kidneys and 10% of the left kidneys.<sup>(23)</sup> Similar case was observed by Patil et al in a rare congenital condition of kidney where bilateral lobulation and malrotation were observed in association with hilar structure of the kidneys.<sup>(24)</sup>

## Conclusion

Renal anomalies are one of the common congenital anomalies inherited by the offspring. Many times it may remain unnoticed till adulthood. Of them, renal agenesis, horseshoe kidneys, renal hypoplasia and lobulated kidneys are relatively predominant. Studies have isolated the gene responsible for the heredity of congenital malformations of the kidneys. As a result, the diagnosis for the clinician to detect the malformations has become relatively easy and play an important role in treatment.

## References

1. Barakat AJ, Drougas JG. Occurrence of congenital abnormalities of kidney and urinary tract in 13,775 autopsies. *Urology*. 1991;38(4):347–350.
2. Rodriguez MM., Congenital Anomalies of the Kidney and the Urinary Tract (CAKUT). *Fetal Pediatr Pathol*. 2014 Dec;33(5-6):293–320
3. Cassandra L. Kniffin. Congenital anomalies of kidney and urinary tract. <http://www.omim.org/entry/610805>.
4. R. Song and I. V. Yosypiv, "Genetics of congenital anomalies of the kidney and urinary tract," *Pediatric Nephrology*, vol. 26, no. 3, pp. 353–364, 2011.
5. Squiers EC, Morden RS, Bernstein J. Renal multicystic dysplasia: an occasional manifestation of the hereditary

- renal adysplasia syndrome. *Am J Med Genet* 1987; 3 [Suppl]: 279.
6. Evans RM. Percutaneous access in difficult kidney. In: Sosa RE, ed., *Textbook of Endourology*. 1st Ed., Philadelphia, WB Saunders. 1997;114–128.
  7. Glodny B, Petersen J, Hofmann KJ, Schenk C, Herwig R, Trieb T, Koppelstaetter C, Steingruber I, Rehder P. Kidney fusion anomalies revisited: clinical and radiological analysis of 209 cases of crossed fused ectopia and horseshoe kidney. *BJU Int*. 2009;103:224–235.
  8. Casey GP, Sedlmayr JC, Oliver P, Cork J. Gross anatomy of a horseshoe kidney: a case report. *FASEB J*. 2009;23:823.8.
  9. Andrews M, Esedov R. Renal agenesis. <http://radiopaedia.org/articles/renal-agenesis>.
  10. Woolf AS. Multiplic causes of human kidney malformations. *Arch Dis Child* 1997;77:471-474.
  11. Potter EL (1972) Normal and abnormal development of the kidney. (Year Book Medical Publishers, Chicago).
  12. A Mishra. Renal agenesis: report of an interesting case. *The British Journal of Radiology*, 80 (2007), e167–e169.
  13. Rabinowitz R, Cubillos J., Renal Anomalies., <http://www.msmanuals.com/professional/pediatrics/congenital-renal-and-genitourinary-anomalies/renal-anomalies>.
  14. Pankau R, Partsch CJ, Winter M, Gosch A, Wessel A. Incidence and spectrum of renal abnormalities in Williams-Beuren syndrome. *Am J Med Genet* 1996;63:301-4.
  15. Vikram Rao KE, Sadananda Rao B., A study of renal artery variations in cadavers. *Asian Pac. J. Health Sci.*, 2015; 2(4):55-61.
  16. Khamanarong K, Prachaney P, Utravichien A, Tong Un T, Shripaoraya Lx. Anatomy of Renal arterial supply. *Clinical anatomy* 2004;17(4):334-336.
  17. Ozkan Ugur, Ozkurt Levent, Tercan Fahri, Osman Kızılkılıç, Koç Zafer. Nihal Koca Renal artery origins and variations: angiographic evaluation of 855 consecutive patients. *Diagn Interv Radiol*. 2006;12:183–86.
  18. Rubenstein M, Meyer. Congenital abnormalities of the urinary system, renal hypoplasia. *JPaed*. 1961;58:356.
  19. Hohenfellner M, Schultz-Lampel D, Lampel A, Steinbach F, Cramer BM, Thuroff JW. Tumor in the horseshoe kidney: clinical implications and review of embryogenesis. *J Urol*. 1992;147:1098–1102.
  20. Pitts WR Jr, Muecke EC. Horseshoe kidneys: a 40-year experience. *J Urol*. 1975;113:743–746.
  21. Lampel A, Hohenfellner M, Schultz-Lampel D, Lazica M, Bohnen K, Thuroff JW. Urolithiasis in horseshoe kidneys: Therapeutic management. *Urology*. 1996;47:182–186.
  22. Ongetti KW, Ogeng’O J, Saidi H., A horse shoe kidney with partial duplex systems. *IJAV*. 2011;4:55-56.
  23. Manisha S More, Manoj D Togale, Shilpa Bhimalli, Daksha Dixit, S P Desai human adult cadaveric kidneys. *Med Pulse – International Medical Journal* June 2015;2(6):364-368.
  24. Patil ST, Meshram MM, Kasote AP. Bilateral malrotation and lobulation of kidney with altered hilar anatomy: a rare congenital variation. *Surg Radiol Anat* 2011;33(10):941-4.