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CASE STUDY

HISTOLOGY OF CONGENITAL POLYCYSTIC KIDNEY-A STUDY ON FOETUSES

[1]Rapotra, M., *[,2]Sharma, A. and [2]Sharma, M.

[1]Department of Anatomy, Pandit Jawahar Lal Nehru Government Medical College and Hospital, Chamba, Himachal Pradesh 176310, India
[2]Department of Anatomy, Government Medical College and Hospital, Chandigarh 160030, India

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ABSTRACT

Multicystic disease of the kidney is a common cause of enlarged abdomen in fetus. It is a congenital mal development, where the renal cortex is replaced by numerous cysts of varying sizes. Current study was carried out in the Department of Anatomy, Government Medical College & Hospital, Chandigarh. It is important to know the normal developmental anatomy and histogenesis of urinary system for better understanding of various congenital renal conditions.

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INTRODUCTION

Polycystic kidney disease is a rare developmental anomaly inherited as autosomal dominant or autosomal recessive trait. ARPKD is relatively uncommon and occurs primarily in neonates and children. It is characterized by fluid filled cystic dilatation of the collecting ducts frequently associated with hepatic involvement and progression to renal failure.Itis included in the diffrential diagnosis of cystic diseases of the kidney (Sunita B. Patil *et al*., 2013). The incidence range of ARPKD is one in 6,000 to one in 40,000 live births (Zerres *et al*., 1996; Zerres *et al*., 1998). Multicystic kidney is the congenital renal cystic malformation most frequently diagnosed in children, with an estimated incidence of 1:1,0001 to 1:4,3002 live births. It is also the most common cystic anomaly detected by fetal ultrasonography and the second most frequent cause of palpable abdominal mass in newborns and infants (Farmer, 2000). It is important to know the normal developmental anatomy and histogenesis of urinary system for better understanding of various congenital renal conditions. Kidney plays a vital role in development of fetuses (Khayati Sant Ram, 2015). In the present study we compared the histology of normal kidneys with polycystic kidneys. We reasoned that such comparison may provide clues concerning the pathogenesis of abnormal kidneys.

Corresponding author:* **Sharma, A.
Department of Anatomy, Government Medical College and Hospital, Chandigarh 160030, India.

MATERIALS AND METHODS

Telltoatal no of foetusesamongst which you found 22 aborted cases

Current study was carried out in the Department of Anatomy, Government Medical College & Hospital, Chandigarh. The material for the study consisted of1100 aborted human fetalspecimens from 11th to 25th weeks of gestational ages. Out of which 22 fetuses having polycystic kidney, 13 were males and 9 were female. Fetuseswere sent by the Department of Obstetrics & Gynaecology of the same institute for routine autopsy. Fetuses with the abnormal kidney findings on USG were included in the study. All the fetuses were reported to have no growth retardation on USG. All fetuses were result of the intra uterine death or spontaneous abortion.Written consent was taken from the parents to perform autopsy and relevant maternal and family history along with antenatal USG findings were recorded. During autopsy external findings observed were sex of the fetus, gestational age of fetus by fetal weight, Head circumference, Abdominal circumference and crown rump length.Other associated anomalies were also noted. Theabdomenwasopened by median incision extending from the juglar notch to pubicsymphysis. Ongross examination, the kidneys were grossly dilated and multiple cysts were seen in almost all cases. To compare the histological changes in multicystic dysplastic kidney we also do the histology of normal fetal kidneys. The fetuses were divided into three groups according to the gestational age i.e. 11-18 weeks, >18-

25 weeks and >25weeks. The kidneys were then dissected and fixed in 10% formalin for 48-72 hours, and processed for paraff insections of 5mm thickness. The sections were stained for lightmicroscopic study with haematoxylin and eosinHistological slideswere made with Haematoxylin and eosin.

RESULTS

On gross examination, Thefetuses did not show any signs of intra uterine growth retardation. Both upper and lower limbs were normal and there were no amniotic bands. The vertebral column was found to be normalnormal. Ongross examination, the kidneys were larger in size and cystic in all cases. Upon opening fetal kidneys, the kidneys showed multiple cysts of various sizes. The cyst wall was not very thick and cysts were translucent white and contained clear white fluid.Normal renalanatomy wascompletely distorted. The cysts completely occupied the medullary region and cortex was pushed towards periphery and formed only a narrow rim surrounding the medulla.Other associated anomalies in affected foetuses wereOligohydramnios, Hydrocephalus, Facial malformations (low set ears, Depressed nasal bridge and Micrognathia), Pulmonary hypoplasia and anal atresia (Fig-1).

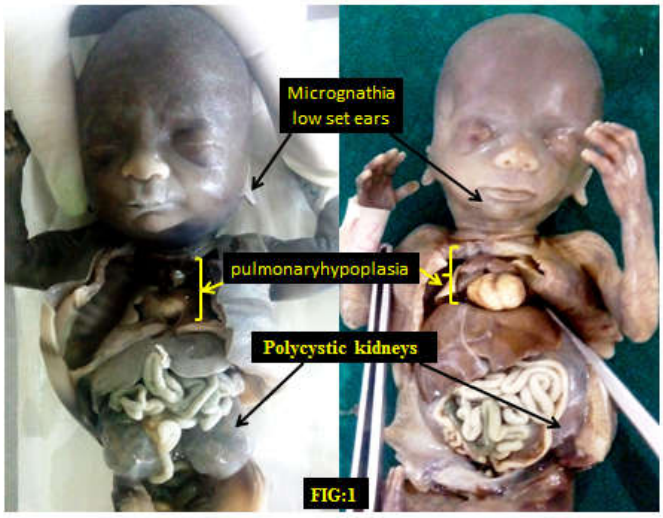

Fig. 1. Fetuses showing bilateral polycystic kidneys with Other associated anomalies

Histogenesis: On histology the observation in various age groups of fetuses are as follows:

Group A (11-18weeks): Capsule was identified, it was made up of thin collagen fibres and fibroblasts. Beneath the capsule a zone was present known as nephrogenic zone the cells in this zone were oval, with eosinophillic cytoplasm and centrally placed nucleus. Some cells were scattered and some were in the groups (Fig-2). Glomeruli were observed, theywere in there developing stage, and between the developing glomeruli in the connective tissue developing tubules were seen. Proximal and distal convoluted tubules were not differentiated (Fig -3).

Group B (>18-25weeks): Capsule was present. Nephrogenic zone was reduced compared to previous gestational age groups. Glomeruli were bilobed were more in number (Fig-4).

Group C(>25weeks): In the cortex DCT and PCT were increased, connective tissue was less; many capillaries were seen adjacent to the glomeruli.In medulla more number of collecting tubules, thin and thick loop of henle were seen.

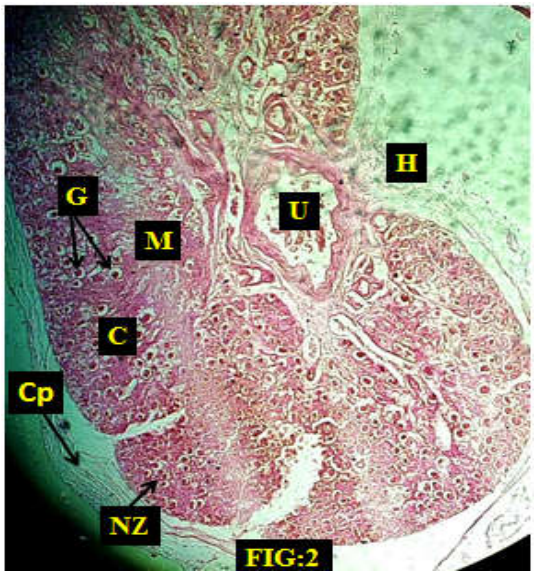

Fig.2. Nephrozenic zone beneath the capsule with undifferentiated cortexand medulla

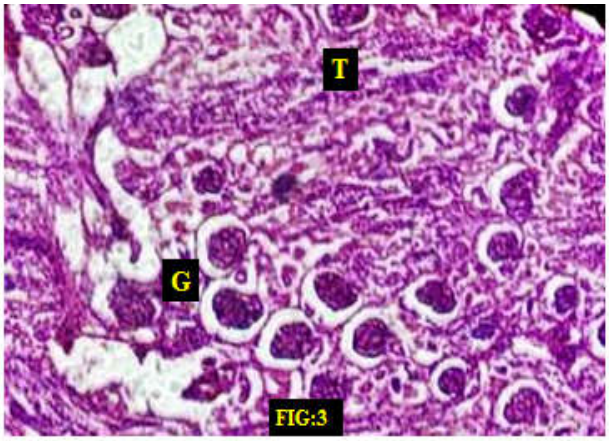

Fig. 3. Cortex with mature and immature glomeruli (G) and tubule formation at 20 weeks of gestation. H&E 60X

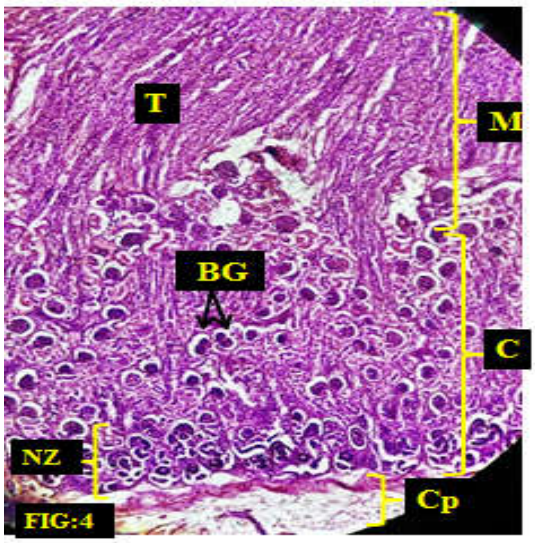

Fig. 4. Section studied from the kidneys using hematoxylin and eosin stain (H and E) shows sub-capsular nephrogenic zone with glomeruli (arrow)

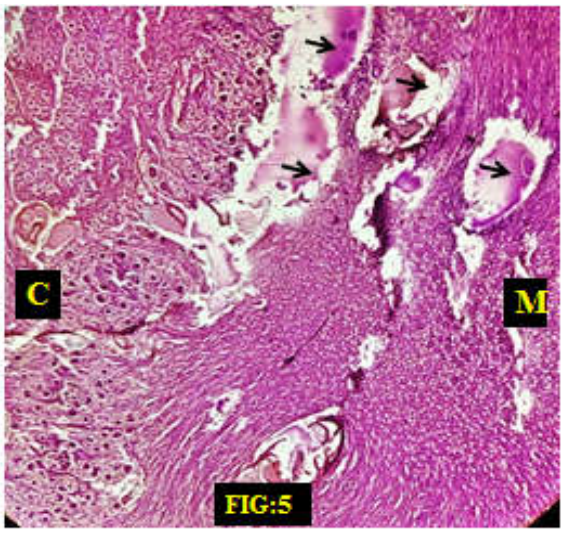

Fig. 5. Lower cortex and medulla shows numerous cysts of varying sizes lined by cuboidal epithelium (arrowheads)

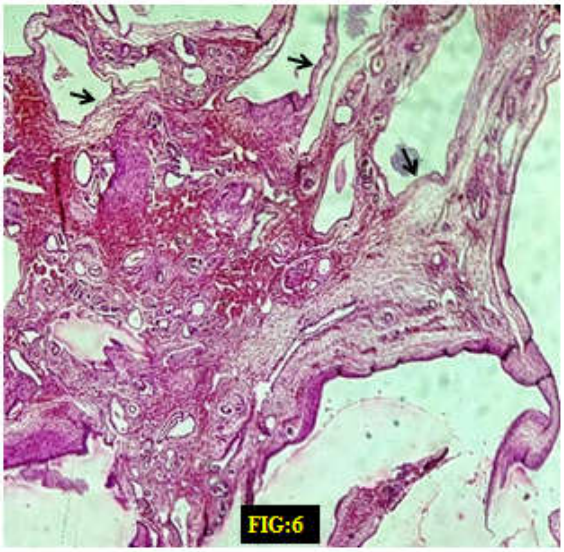

Fig. 6. The microscopic appearance of a multicystic dysplastic kidney (cystic renal dysplasia, or Potter type II) is characterized by large cysts lined by flattened cuboidal epithelium and an intervening parenchyma that is fibrotic with islands of bluish cartilage and rare glomeruli

Thickness of cortex and medulla increased with increase in gestational age. The microscopic appearance of a multicystic dysplastic kidney (cystic renal dysplasia, or Potter type II) is characterized by large numerouscysts lined by a single layer of flattened to low cuboidal epithelium and an intervening parenchyma that is fibrotic with islands of bluish cartilage and rare glomeruli (Fig-5). No difference was appreciated in the histology of proximal and distal convoluted tubules Occasional glomeruli were seen and appeared normal (Fig-6).

DISCUSSION

The Potter classifiation of renal cystic disease separates cystic kidneys intothe followingfourtypes:

Type 1-Infantile polycystic kidneydisease
Type 2-Cysticdysplastic kidney disease
Type 3-Adult polycystic kidney disease
Type 4-Partial or intermittent urinary outflw obstruction (obstructive dysplasia) (Rizk and qwChapman, 2003; Gunay-Aygun, 2009; Osanathanondh and Potter, 1964).

The Potter classifiation of renal cystic diseases has been replaced by a classification based on the genetic or nongenetic origin of the renal cystic diseases. Genetic diseases include classic diseases such as autosomal recessive (ARPKD) and dominant (ADPKD) polycystic kidney diseases and more recently recognized diseases, such as glomerulocystic kidney disease (GCKD), medullary cystic dysplasia associated with syndromes, and nephronophthisis– medullary cystic dysplasia complex (Rizk and qwChapman, 2003; Gunay-Aygun, 2009; Osanathanondh and Potter, 1964). Multicystic disease of the kidney is a common cause of enlarged abdomen in fetus second only to hydronephrosis. It is a congenital maldevelopment, where the renal cortex is replaced by numerous cysts of varying sizes. Dysplastic parenchyma anchors the cysts and the arrangement resembles branches of grapes. Calyceal drainage system is absent. It is typically a unilateral disorder, when bilaterally occur, it is incompatible with extra uterine life (Irsutti *et al*., 2000). In bilateral disease, neonates may also display oligohydramnios and pulmonary hypoplasia.

Conclusion

Although the antenatal diagnosis of fetal anomalies has improved largely due to the availability of high resolution ultrasound equipments, still cystic kidneys discovered incidentally during routine antenatal ultrasonography frequently pose significant diagnostic dilemma. Ultrasound can detect the cysts as early as 12- 13 weeks of gestation Correct and timely antenatal diagnosis of polycystic kidney disease with associated fetal anomalies is important so that proper counselling and appropriate obstetric and paediatric management can be extended (Sunita B. Patil *et al*., 2013). Routine antenatal scans are done to detect the anomalies in fetus, some of which may be incompatible with life. Performing autopsies on such fetuses can result in confirmation of ultrasonographic findings and finding out other associated anomalies or related syndromes (Lavanya *et al*., 2014).

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