A HISTOPATHOLOGICAL OBSERVATION MADE ON CYSTIC KIDNEYS OBTAINED FROM HUMAN CADAVERS

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ABSTRACT

Introduction: Cysts in the kidneys have been described as being heterogeneous, and occur due to hereditary, developmental and acquired disorders. They account for 6-8% of diseases that go for dialysis. Evaluating cystic diseases is important for pathological diagnosis and treatment procedures for the patient.

Objective: Present study aimed at finding the types of cysts prevalent in human cadaveric kidneys by making a histopathological observation. The study was made to review the incidence of cysts in kidneys and to find out what are the commonest types of cysts that are identified.

Material and Methods: Thirty formalin fixed adult cadavers were used for the study. Twenty were male and ten were female cadavers used for routine dissection and teaching purposes for the first year medical students in the department of Anatomy, P.E.S Institute of Medical Sciences and Research. The kidneys were dissected out from the cadavers from both sides. The fat and fascia were removed carefully and first photographed for gross appearance, before handing the specimens to the department of pathology for further study by making sections.

Results: Cystic kidneys were seen in seven cadavers out of the thirty cadavers. The other cadavers had no cystic kidneys. The incidence of cysts has been calculated in this study as 23.3%. The normal kidneys were 76.6%. In three female cadavers unilateral cysts were seen in the right kidney. In male cadavers, one had unilateral cyst and the remaining three presented with bilateral cystic kidneys. One among the four showed multiple bilateral cysts ranging from one to forty five cysts.

Conclusion: From this study it is evident that cysts of the kidney may occur without disturbing the normal functioning of the kidneys. The incidence of multiple cysts was more in males than in females. No of cysts were more in the right than in the left kidneys. For a person to lead a normal life one fifth of the kidney is sufficient if it functions normally.

KEY WORDS: Cystic kidneys, multiple cysts, polycystic kidneys, bilateral cysts and unilateral cysts.

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INTRODUCTION

The kidney parenchyma is organized into a distinct outer cortex and an inner medulla. The cortex is a reddish-brown band, 1-2cm thick, andis covered by a capsule. It consists of renal

corpuscles, convoluted portions of the proximal and distal tubules, short lengths of the straight portions of the tubules. All these constitute the cortical labyrinth. The cortex is highly vascularised and most of the blood passes through the cortex in the kidney. The medulla consists of the straight portions of the proximal and distal tubules, loops of Henle, collecting ducts, vasa recta. The medulla is organized into 8-18 pyramids which are radially striated, conical structures with base adjacent to the cortex. The apex projects into renal sinus [1].

Cysts have been described in both the cortex and medulla.

Based on pattern of inheritance the types of cysts seen in the kidneys are classified as adult polycystic kidney disease and adult-onset medullary cystic disease both of which are autosomal dominant. Autosomal recessive inheritance has been observed in childhood polycystic kidney disease and familial juvenile nephronophthisis. No pattern of inheritance has been described in medullary sponge kidney, simple cysts and acquired renal cystic disease [2].

In autosomal adult polycystic kidney disease the kidneys have multiple expanding cysts that destroy the renal parenchyma. Functioning nephrons are seen between the cysts under microscopic examination. The cysts are filled with a clear serous fluid, sometimes with haemorrhagic fluid. The cysts arise from the tubules throughout the nephron. The cysts have a variable epithelial lining [3].

The cystic diseases of renal medulla are medullary sponge kidney, nephronophthisis and adult-onset medullary cystic disease [3].

Medullary sponge kidney has multiple cystic dilatations of the collecting ducts in the medulla. The cysts are lined by cuboidal epithelium or occasionally by transitional epithelium. The latter two diseases mentioned above have cysts in the medulla usually at the cortico-medullary junction. The cysts are lined by flattened or cuboidal epithelium and are surrounded by inflammatory cells or fibrous tissue. The cortex shows thickening of basement membranes of proximal and distal tubules.

Simple cysts occur as wide cystic spaces ranging from 1to 10cm in diameter [4]. They have a gray, glistening smooth membrane and it is lined by a single layer of cuboidal or flattened cuboidal epithelium which may become atrophic. Usually found during post-mortem they seem to

have no clinical significance.

Acquired dialysis associated cystic disease is seen in patients who are under prolonged dialysis for end stage renal disease. The cysts are up to 2 cm in diameter and are lined by hyperplastic or flattened tubular epithelium. They are often contain calcium oxalate crystals which cause the cysts by obstructing the tubules. They may also form due to interstitial fibrosis causing obstruction of tubules [5].

Adult polycystic kidney disease (ADPKD) is an autosomal dominant disease with high penetrance and occurs in 1 out of 400 to 1000 persons and accounts for 5% to 10% of chronic renal failure [6]. It is caused by mutations in one of two genes, PKD1 and PKD 2. Polycystin 1 is localized in the tubular epithelial cells of the distal nephron. Polycystin 2 is present in all segments of the renal tubules. They form a cilia centrosome complex. Each of the epithelial cells of the kidney contains a single non motile primary cilium. It is a hair like organelle that projects into the lumen of the tube from the apical surface of the cell. The cilium contains microtubules which are attached to the centriole. The primary cilium functions as a mechanosensor to monitor changes in fluid flow and shear stress. The polycystin 1& 2 form a protein complex that acts to regulate intracellular calcium influx. The ciliary bending during fluid flow opens calcium channels. Disruption of polycystin activity Mutated proteins affect second messengers and influence proliferation, apoptosis, ECM interactions, and secretory function, leading to formation of tubular cysts. Cysts arise anywhere along the nephron, they may be 3-4 cm in diameter, and may compress the adjacent parenchyma. In late disease interstitial inflammation and fibrosis.

MATERIALS AND METHODS

Thirty formalin fixed adult cadavers were used for the study. Twenty were male and ten were female cadavers used for routine dissection and teaching purposes for the first year medical students in the department of Anatomy, P.E.S Institute of Medical Sciences and Research. The kidneys were dissected out from the cadavers from both sides. The fat and fascia were removed carefully and first photographed for

gross appearance, before handing the specimens to the department of pathology for further study by making sections.

OBSERVATIONS

Fig. 1: Showing the cystic wall and lining epithelium (Low Magnification 10X H & E).

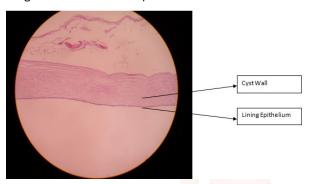
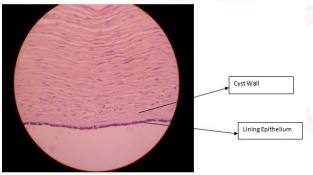


Fig. 2: Showing the cystic wall and lining epithelium (High Magnification 40X H & E).



A pathological report from department of pathology, P.E.S Institute of Medical Sciences and Research showed the findings as follows: Anatomically most cysts were located in the renal cortex. Pathological evaluation revealed non-communicating varying sized cysts within the parenchyma. The cysts are seen supported by thin fibro-collagenous wall with no definitive epithelial lining. Haemorrhagic material was noted in the lumen of few cysts. No features of dysplasia or malignancy were seen in the sections studied. Section from cyst from 6th case also revealed dense chronic inflammation within the adjacent parenchyma. Histological impression of simple cysts was rendered.

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inflammation within the adjacent parenchyma. Histological impression of simple cysts was rendered.

DISCUSSION

In a study by Jaswinder Kaur [4] out of 30 cadavers cysts were reported to be seen in 4 cadavers more in incidence in males, compared to females. Even in the present study male cadavers had cysts more than the female cadavers.

Kaur Manpreet et al [5] observed and reported that multiple cysts were present only on the left side. They concluded that unilateral polycystic kidney is seen not so frequently. They cited Levin et al that unilateral renal cystic disease had at least three aspects from ADPKD, unilateral localization, negative family history and no progression to chronic renal failure. They have no cysts in other abdominal organs. In the present study unilateral cystic kidney was seen in one male cadaver. Sathialakshmi V et al [6] studied cystic diseases in various organs. They reported bilateral polycystic kidney in a male cadaver, similar to the present study.

Dr.Shroff Gautam A et al [7] studied ultrasonography of patients between 25-29 weeks gestation for kidney development. They found unilateral multi cystic dysplastic left kidney, right hypoplastic kidney with left lobulated kidney, right kidney agenesis & left kidney enlarged with cystic mass. They opined that failure of the ureteric bud to integrate and branch appropriately into the metanephros during development of kidney resulted in multicystic disease of kidney. The present study was done in adult cadavers with probably cysts present since birth. No abnormal findings suggestive of congenital anomalies were seen and no observable changes were seen in the individual organ systems.

Vinnakota Sunitha [8] studied still born foetuses at various stages of development from 10 weeks to 40 weeks. They found that lobulation is seen at 12 weeks and is not present at 16 weeks. They also observed juxta glomerular apparatus at 14 weeks of gestation. Difference in staining pattern of tubules at 16 weeks of gestation. They concluded that major development of kidney takes place at 20-22 weeks. Vinnakota Sunitha

also reported a case of bilateral polycystic kidneys with hepatic fibrosis and hypoplastic lungs in a 32 weeks still born male foetus.

The present study did not compare foetal kidneys. Eswari A.K, et al [9] discussed that tubular and glomerular microcysts are seen as early as 12 weeks of gestation and concluded that time of formation of cysts is not known. According to the present study one male cadaver which was aged 85 years had large cysts on both sides. The personal history of the individual is known as it was a donated cadaver. The history revealed treatment of hypertension, no history of renal disease till the end. This suggests formation of cysts did not affect normal excretory function of the individual. The cysts were probably simple cysts and this was confirmed by histological studies.

Adrian S.Woolf et al [10] have described three types of diseases associated with glomerular cysts. One of them is urinary obstruction. The author cited studies which described that, normal glomeruli can become cystic after birth. The Bowman's space becomes enlarged after collapse of glomerular tufts secondary to glomerulosclerosis or mesangiolysis or haemolytic uremic syndrome. When cysts form in the kidney they are filled with fluid. Cysts can enlarge in the kidneys while replacing much of the normal structure, resulting in reduced kidney function leading to kidney failure.

CONCLUSION

The study observed cysts in cadaveric kidneys of various age groups and in both sexes. The cysts were present in male cadavers more than in female cadavers. The size and number of cysts was also variable and though the cause of death of the individual cadavers was not known in this

study, the chance finding of cysts in adult kidneys indicate that an autosomal dominant disease manifests itself late in life and does not interfere with the functioning of the individuals organs. Most probably the cysts were formed due to obstruction in the glomeruli and did not go as far as renal failure for manifestation clinically.

Conflicts of Interests: None

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