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ACQUIRED CYSTIC DISEASE
OF THE KIDNEY IN
UREMIC AND HEMODIALYSIS PATIENTS

THESIS
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C O N T E N T S

-	INTRODUCTION AND AIM OF THE WORK	ii
-	REVIEW OF LITERATURE	
	A) Classification of renal cysts	I
	B) Acquired cystic disease of the kidney in uremic and hemodialysis patients :	
	- Incidence and aetiology	8
	- Composition of cyst fluid	I8
	- Pathology	22
	- Clinical aspects	40
	C) Ulatrasonography	49
-	MATERIALS AND METHODS	60
-	RESULTS	63
-	DISCUSSION	9I
-	SUMMARY	IO7
-	CONCLUSION	III
-	REFERENC ES	II3

INTRODUCTION

INTRODUCTION AND AIM OF THE WORK

With the advent of long-term intermittent hemodialysis , patients with chronic renal failure are leading prolonged lives. Hemodialysis programmes , as a new therapy, created new diseases or complications.

Acquired cystic disease of the kidney is such a complication of uremia and hemodialysis, which had not received ,comparatively, the suitable attention, though important clinical problems may result.

Ultrasonic has been used with increasing frequency to evaluate initial disease in uremic patients and to re-evaluate disease in patients under regular maintenance hemodialysis.

Ultrasonography is of great help in recognizing the acquired uremic renal cystic disease, moreover , the development of a special modified ultrasonic transducer containing a central lumen, permits ultrasound to be used as a guide in renal cyst aspiration.

The aim of this work is to study incidence and epidemiology of acquired cystic disease of the kidney in uremic and hemodialysis patients with the aid of ultrasonography.

REVIEW OF LITERATURE

CLASSIFICATION OF RENAL CYSTS

Renal cysts are common radiographic and morphologic findings , they are a heterogenous group comprising heretable ,developmental and acquired disorders (Kenneth , 1975).

Finding the cysts is relatively easy , but identifying them and interpreting the findings are relatively difficult. The radiologist seeing parenchymal defects , the urologist seeing cysts on the capsular surface and even the pathologist seeing the entire sectioned organ , see from restricted points of view. Each may use a different term although he recognizes the same pattern , each may use the same term and intend something different (Kenneth, 1975).

The problem with renal cysts is that they are despite their great heterogeneity , mostly similar. Cysts must ,therefore , be grouped and subdivided. It is important to designate cysts that have differing patterns of inheritance , differing natural histories, differing prognostic implications and differing therapeutic implications (Kenneth , 1975).

Cysts may arise in any part of the nephron and collecting system, but they have a predilection for the loop of Henle and for the collecting tubules and peripheral portions of collecting ducts (Fetterman, 1974) and (Potter, 1972).

Some patterns are characteristic of ceratin conditions. The cysts in infants with polycystic disease of autosomal recessive type are characteristically located in collecting tubules and ducts (Potter, 1972) and (Heggo, 1965).

Glomerular and tubular microcysts in peripheral cortex are commonly associated with syndromes of multiple malformations (Brenstein, 1971).

Dysplastic cysts in peripheral cortex are commonly associated with lower urinary tract obstruction (Heggo, 1965) and (Bernstein, 1968).

Kenneth, 1975, concluded that, the tendency of microcysts to localize in the peripheral cortex seems, like the similar localization of dysplastic cysts to hold some clue to pathogenesis. The responsible agents metabolic or otherwise, seem to have its greatest effect on the part of the cortex undergoing differentiations

late in gestation, the inner two thirds of the cortex are often unaffected.

Polycystic disease, a lesion that affects only certain nephrons because of their immaturity without affecting other nephrons that have gone through the same stages of development, may be caused by external factors acting through a limited period of time (Kenneth, 1975) .

A generalized susceptibility would be more compatible with an abnormality residing in the nephron itself. If some cystic abnormalities are indeed secondary to extrinsic factors that affect immature tubular structures, then those cysts could be regarded as developmental. Other cysts arising in later life, even in association with heritable disorders, would not in a strict sense be developmental. Cysts arising from a genetic abnormality residing within the tubular cells themselves would properly be regarded as hereditary , (Kenneth , 1975).

Cysts arising in completely formed nephrons and collecting tubules seem to well established by numerous experimental studies. A partial list of nephrotoxic agents capable of acting on mature kidneys includes the anti-helminthic diphenylamine (Thomas , 1957) and

(Safouh , 1970) , the citrus fungistat biphenyl(Booth, 1961), the defoliant 2,4,5-T (Courtney, 1970), the antioxidants dihydromethyl quinolone(Wilson , 1959), nordihydroguaiaretic acid (Goodman, 1970), the experimental antihypertensive agent aminodiphenylthiazole (Carone, 1972), the anti-inflammatory agent tetrahydrocarbazole (McGeoch, 1972), and alloxan (Vargus, 1970). Many are phenyl derivatives, a point that may or may not be pertinent. Most including diphenylamine, aminodiphenylthiazole and nordihydroguaiaretic acid which result in early cellular necrosis, tetrahydrocarbazol has been reported to cause cellular hyperplasia(McGeoch,1972).The administration of such agents to animals after the cessation of nephrogenesis would seem to preclude the possibility of a heritable disorders, although it might identify a genetic predisposition in susceptible animals (Kenneth, 1975).

The ability of corticosteroid to produce cysts in newborn animals, may on the other hand , be related to nephrotic immaturity(Filmer, 1973), (Ojeda,1972) and (Vlachos, 1972). It is reasonably certain that some human cystic conditions are acquired as renal parenchymal cavitation follow inflammatory and localized necrotizing disorders (Kenneth, 1975).

The classification presented here has been developed by Kenneth, 1975, to incorporate radiographic, functional and genetic contributions in its clinical - pathological correlations.

The major categories of classification are shown in the following table:-

- I- Renal dysplasia
 - A- Multicystic dysplasia.
 - I- Unilateral multicystic kidney.
 - 2- Bilateral multicystic dysplasia.
 - B- Focal and segmental cystic dysplasia.
 - C- Cystic dysplasia associated with lower urinary tract obstruction.
 - D- Familial cystic dysplasia.
- 2- Polycystic disease
 - A- Infantile polycystic disease
 - I- Polycystic disease of early infancy.
 - 2- Polycystic disease of childhood .
 - 3- Congenital hepatic fibrosis.
 - B- Adult polycystic disease.
- 3- Renal cysts in hereditary syndromes
 - A- Meckel's syndrome.
 - B- Zellweger's cerebro hepato renal syndrome.
 - C- Jeune's asphyxiating thoracic dystrophy.

- D- Tuberous sclerosis complex and Lindau's disease.
- E- Cortical cysts in syndromes of multiple malformations.
- 4- Renal cortical cysts
 - A- Diffuse glomerular cystic disease.
 - B- Peripheral cortical microcysts.
 - C- Juxta-medullary cortical microcysts.
 - D- Simple cysts, solitary and multiple.
- 5- Renal medullary cystic disorders
 - A- Medullary sponge kidney.
 - B- Medullary cystic disease complex.
 - I- Familial juvenile nephronophthiasis
 - 2- Medullary cystic disease.
 - 3- Renal-retinal dysplasia.
- 6- Acquired miscellaneous parenchymal renal cysts
 - A- Inflammatory and necrosis
 - I- Medullary necrosis.
 - 2- Lithiasis.
 - 3- T.B.
 - 4- Echinococcosis.
 - B- Neoplasia
 - I- Cystic degeneration of carcinoma.
 - 2- Multilocular cystadenoma (Benign cystic nephroma).
 - 3- Dermoid cyst
 - C- Endometriosis
 - D- Traumatic intrarenal hematoma.

- 7- Extra parenchymal renal cysts
 - A- Pyelogenic cyst(Pelvic diverticulum)
 - B- Parapelvic cyst(Lymphangiectasis)
 - C- Perinephric cyst.