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Ultrasonographic and Clinicopathological Studies on Feline polycystic kidney disease.

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Abstract

Feline polycystic kidney disease (PKD) is one of the most important causes of kidney failure in Persians and Persian related cats and the most prevalent inherited feline disease. The aim of the present work is to investigate the cases of feline polycystic kidney disease through ultrasonography, clinicopathological as well as cytological examinations in Persians and Persian related cats. The present study was carried out on fourteen Persians and Persian related cats which were admitted to Veterinary Clinics in Giza governorate (9 diseased and 5 apparently healthy cats) between November 2014 and December 2015. All cats were ranged from 6-15 years. All cats were subjected to ultrasonographic, clinicopathological examinations, while cytological and histopathological examinations were done for diseased animals after necropsy. Ultrasonography showed bilateral renomegaly with multiple anechoic round to irregularly cysts inside the renal cortex and medulla. Anemia was the most common abnormality revealed by CBC and was characterized as nonregenerative normocytic, normochromic anemia. Serum biochemistry revealed severe azotemia with hyperphosphatemia, hypoproteinemia and hypocalcemia as a consequence of progressive loss of renal function. Renal impression smears of diseased cats showed tubular casts with infiltration of the renal smears with reactive lymphocytes. In conclusion, the clinical and clinicopathological abnormalities identified in diseased cats were attributed to the severe renal impairment. Since PKD is hereditary disease, the best way to prevent it is to identify PKD-affected cats using ultrasound and remove them from the breeding pool by spaying and neutering.

Introduction

Feline polycystic kidney disease (PKD), also known as autosomal- dominant polycystic kidney disease (ADPKD), is one of the most important causes of kidney failure in Persians and Persian related cats and the most prevalent inherited feline disease **(Scalon et al., 2014)**. Polycystic kidney disease characterized by multiple cysts of various sizes that occur in the renal cortex and medulla and occasionally in the liver and pancreas **(Beck and Lavelle, 2001)**.

The most important manifestation of this disease is development of renal failure. Initially, cysts developed from renal tubular cells and separated from the nephron as they form. As cysts enlarge, they compress surrounding normal renal parenchyma and lesions of chronic interstitial nephritis develop. As the disease advances, cysts increase in number and size, and renal volume increase **(Reichle etal., 2002)**. Frequent complications of polycystic kidney disease include dangerously high blood pressure, pain in the back or sides, hematuria, recurrent urinary tract infections, kidney stones, and heart valve abnormalities **(Harris and Torre, 2015)**.

The two major forms of polycystic kidney disease are distinguished by the age of onset. The autosomal dominant form (APKD) has signs typically begin in adulthood, although cysts in the kidney are often present from birth or childhood. It is a bilateral renal condition, but cysts also occur in the liver (60%), pancreas (10%) and various other organs. The second form is autosomal recessive form of polycystic kidney disease (ARPKD) is much rarer and lethal early in life in association with massively enlarged kidneys **(Garantham, 2002)**.

Mutations in the PKD1and PKD2 genes cause polycystic kidney disease and lead to the formation of thousands of cysts, which disrupt the normal functions of the kidneys and other organs **(Lee et al., 2010)**.The aim of the present work is to study the cases of feline polycystic kidney disease through ultrasonography and clinicopathological as well as cytological examinations of Persians and Persian related cats.

Material And Methods

Animals and Experimental Design:

The present study was carried out on fourteen Persians and Persian related cats which were admitted to Veterinary Clinics in Giza governorate (9 diseased cats and 5 apparently healthy cats) November 2014 and December 2015. All cats were ranged from 6-15 years. The cats were divided into two groups group (1) composed of 5 apparently healthy cats. Group (2) composed of nine cats suffered from Polyuria, polydipsia, dehydration, lethargy, inappetence/anorexia, vomiting, constipation, weight loss, muscle wasting, and oral ulceration. Clinical signs were recorded , clinical examination and collection of blood samples from all animals were performed. All animals were subjected to ultrasonographic, clinicopathological examinations, while cytological and histopathological examinations were done for diseased animals after necropsy.

Clinical Examination: All cats were thoroughly investigated and clinically examined by abdominal palpation and tactile percussion according to the method described by **Kelly (1984)**.

UltrasonographicExamination: Ultrasonography was performed after 24 hrs fasting. The examined cats were positioned in dorsal recumbency. Cranial ventral abdomen were clipped and sheaved then covered with coupling gel. Transverse and longitudinal scans were carried up using Pie-Medical Scanner (Maastricht, Netherlands) and sector transducer with alternating frequency of 5.0-7.5 MHz according to the method described by **Nyland et al., (1989)**.

Blood and tissue samples: Blood samples collected from anterior median vein in all cats. The blood samples were divided into two parts. The first part was anticoagulated with EDTA and used for hematological studies. The second one collected in plain centrifuge tube and was allowed to clot, then centrifuge at 3000 rpm for 10 minutes and the serum was separated for biochemical studies.

All nine cats suffered from PKD were euthanased by barbiturate overdose within one week of renal ultrasonographic diagnosis of the disease. After necropsy, impression smears from polycystic kidneys of the diseased cats were taken after cutting kidney into two halves to obtain a freshly cutted surface. The cutted surface was blotted on clean filter paper. The surface was made small enough to make several rows of imprints, another smears were taken from renal cysts fluids. The other half of the kidney was embedded in paraffin for histopathological examinations.

Clinicopathological studies: Estimation of erythrocytic (RBCs) count, hemoglobin (HB) concentration, packed cell volume (PCV), total and differential leukocyte counts were performed according to **Fieldman et al. (2000)**. Red cell indices including mean corpuscular volume (MCV) and mean corpuscular hemoglobin concentration (MCHC) were calculated according to the same author.

Serum biochemical analysis including measurement of serum total proteins, albumin, creatinine concentrations and BUN were carried according **to Dumas and Biggs (1972), Tabacco et al. (1979) and Fabiny and Ertingshausen (1971)**, respectively. Serum calcium and inorganic phosphorus were measured according to **Bauer (1981) and Dryer and Routh (1963)**. The mentioned serum biochemical parameters were assayed using reagent kits supplied by StanBio -Laboratories incorporation, USA.

Cytological Examination: Both renal impression smears and renal cyst fluid smears were stained by field stain **(Tankeyul et al., 1987)**, washed and examined under the microscope **(Rick et al., 1999)**.

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Histopathological Examination: Tissue specimens were collected and fixed in 10% neutral buffered formalin for preparing paraffin tissue sections at 4-6μ thickness. These sections were stained with hematoxylin and eosin **(Bancfort and Stevens, 1996).**

Statistical Analysis: The results obtained were expressed as mean ±SD. Student's t-test was used to compare the mean data between groups. Statements of statistical significance were based on $P<0.05$ **Sendecor and Cochran (1982).**

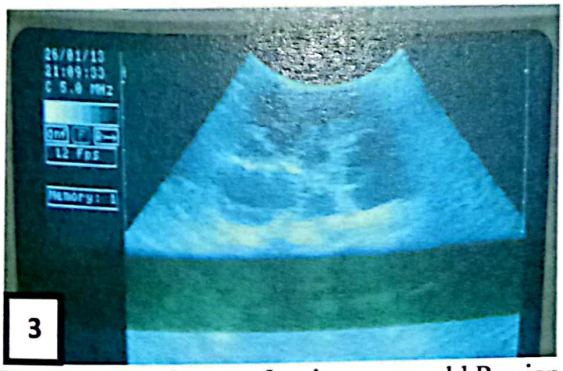
Results

Clinical Examination: All nine cats suffered from lethargy, depression. **Fig.(1)** Anorexia, vomiting with weight loss.. Polyuria and polydipsia. Enlarged, lobulated (only in 3 cats) with tender kidneys by abdominal palpation. Uremic syndrome (Dehydration: Decreased skin turgor, tachypnea, sunken eyes, Pallor mucous membranes, Halitosis, oral ulcerations **Fig. (2)** and Muscle atrophy along the spine and limbs).

Fig .(1): Nine years old cat suffered from lethargy, depression.
Fig .(2): Oral ulcerations (arrow) in six years old cat.

Ultrasonographic Examination:

Kidneys: Bilateral renomegaly with multiple anechoic round to irregularly cysts (1 mm to >1 cm) inside the renal cortex and medulla **Fig. (3& 4).**



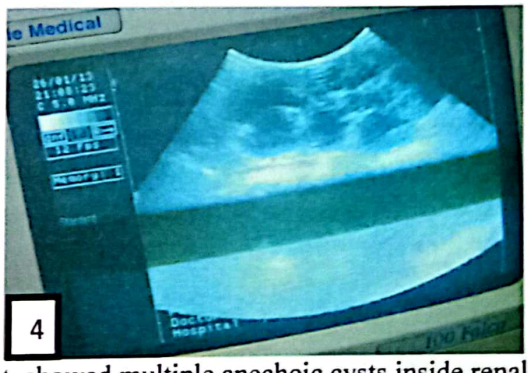


Fig.(3): Renal scan of a nine years old Persian cat, showed multiple anechoic cysts inside renal cortex and medulla with distant acoustic enhancement, positive for PKD.
Fig.(4): Renal scan of six years old Persian cat, showed multiple anechoic cavitations inside renal cortex and medulla with obvious distant acoustic enhancement, positive for PKD.

Clinicopathological studies: Results of hemogram and red cell indices (MCV and MCHC) are illustrated in table (1). In comparison with apparently normal cats, PCV, Hb concentration and RBCs count showed significant decrease in diseased cats with insignificant changes in MCV and MCHC values. Microscopical examination of the stained blood films from diseased cats showed severe hypochromacia with many target cells. The leukogram showed neutrophilic leucocytosis with monocytosis .

Statistical analysis of different serum biochemical parameters are illustrated in tables (2). Measurement of serum total proteins, albumin, globulin and serum calcium concentrations showed significant decrease in comparison to the control group. In the other hand, creatinine, BUN and serum phosphorus concentrations showed significant increase in all diseased cats in comparison to control group.

Cytological Examination:

Renal cytology: Renal impression smears of diseased cats showed few renal tubular epithelial cells which are rather large to polygonal cells that occur singly. Tubular casts were seen in all renal smears **Fig. (5 & 6)**. In addition to infiltration of the renal smears with lymphocytes **Fig. (7)**. Renal cysts contain fluids that clear or slightly yellow and often contain fibrin flocculent material or hemorrhagic fluid. Renal cyst fluid smears revealed low cellularity and macrophages with foamy cytoplasm and few neutrophils **Fig. (8)**.

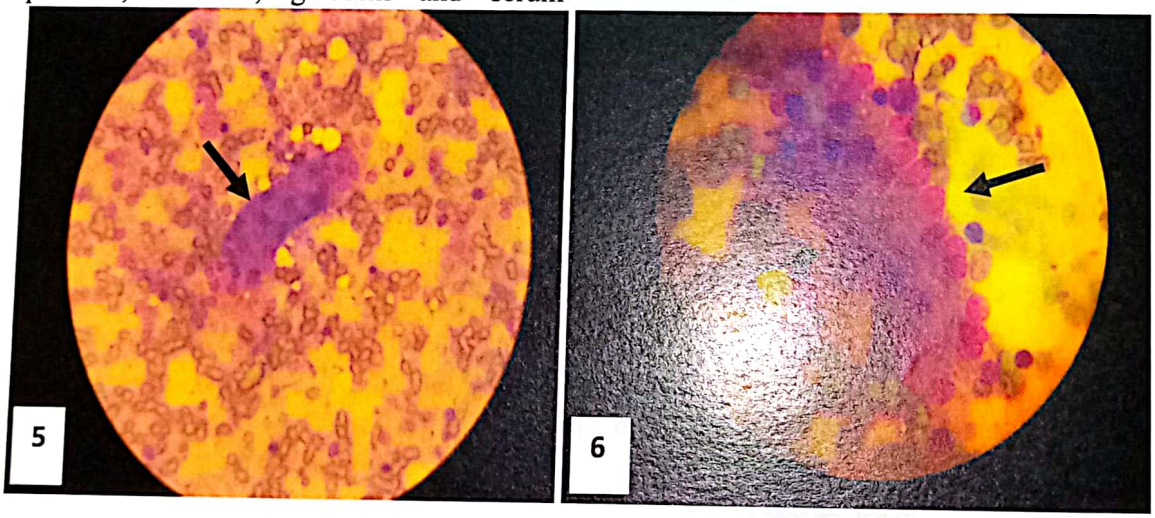


Fig. (5): Renal impression smear showed tubular cast (arrow) of a nine years old Persian cat suffered from polycystic kidney disease (x400 field stain).

Fig.(6): Tubular cast with lymphocytic infiltration (arrows) with oil immersion lens (x1000 field stain).

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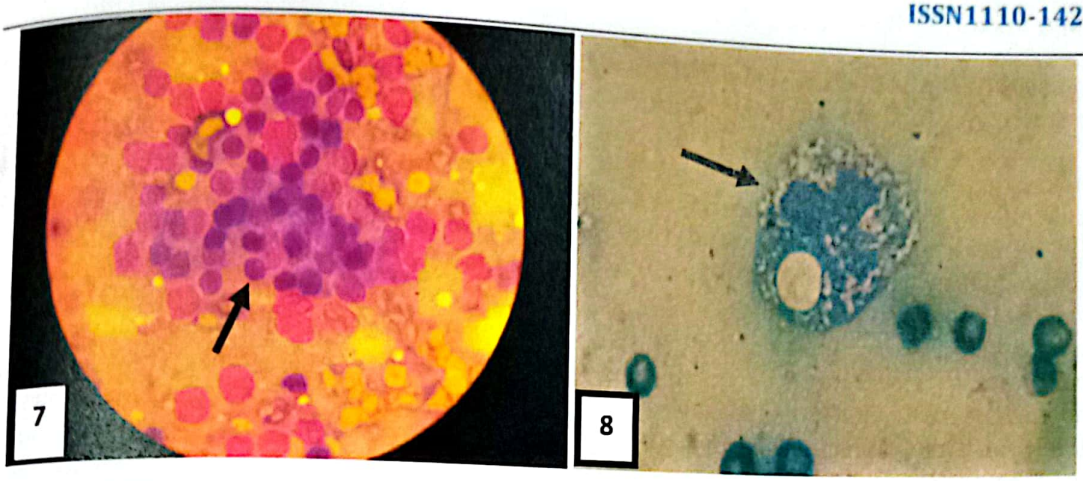

Fig. (7): Infiltration of the renal smears with lymphocytes (arrow) (x1000 field stain).
Fig.(8): Renal cystic fluid showed large activated macrophages (arrow) that have abundant cytoplasm containing dark pink material with scattered erythrocytes(x1000 field stain).

Histopathological Examination:

Gross lesion: the affected cats were identified at necropsy by presence of many renal cysts in the renal cortex and medulla of both kidneys and their number, size, and appearance varied markedly among cats **Fig.(9)**. Microscopically, renal cysts were lined by epithelium which varied from cuboidal to squamous. Some cysts were surrounded by interstitial fibrosis or inflammation. In addition to renal cysts, multifocal chronic tubulointerstitial nephritis was present in affected cats with lymphocytic interstitial infiltration, interstitial fibrosis and tubular epithelial atrophy **Fig.(10)**.

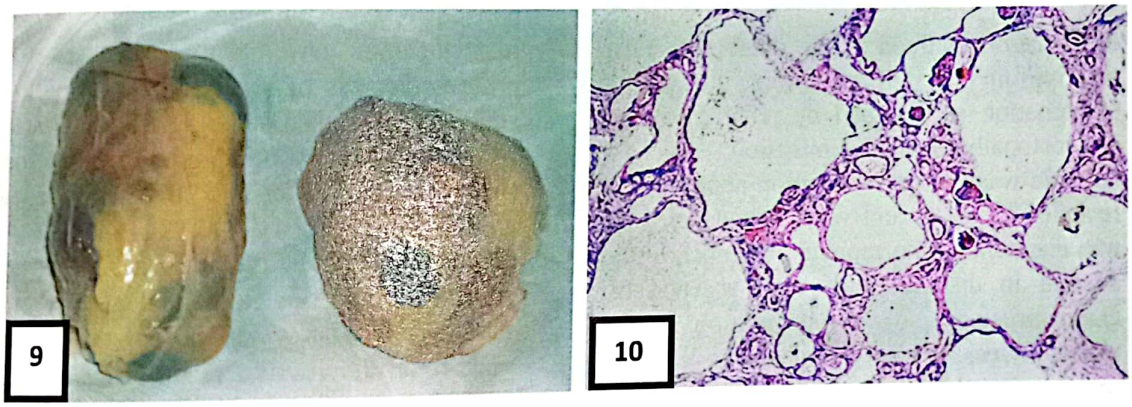

Fig. (9): polycystic kidneys of a nine years old Persian cat
Fig.(10): Histolopathological section showing a renal cysts with tubular epithelial atrophy (x100H&E)

Discussion

Feline PKD is an inherited disease in Persian and Persian-related cats. Approximately 37% of Persian cats worldwide **(Bartola, 2000)** are PKD affected. In the United States 6% of the cat population has PKD, making it the most prominent inherited feline disease **(Lyons et at., 2004)**. ADPKD in humans and cats results in slowly progressive renal disease. In the most common form of human ADPKD, end-stage renal failure occurs typically by 41 to 65 years of age. Cats with ADPKD may develop end-stage renal failure from 3 years-of-age, although renal failure does not usually become evident until much later in life **(Barrs et al., 2001).**

In our study, polycystic kidney disease was recorded by physical examination as

renomegaly and lobulation (only in 3 cats) through abdominal palpation. Ultrasonography is the imaging method of choice for the diagnosis of PKD according to **Walter et al. (1988), Nyland et al. (1995)** and **O'Leary et al. (1999)**. Ultrasonography was used to record the presence of renal cysts which identified as circular, anechoic cysts with strong distal acoustic enhancement inside the renal cortex and medulla of affected kidneys. **Biller et al (1996)** states the earliest age at which cysts could be identified was 7 weeks. In the present investigation, the youngest cat presented for examination was 6 years and was found to have 1mm -1 cm cysts in the kidneys. These cats were also positive for PKD on ultrasound examination as part of the present study at the Cairo university hospital and private clinics at Giza governorate.

The ultrasonographic pattern of multiple anechoic cysts with distal enhancement is highly specific for PKD. So, the sensitivity and specificity of ultrasonographic examination, defined as the number of cats positive in all 9 cats on ultrasound examination as assessed by renal cytology and histopathological examination.

Anemia was the most common abnormality revealed by CBC and was characterized as non regenerative normocytic, normochromic anemia in diseased cats with absence of characteristics of regeneration (ie, reticulocytes, polychromasia). The mechanism that may contribute to such anemia is erythropoietin (EPO) deficiency resulting from an inability of the renal fibroblasts to produce EPO to maintain the erythrocytic mass in response to tissue hypoxia. As the renal function deteriorates, the anemia becomes more marked. The anemia of chronic renal failure is not normally observed until the glomerular filtration rate drops to 20–40 ml/min, which is equivalent to a serum creatinine of greater than 177 µmol/l (3 mg/dl) **(Chandra et al., 1988 and Bosman et al., 2001)**. In our study creatinine concentrations were exceed 3mg/dl in all diseased cats indicating that EPO deficiency is the major explanation.

The leukogram showed neutrophilic leucocytosis with left shift and monocytosis as a response of bone marrow to chronic interstitial nephritis by releasing the storage pool of post-mitotic mature and band neutrophils which results in a neutrophilia with a left shift in diseased cats. Monocytosis is usually seen with inflammation when it is long-standing disease. Cats with blood leukocyte counts higher than the normal reference had significantly shorter renal survival times compared with cats with blood leukocyte counts within the normal range as it reflect progression of the disease **(King et al., 2007)**.

The Clinicopathological abnormalities identified in diseased cats were attributed to the severe malformation and malfunction of both kidneys. The impaired renal function also explains the severe azotemia, attributed to renal failure. Elevated plasma inorganic phosphate, hypoproteinemia and hypocalcemia may be a consequence of progressive loss of renal function which is supported by histopathological results as well as ultrasonographic examinations **(Medina -Torres et al., 2014, Barthez et al., 2003and Lawson et al., 2015)**.

Cytological evaluation of renal impression smears of diseased cats showed few renal tubular epithelial cells due to atrophy of most renal tubules as a result of cystic fluid pressure on the renal tubules. The cysts in PKD derive from focal outgrowths of renal tubule segments. Cellular proliferation of the abnormal renal tubule epithelial cells **(Cowley et al., 1991)** and abnormalities in the transport of solutes and fluid across the epithelium **(Wallace et al., 1996)** combine to generate an expanding neoplastic structure. Renal cysts are in fact benign epithelial neoplasms that differ from more common tumors by being filled with fluid rather than cells and debris. The fluid derives from non reabsorbed glomerular filtrate accumulated in a tubule segment that

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is expanding by increasing the rate of proliferation. When these cysts exceed a diameter of approximately 2 mm, they separate from the parent tubule and become isolated sacs **Garantham (2002).** The subsequent enlargement of the cysts leads to secondary effects in the kidneys including lymphocytic inflammation, tubular cast and destruction of adjacent normal parenchyma, ultimately leading to renal insufficiency and death in affected individuals.

In conclusion, polycystic kidney disease in Persian and Persian related cats was easily recognized by ultrasonographic examination. The clinical and clinicopathological abnormalities identified in diseased cats were related and attributed to the severe renal impairment. Ultrasonography was decisive and prior to physical, clinicopathological examination to identify PKD-affected cats and remove them from the breeding pool by spaying and neutering.

Table (1): Mean value hemogram of both apparently normal and PKD affected cats.

Parameter	PKD affected cats	Control
RBCs count ($\times 10^6$/µl)	3.96 ± 0.11 *	6.86 ± 0.24
PCV(g/dl)	25.66 ± 0.33 *	40.4 ± 0.40
HB (%)	8.48 ± 0.30 *	12.93 ± 0.12
MCV(Fl)	64.92 ± 1.57	59.29 ±1.76
MCHC (g%)	33.48 ± 0.78	31.97 ±0.11
TLC ($\times 10^3$/ µl)	8.32 ±0.31 *	4.81 ± 0.23
Neutrophil ($\times 10^3$/ µl)	7.13 ±0.19*	3.66 ± 0.13
Band cells ($\times 10^3$/ µl)	1.42±0.14*	0.05±0.01
Lymphocyte ($\times 10^3$/ µl)	0.47 ±0.01	1.03 ± 0.12
Eosinophil ($\times 10^3$/ µl)	0.22 ±0.05	0.12 ± 0.17
Monocyte($\times 10^3$/ µl)	0.49 ±0.01*	0.18 ± 0.09

*** Significantly differ at P<0.05.**

Table (2): Mean value serum biochemical parameters of both apparently normal and PKD affected cats.

Parameter	PKD affected cats	Control
Total Protein (g/dl)	3.97±0.11*	8.12±0.36
Albumin (g/dl)	2.42±0.06*	4.26±0.23
Globulin (g/dl)	1.55±0.16*	3.83±0.17
A/G	1.72±0.22	1.11±0.04
Creatinine (mg/dl)	3.86±0.29*	1.04±0.07
BUN (mg/dl)	159.37±9.30*	22.91±0.67
Calcium (mg/dl)	4.40±0.17*	10.17±0.23
Phosphorus (mg/dl)	7.56±0.19*	4.8±0.18

*** Significantly differ at P<0.05.**

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الملخص العربي

مرض الكلى المتعدد الكيسات في القطط واحد من أهم أسباب الفشل الكلوي في القطط الفارسية و الفارسية ذات الصلة و هو المرض الوراثي الاكثر انتشارا بين القطط. يهدف هذا العمل لدراسة حالات مرض الكلى المتعدد الكيسات في القطط عن طريق الموجات فوق الصوتية، والاختبارات المعملية، وكذلك نتائج الفحص الخلوي. وقد أجريت هذه الدراسة على أربعة عشر قطة فارسية و فارسية ذات الصلة من العيادات البيطريه فى محافظه الجيزة (9 قطط مريضة و5 قطط اصحاء ظاهريا) بين نوفمبر 2014 وديسمبر 2015. وتراوحت أعمار القطط بين 6-15 سنوات. تم إخضاع جميع الحيوانات إلى الأشعة فوق الصوتية، والاختبارات المعملية ، في حين تم القيام بالفحوص الخلوية والتشريحية المرضية للحيوانات المريضة بعد التشريح. ظهرت الموجات فوق الصوتية ضخامة الكليتين مع وجود كيسات متعددة بشكل غير منتظم داخل قشرة الكلى والنخاع. فقر الدم غير التجديدي كان الأكثر شيوعا، أما كيمياء الدم تميزت بالآزوتيمية شديدة مع فرط نقص بروتينات الدم ونقص كلسيم الدم نتيجة الفقدان التدريجي لوظيفة الكلى. وأظهر الفحص الخلوي الكلوى للقطط المريضة وجود انابيب كلوية مع الخلايا الليمفاوية. أما مسحات سائل الكلى فهو قليل الخلايا مع وجود خلايا ضامة ذات سيتوبلازم رغوي. نستنتج من هذة الدراسة وجود تشوهات سريرية وإكلينيكية في القطط المريضة ويعزى إلى القصور الكلوي الحاد. لذلك فإن أفضل طريقة لمنع مرض الكلى المتعدد الكيسات في القطط هو تحديد القطط المريضة باستخدام الموجات فوق الصوتية و منعها من التزاوج للحد من انتشار المرض.