Article

Feline polycystic kidney disease

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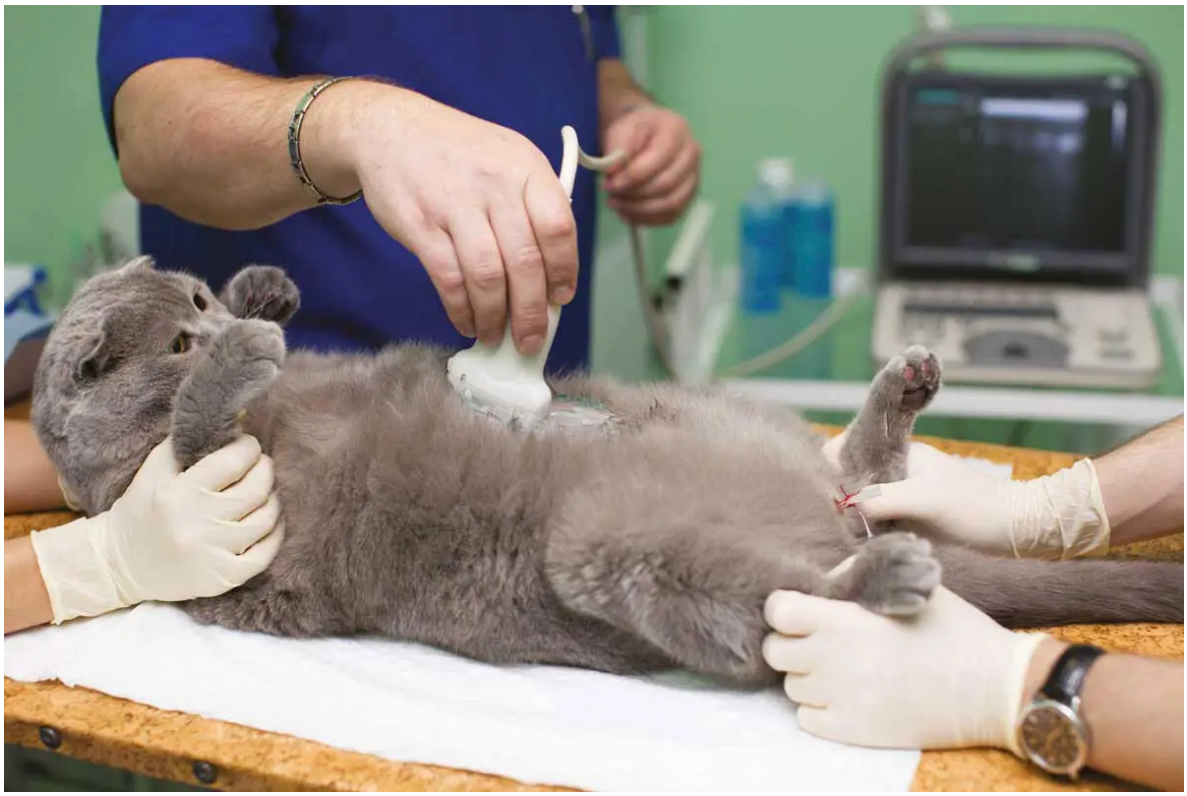
Once known as the Persian Cat Disease, our knowledge of this condition, and the ways in which it can be diagnosed and perhaps eradicated, continue to evolve.



Key points

1

Autosomal Dominant Polycystic Kidney Disease (ADPKD), or Polycystic Kidney Disease (PKD), is an autosomal dominant hereditary disease characterized by the formation of renal cysts.

2

The condition involves both kidneys in affected cats, but it can also occasionally lead to cyst formations in other organs such as the liver and pancreas.

3

Ultrasonography is the most practical non-invasive diagnostic method for identifying PKD in adult cats.

4

PKD is an incurable and progressive condition; treatment options will be based on clinical signs and in line with any other cause of chronic kidney disease.

Introduction

Major changes in society over the last 30-40 years have transformed veterinary medicine for companion animals. The prevalence for large households with many children has lessened, leading to a reduction and simplification of living spaces. Furthermore, people are now less likely to live close to their workplace, and will often leave home early each day and return late from work. However, the inherent human need to take care of a living creature has not diminished, and over the last few decades many pets have moved from the backyard into the domestic home. The upshot of these changes is an increase in the worldwide popularity of cats, partly because they are considered independent and less reliant on the presence of their caregiver. Therefore human-cat relationships are increasingly significant, and the domestic feline population continues to increase; a recent estimate (in 2023) put the number of household-owned cats (i.e., not strays) in Europe at around 129 million (for comparison, the number of dogs in the same category is estimated at around 106 million) (1). Given the need to provide state-of-the-art care for the feline species, this article offers an update on a condition that was first identified in Persian cats some 35 years ago (2).

Etiopathogenesis

Autosomal Dominant Polycystic Kidney Disease (ADPKD), or in its simplified version Polycystic Kidney Disease (PKD), is an autosomal dominant hereditary condition, characterized by the formation of renal cysts; both kidneys will be involved in an affected individual, and occasionally cystic formations in other organs such as the liver and pancreas will also develop. The condition occurs in many species, including humans, and in any breed of cat, but its prevalence in Persian cats has led to it being known as the "Persian cat disease".

The *Polycystic Kidney Disease 1* (*PKD1*) gene, located on chromosome 16p13.3, is responsible for encoding polycystin-1 (PC1), a vital protein found in renal tubule cells, and a mutation in this gene is considered the main cause of ADPKD (3). This mutation produces defective polycystin-1, resulting in abnormalities such as amino acid sequence modifications, reduced functional capacity, and inhibited control of cell growth (Figure 1) (3). This leads to a loss of renal tubular integrity, with a progressive dilation of the tubules to form cysts which are filled with fluid, generally glomerular filtrate. Many cysts of variable diameter can develop throughout the kidneys, causing severe damage to the renal parenchyma (Figure 2) with destruction of the nephrons, a reduction in functional renal mass, and subsequent chronic kidney disease (CKD). About 85% of PKD cases are caused by *PKD1* mutations, while the remaining 15% are associated with the *PKD2* gene (Polycystic Kidney Disease 2) (3).

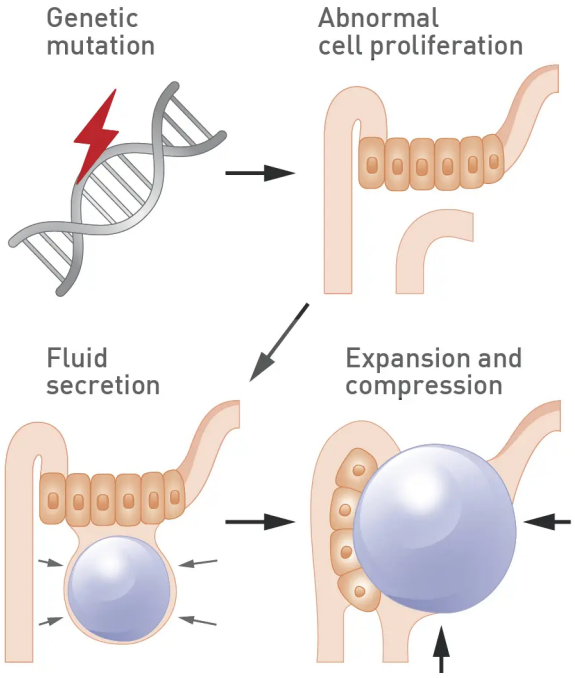


Figure 1. A diagram illustrating the sequence of PKD formation: PKD1 gene mutation leads to abnormal renal tubule cell proliferation, resulting in intercellular integrity loss and cystogenesis. Note also the compressive effects of the cyst, an important factor for the onset and progression of chronic kidney

disease. © AI created/redrawn by Sandrine Fontègne

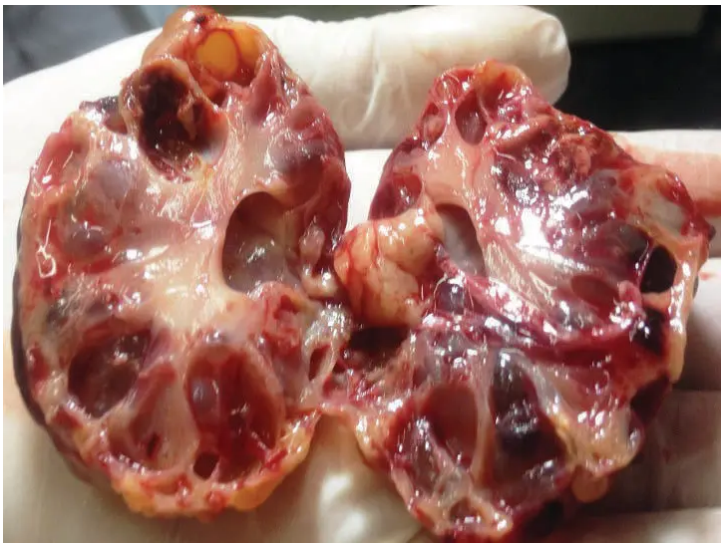


Figure 2. A necropsy sample from a Persian cat with PKD; multiple cystic areas are present throughout the kidney. © Tathiana Mourão Anjos DVM

Epidemiological studies

Literature reports detailing the incidence of feline PKD cases started to appear around thirty years ago. An Italian study evaluating ultrasound findings in Persian and Exotic Shorthair cats reported 41% of animals studied had renal cysts (4), whilst a French review identified the presence of cysts as ultrasonographic findings in 41.8% of Persian cats and 39.1% of Exotic Shorthair cats (5). The incidence was also high outside Europe - for example, a 2001 Australian study noted cystic findings in 45% of Persian cats evaluated by ultrasound imaging (6). The development of molecular diagnostic methods that enabled detection of *PKD1* gene mutations further aided awareness of the condition (7,8). The authors of one of these papers, published in 2004, advised that "Persian and Persian-related cats should be screened for PKD by ultrasound before they are bred" but went on to note that although breeders were advised not to breed from affected cats, it was recognized that "some individuals carrying the defective gene might still be used for breeding." Several reasons were cited for this, including the fact that cats did not show clinical signs of renal disease; some breeders were unaware of the condition; ultrasound imaging was either unavailable or cost-prohibitive; breeding decisions were made before accurately identifying affected cats; and that the high prevalence of the condition meant that many breeders might lose a large proportion of their breeding stock. The authors went on to report that "a genetic test for feline PKD (would) provide breeders with an efficient and accurate means to selectively breed their cats and remove PKD from the population" and advised that "because PKD has been found in other cat breeds related to Persians, the incidence of PKD in these breeds should (also) be evaluated".

Research in the early years of this century therefore raised awareness of PKD and may have had a positive impact on breeders' behavior, such that animals known to carry the defective gene were excluded from breeding programs. This may be reflected in the findings of more recent studies on the incidence of PKD, which have started to report different results. Given the increased awareness of the disease, and alongside new molecular diagnostic methods that can detect *PKD1* gene mutations, there has been a significant reduction in the number of cases detected. A recent study in Mexico evaluated PKD prevalence using a Polymerase Chain Reaction-Restriction Fragment Length Polymorphism (PCR-RFLP) assay to genotype a cohort of Persian cats (8). The results showed that 23% tested positive for PKD, but also noted that, although diagnostic ultrasonography has high sensitivity to detect the formation of cysts in adult cats, it is not reliable in cats under the age of 10 months. However, the authors reported that molecular testing can recognize affected kittens as early as the first day of life, so this diagnostic technique is invaluable for enabling breeders to better control and eradicate PKD from feline populations.

In a recent Brazilian study, genomic DNA was extracted from peripheral whole blood samples or oral swabs and evaluated using PCR-RFLP methodology (9). Of 537 Persian cats studied, only 27 had the single nucleotide variant (C to A) at position 3284 in exon 29 of the *PKD1* gene. This pathogenic variant has been identified only in a heterozygous state. The prevalence of PKD in Persian cats and Persian-related breeds was 5.03% and 1.6%, respectively. There was no significant correlation between the cat breed, gender or age and the prevalence of PKD, as evidenced in other studies. It should be emphasized that the prevalence of PKD in these cats was lower than those reported in other parts of the world and at other time points, and the authors suggest this finding may be related to genetic counselling and subsequent selection of PKD-free cats for breeding. However, a recent Japanese study also suggested that PKD is becoming less common; of 1,281 cats evaluated using real-time PCR, 23 (1.8%) had the conventional *PKD1* variant, and only four cats were Persians (10).

Although studies on the prevalence of PKD in the USA are generally outdated, with a reported incidence of between 38% (7) and 49% (11) of cases in Persian cats, a more recent paper noted a significant reduction (↓80%) in genetic testing for feline PKD at the UC Davis Veterinary Genetics Laboratory (12), indicating a decrease in the number of animals tested positive for this condition. These data highlight the idea that breeding programs have been carried out by cat breeders in the USA in recent years to successfully reduce the prevalence of PKD. Perhaps this is not surprising; in veterinary medicine nowadays, knowledge and perception of a hereditary condition gradually become more widespread, better molecular diagnostic methods are developed, and the importance of identifying carrier animals and eradicating them from breeding programs is recognized, greatly reducing the overall incidence of cases.

Identification and evaluation methods for PKD

Renal ultrasonography is the most practical non-invasive diagnostic method for

identifying PKD in adult cats (Figures 3 and 4). The sensitivity of ultrasonography alone is 75% when performed in animals at the age of 16 weeks, and 91% when done at 36 weeks of age, so sensitivity increases with age. Ultrasound has a specificity of 100% when testing animals for possible PKD when performed at 3 months of age (13).

Conventional or real-time PCR are currently considered the most popular options of the existing molecular techniques. Such methods can allow diagnosis of inherited diseases, identify gene mutations or polymorphism, perform genetic typing, and evaluate gene expression of the animal studied (9). However, ultrasound imaging has some advantages over genetic testing, because the former can identify other forms of cystic kidney disease, as well as assessing the severity and progression of the condition (14). Therefore, a combination of genetic testing and imaging can be important for both early detection and follow-up of PKD (3).

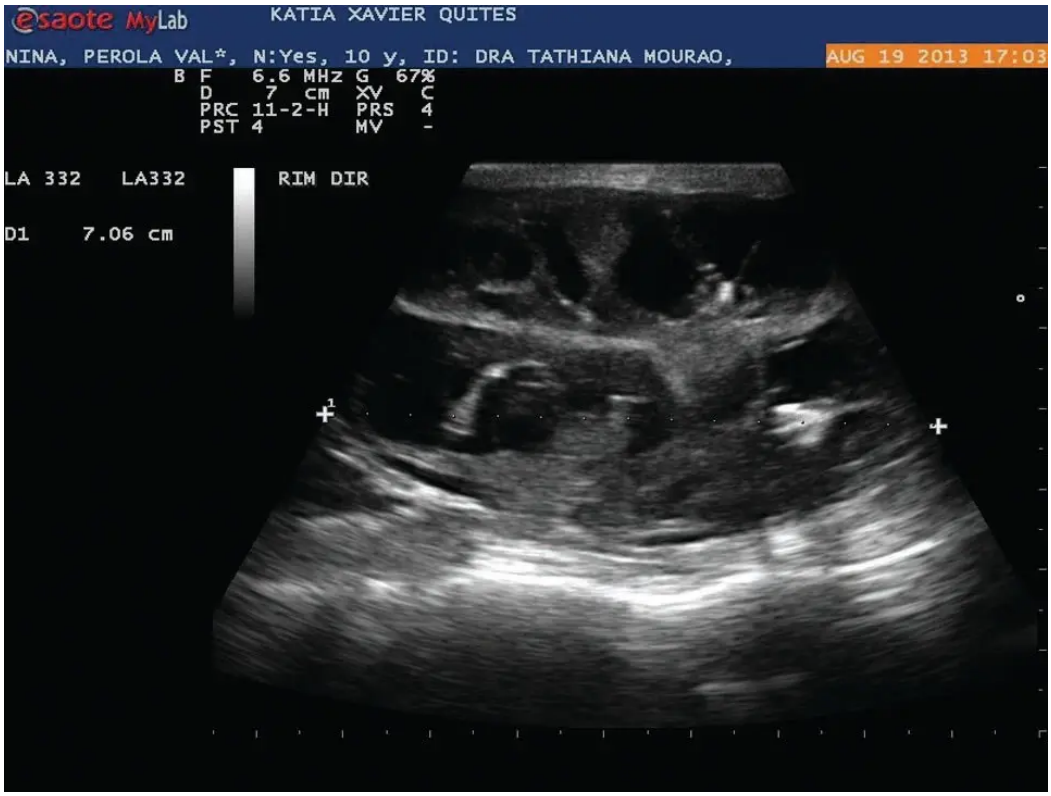
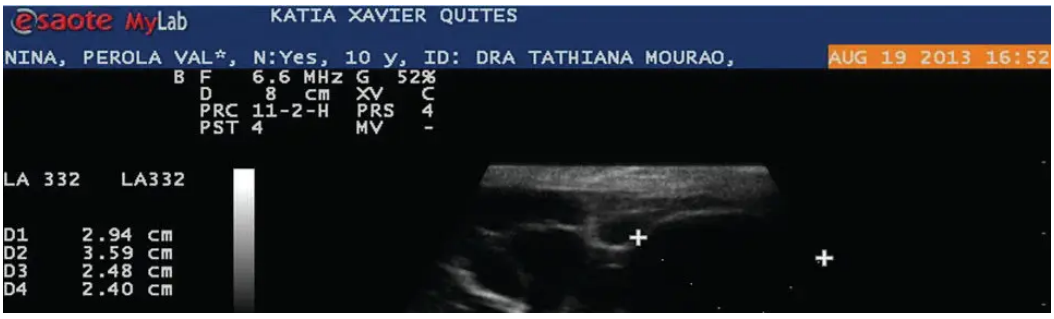


Figure 3. Ultrasound image of the right kidney in a Persian cat with PKD. Note the presence of multiple cysts showing circular and semicircular anechoic areas scattered between the cortical and medullary regions. © Tathiana Mourão Anjos DVM



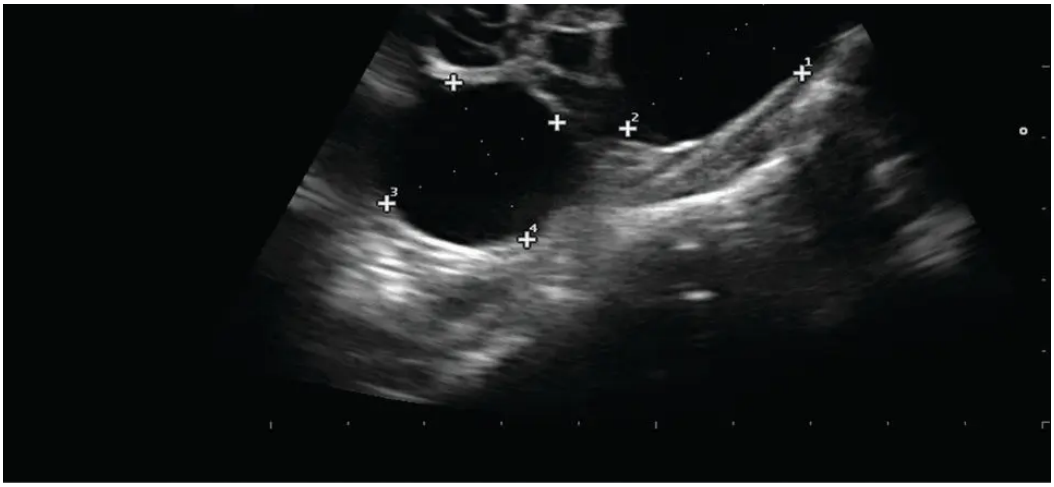
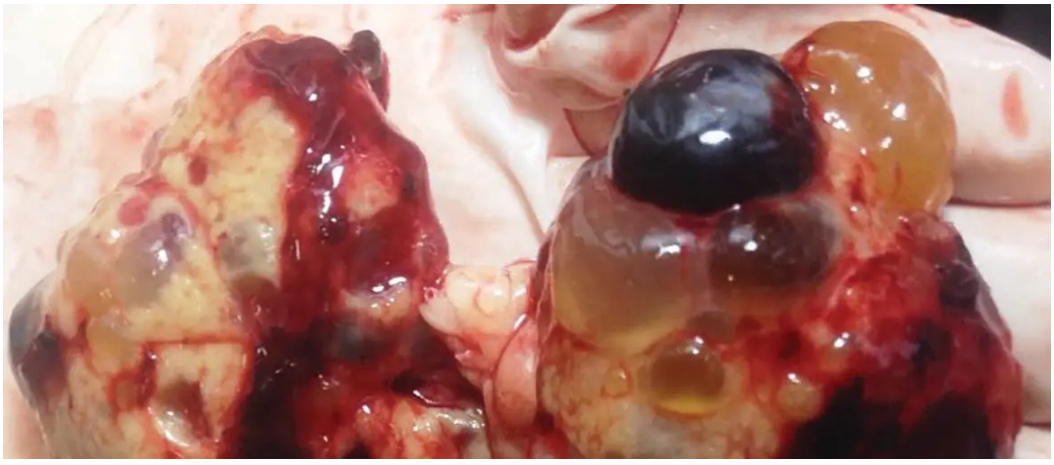


Figure 4. Ultrasound image of a kidney in a Persian cat with PKD. Large cysts of significant dimensions are visible. © Tathiana Mourão Anjos DVM

Clinical presentation and management of PKD

Although PKD can develop during pregnancy, and many kittens are born with cysts, these increase in both number and size with age. Many cats can therefore be subclinical carriers for several years, and the clinical signs are not pathognomonic for PKD, as it manifests as chronic renal failure. The average age of onset of clinical signs is seven years, although they can appear at any time between three and ten years of age. There is significant individual discrepancy in the progression of the disease, suggesting that various factors are at play (3). Clinical signs can include apathy, anorexia, weight loss, wasting, polyuria and polydipsia, as well as gastrointestinal disorders. During clinical evaluation, dehydration, pale mucous membranes and weight loss, as well as enlargement and irregular contouring of the kidneys on palpation (signs related to chronic kidney disease) may be observed (Figure 5). Laboratory tests are not specific, and various abnormalities caused by renal failure (*e.g.*, azotemia, hyperphosphatemia, non-regenerative anemia and proteinuria) are commonly found (3).



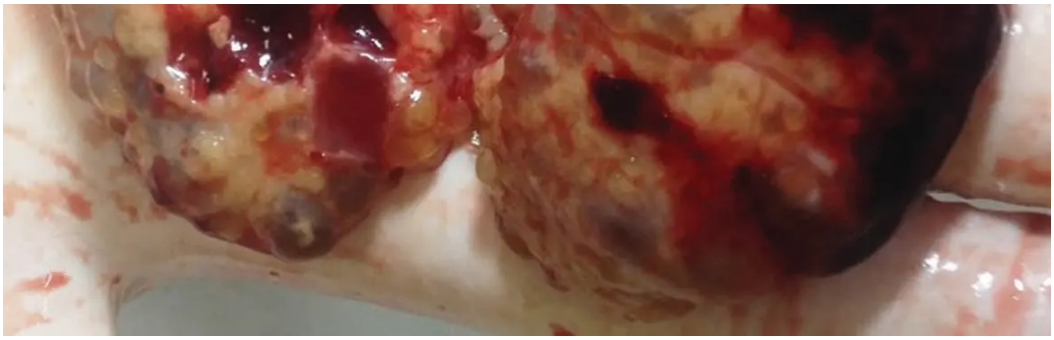


Figure 5. A necropsy sample from a Persian cat with PKD showing the exterior surface of a kidney with multiple cysts; irregular contours of the kidneys may be detected at clinical examination when palpating the abdomen of an affected animal. © Tathiana Mourão Anjos DVM

Whilst the *PKD1* gene mutation primarily causes renal tubular changes, it can also induce cystic formations in the liver and pancreas, although these are relatively rare; hepatic cysts may be found in around 12% of PKD-affected animals (Figure 6). If detected, they should be considered as a diagnostic pointer. Rarely, clinical signs associated with liver failure are seen, and the underlying nature of this process has not yet been established (3).

Unfortunately, PKD is an incurable and progressive disease. Treatment is based on clinical signs and conservative management of CKD, following the International Renal Interest Society (IRIS) guidelines (15). These include provision of a specific diet from stage II CKD onwards to promote patient quality of life and longevity. Commercial renal diets aim to reduce nitrogenous waste by providing appropriate levels of high biological value protein, control blood pressure and hyperphosphatemia by restricting sodium and phosphorus intake respectively, stimulate nitrogen-dependent intestinal bacterial growth, and promote antioxidant activity through the use of omega-3 fatty acids and minerals (16).

Once CKD has been diagnosed, the prognosis will depend on many factors. It may be cautious in older patients that have few cysts, but poor in younger animals that have numerous cysts. The prognosis is also based on how these features link to CKD: the greater the number of cysts, the smaller the number of functional nephrons, the poorer the renal function, the greater the expected disease progression, and the worse the prognosis (3,15).

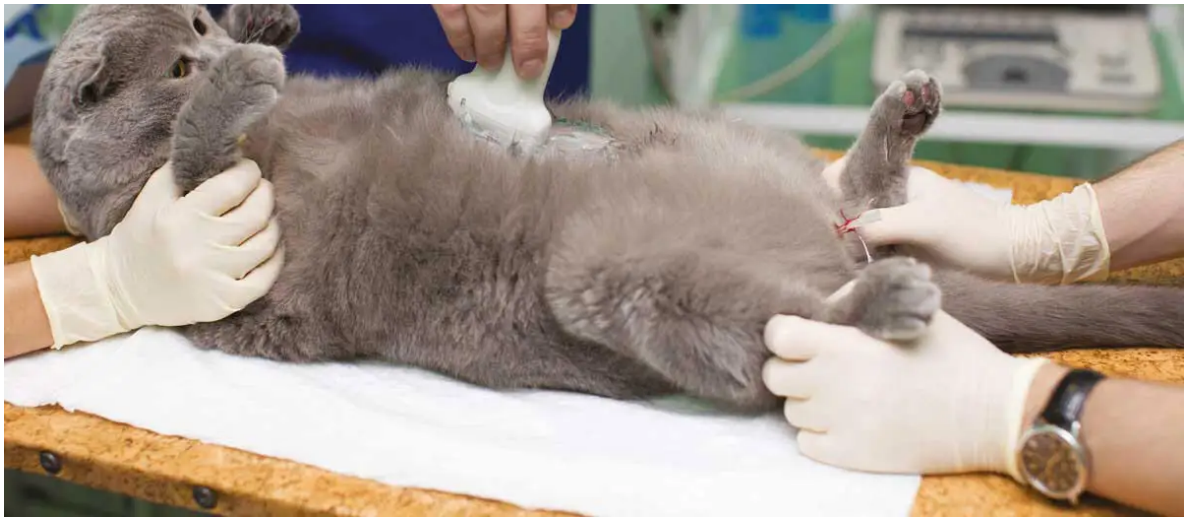


Figure 6. Ultrasonography may not only detect polycystic kidneys; in some affected cats it may also identify hepatic cysts. © Shutterstock

Medical insights and future perspectives

Insights into the human situation with respect to PKD may guide possible future perspectives for the condition in cats (16); although important updates in the medical field may not yet have found their way into the veterinary literature, it is pertinent to mention a couple of factors. In humans, glomerular filtration rate (GFR) is considered the best indicator of overall kidney function, and allows progression of PKD to be assessed; however, this is rarely performed in the veterinary clinic, and creatinine levels are still seen as a critical marker for disease progression in animals (16). An interesting development in the human field is the Mayo Imaging Classification (MIC), a risk stratification tool for patients with PKD (17). It uses computed tomography or magnetic resonance imaging to evaluate the total volume of a patient's kidneys; once adjustments have been made for the individual's height and age, the result can be used to predict the rate of disease progression. Finally, tolvaptan, a selective vasopressin V2 receptor antagonist, is the only product currently approved for use by U.S. Regulatory Agencies as a disease-modifying therapy for PKD in humans (16). However, such predictive methods for assessing progression, and possible drugs for preventing progression, have yet to be studied in animals, in particular Persian cats.

"The greater the number of cysts, the smaller the number of nephrons, the poorer the renal function, the greater the expected disease progression, and the worse the prognosis."

— Júlio César Cambraia Veado

Conclusion

PKD is not a condition specific to Persian cats, and it can occur in other breeds and other species, including humans. An autosomal dominant disease, it results from a mutation in either the *PKD1* or *PKD2* gene, and although it is inherited, signs generally do not appear until adulthood. Veterinarians play a crucial role in identifying, diagnosing and recommending the removal of mutation carriers from breeding programs, and their efforts – in combination with breeders – has helped reduce the prevalence and incidence of PKD, such that the condition is now seen less commonly. Based on this scenario, it is possible that PKD could in future become a rarity, and will no longer carry the stigma of the "Persian cat disease".

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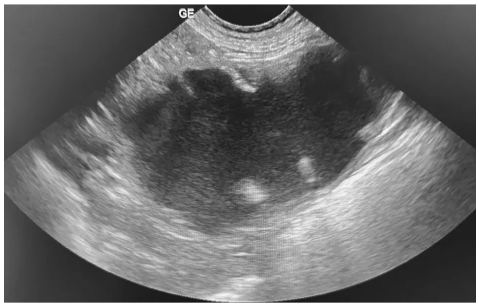

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