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Anatomopathological aspects of chronic kidney disease in cats

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Abstract: Chronic kidney disease (CKD) is one of the most frequently encountered conditions in cats, particularly in geriatric cats, and represents a major cause of morbidity and mortality. The present study describes post-mortem renal morphological changes associated with several ante-mortem biochemical parameters in cats diagnosed with renal failure. Renal tissue samples with macroscopic lesions identified at necropsy were collected from two of the eleven examined cats. Certain renal areas showed visible atrophy, and the consistency in these regions was markedly increased, making sectioning difficult. Tissue fragments were processed, embedded in paraffin, and stained using the hematoxylin–eosin–methylene blue trichrome technique (HEA). Microscopically, moderate scarring of the renal parenchyma was observed due to expansion of the interstitial matrix (interstitial fibrosis), as well as well-demarcated areas of tubular atrophy. Glomerulosclerosis, with glomerular cell proliferation, urinary casts, and rare hyperplasia of renal arterial walls, were also observed. Serum biochemical analysis performed prior to death revealed, in most cases, significant increases in blood urea nitrogen (BUN) and creatinine (CREA), indicative of severe azotemia, as well as elevated serum phosphorus levels. Additionally, variations in electrolytes were observed, characterized by changes in sodium (Na^+), potassium (K^+), and chloride (Cl^-) concentrations, with a tendency toward hyperkalemia in some cases and associated electrolyte imbalances. Blood glucose (GLU) levels showed individual variation without a consistent pattern, suggesting the influence of systemic factors and overall clinical condition. The severity of biochemical alterations, particularly elevated urea and creatinine levels, was associated with renal changes observed microscopically, consistent with chronic kidney disease in cats. These findings highlight the importance of integrating clinical and biochemical data with post-mortem morphological evaluation for a better understanding of disease progression and for establishing the anatomopathological diagnosis.

Keywords: chronic kidney disease; feline; interstitial fibrosis; glomerulosclerosis; histopathology

• Introduction

Chronic kidney disease (CKD) is one of the most frequently diagnosed conditions in cats, particularly in geriatric animals, and is associated with a high rate of morbidity and mortality [Brown et al., 2016]. This condition is characterized by the progressive and irreversible loss of functional nephrons, accompanied by the development of chronic structural changes at the renal level, ultimately leading to impaired renal function and the onset of chronic renal failure [Brown et al., 2016].

From a histopathological perspective, kidneys affected by CKD in cats commonly exhibit interstitial fibrosis, tubular atrophy, glomerulosclerosis, and vascular alterations, all considered characteristic lesions of chronic renal disease progression [Chakrabarti et al., 2013]. Interstitial fibrosis and tubular atrophy have been shown to represent some of the most important microscopic changes associated with the severity of renal dysfunction in cats, with the extent of these lesions correlating with serum urea and creatinine levels [Chakrabarti et al., 2013]. In addition, glomerulosclerosis, glomerular cellular proliferation, tubular casts, and alterations of renal vascular walls have been described, all contributing to progressive remodeling of the renal parenchyma [Chakrabarti et al., 2013].

Tubulointerstitial changes constitute the dominant morphological component in feline CKD, with interstitial fibrosis being regarded as a central feature in the progression of chronic renal lesions [Brown et al., 2016]. The mechanisms involved in the development of renal fibrosis include expansion of the extracellular matrix and activation of myofibroblasts, processes responsible for the progressive scarring of renal parenchyma [Lawson et al., 2018]. A multifunctional cytokine involved in the regulation of cell proliferation, differentiation, inflammation, and tissue repair processes (TGF-β1) can induce fibroblast-to-myofibroblast differentiation, a process considered essential in the progression of renal fibrosis [Lawson et al., 2018].

In addition to morphological alterations, chronic kidney disease in cats is associated with important biochemical changes reflecting the progressive decline of renal function and impairment of filtration and excretory mechanisms. Among the most frequently reported alterations are increased serum blood urea nitrogen (BUN) and creatinine (CREA) concentrations, considered key markers of azotemia and reduced glomerular filtration rate [Deguchi et al., 1997; Sparkes et al., 2016]. Furthermore, elevated serum phosphorus levels have been described in cats diagnosed with CKD, with hyperphosphatemia being commonly associated with the progression of chronic renal failure and disturbances of mineral homeostasis [Sparkes et al., 2016; Grelová et al., 2022].

Biochemical alterations in feline CKD also include variable electrolyte imbalances, characterized by changes in serum sodium (Na^+), potassium (K^+), and chloride (Cl^-) concentrations, resulting from impaired renal capacity to maintain hydroelectrolytic balance [Deguchi et al., 1997; Sparkes et al., 2016]. In some cases, a tendency toward hyperkalemia has been reported, associated with reduced renal potassium excretion and advanced stages of renal failure [Deguchi et al., 1997].

Glucose (GLU) levels may show individual variation in cats with CKD, without a consistent pattern, these changes being influenced by the overall metabolic status, concurrent diseases, and the clinical condition of the animal [Gorman et al., 2016].

• Material and method

The study was conducted on a group of 11 cats (P1–P11) evaluated within an investigation of chronic kidney disease, in which serum biochemical analyses were performed to determine the main parameters of renal function and metabolic balance, including blood urea nitrogen (BUN), creatinine (CREA), glucose (GLU), triglycerides (TG), sodium (Na^+), potassium (K^+), chloride (Cl^-), and phosphorus (PHOS). The results were interpreted in relation to feline species-specific reference intervals.

Out of the 11 animals included in the study, renal tissue samples were collected post-mortem from only two cats, in which necropsy examination revealed suggestive gross renal lesions. Tissue fragments were processed using standard paraffin-embedding techniques, sectioned, and stained with hematoxylin–eosin–methylene blue (HEA), and subsequently examined microscopically for the evaluation of renal structural changes. The histopathological analysis aimed to identify lesions characteristic of chronic kidney disease.

The serum biochemical parameters determined in the study were intended to highlight alterations in renal function and to support the morphological lesions identified at the renal level.

• Results and discussions

On gross examination, certain renal regions showed visible focal atrophy, accompanied by a marked increase in parenchymal firmness, a feature that resulted in technical difficulty during tissue sectioning (FIG.1).

Microscopic examination revealed moderate to severe interstitial fibrosis (Fig. 3), characterized by expansion of the extracellular matrix and progressive replacement of functional renal parenchyma. Well-demarcated areas of tubular atrophy (Fig. 2) were identified, associated with epithelial degeneration and loss of normal tubular architecture. At the glomerular level, marked glomerulosclerosis was observed (Fig. 3), occasionally accompanied by residual glomerular cellular proliferation. Proteinaceous urinary casts were identified within the tubular lumen (Fig. 2). Additionally, mild hyperplasia of the renal arterial walls was noted.

The mean values of the main biochemical parameters relevant for the assessment of renal function (BUN, creatinine, phosphorus, electrolytes, and associated metabolites) in the 11 cats included in the study are presented in a summarized form in Table 1, in comparison with feline species-specific reference intervals.

Table 1. Serum biochemical parameters in cats included in the study (P1–P11), in relation to feline species-specific reference intervals

Parameter	Mean value	Reference interval
BUN (mg/dL)	131.6	19 – 34
GLU (mg/dL)	211.8	60 – 120
TG (mg/dL)	132.7	8.9 – 115.1
CREA (mg/dL)	7.9	0.9 – 2.2
Na^+ (mmol/L)	143.0	146 – 156
K^+ (mmol/L)	5.0	3.7 – 6.1
Cl^- (mmol/L)	103.1	115 – 130
PHOS (mg/dL)	12.9	3.0 – 6.1

Fig.1. CKD – macroscopic appearance of the kidneys in felines

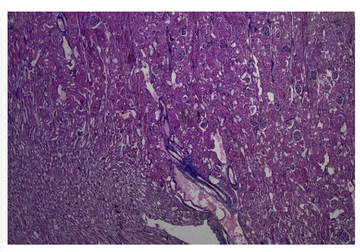

Fig.2. CKD – areas of tubular atrophy and protein casts

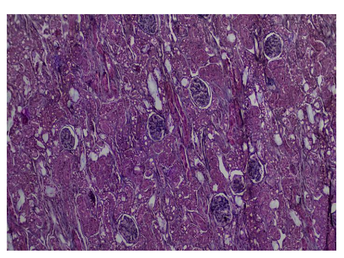

Fig.3. CKD - fibroconnective hyperplasia and glomerulosclerosis

The results obtained in the present study highlight renal histopathological changes characteristic of chronic kidney disease (CKD) in cats, predominantly represented by moderate to severe interstitial fibrosis, tubular atrophy, glomerulosclerosis, and the presence of proteinaceous casts.

These findings are consistent with data reported in the literature by Chakrabarti et al. (2013), Brown et al. (2016), and Lawson et al. (2018), who describe feline CKD as a progressive condition characterized by irreversible renal remodeling, with predominant tubulo-interstitial involvement and secondary glomerular impairment.

From a clinical and paraclinical perspective, serum biochemical analysis performed in the studied animals revealed alterations compatible with renal dysfunction. These changes included abnormalities in azotemic parameters and hydro-electrolytic balance, findings frequently reported in feline chronic renal failure. In previous studies, Deguchi et al. (1997), Sparkes et al. (2016), and Lawson et al. (2018) demonstrated that the biochemical alterations characteristic of feline CKD, particularly increased serum urea and creatinine concentrations, together with disturbances in phosphorus-calcium balance and electrolyte imbalances, represent markers of global renal dysfunction and support the presence of structural renal lesions.

• Conclusions

Histopathological examination revealed the presence of renal lesions characteristic of chronic kidney disease in cats, predominantly represented by glomerulosclerosis, interstitial fibrosis, tubular atrophy, and the presence of intratubular proteinaceous casts.

Serum biochemical investigations demonstrated alterations in parameters specific to renal function and metabolic balance, findings consistent with the development of chronic renal dysfunction.

By correlating the histopathological and biochemical findings, the obtained results confirm the diagnosis of chronic kidney disease in cats, supporting the progressive and irreversible nature of renal impairment.

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