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Renal amyloid protein deposition in a Shar Pei dog

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ABSTRACT

Renal amyloidosis is a glomerular disease with a familial predisposition, particularly common in Shar Pei dogs, and is associated with clinical manifestations of chronic kidney disease (CKD). This case report describes a female Shar Pei diagnosed with renal amyloidosis, similar to her father and brother. A nine-year-old female Shar Pei presented with a history of vomiting, halitosis, hyporexia, polyuria, and polydipsia. Structural and functional renal abnormalities were observed, including azotemia, cortical hyperechogenicity, and a mild reduction in corticomedullary differentiation. Given the patient's clinical condition, euthanasia was performed, and renal tissue samples were submitted for histopathological analysis. The presence of eosinophilic fibrillar material within the glomeruli and some tubules confirmed the diagnosis of renal amyloidosis.

Keywords: chronic kidney disease, glomerulopathies, amyloid protein.

Deposição de proteína amiloide renal em cão da raça Sharpei

RESUMO

A amiloidose renal é uma doença glomerular de características familiares, especialmente para Sharpeis e está associada com manifestações clínicas de doença renal crônica (DRC). O objetivo do presente relato é descrever o caso de uma fêmea, da raça Sharpei, diagnosticada com amiloidose renal, de forma semelhante aos seu pai e irmão. Cão, fêmea, nove anos, Sharpei, foi atendida mediante histórico de vômitos, halitose, hiporexia, poliúria e polidipsia. Alterações estruturais e funcionais dos rins, tais como azotemia, hiper ecogenicidade cortical e redução discreta da definição corticomedular foram encontradas. Diante do quadro clínico, a paciente foi submetida à eutanásia e fragmentos renais foram encaminhados para análise histopatológica. Observou-se a presença de material de características eosinofilicas e fibrilares no interior dos glomérulos e em alguns túbulos, sendo o diagnóstico definitivo de amiloidose renal.

Palavras-chave: doença renal crônica, glomerulopatias, proteína amiloide.

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INTRODUCTION

Amyloidosis is characterized by the deposition and accumulation of amyloid protein in tissues and organs, such as the kidneys and liver (DiBartola *et al.* 1990) and is frequently observed in the renal region (Sonne *et al.* 2008; Woldemeskel *et al.* 2012; Segev *et al.* 2012; Serakides & Silva 2023). This condition is associated with glomerular dysfunction and, consequently, renal impairment due to structural and/or functional alterations, leading to glomerular loss and fibrosis. Amyloid protein deposition can result from primary causes, such as immunoglobulin involvement, or occur secondary to chronic inflammation (Jones *et al.* 1997; Serakides & Silva 2023; Giaretta & Barros 2023). The disease is commonly diagnosed in breeds predisposed to amyloidosis, such as Shar Peis and Beagles, although its occurrence is not exclusive to these breeds (Sonne *et al.* 2008; Segev *et al.* 2012; Jung *et al.* 2014). Notably, animals with familial relationships to affected individuals have an increased risk of developing the disease, emphasizing the genetic component associated with its pathogenesis (Sonne *et al.* 2008; Segev *et al.* 2012; Jung *et al.* 2014).

Renal amyloidosis is characterized by structural and/or functional glomerular lesions, which may present an acute or chronic course. These conditions are classified as glomerulopathies and generally carry a guarded to poor prognosis (Vaden 2011). Furthermore, a hereditary component has been identified in some cases (Sousa *et al.* 2024). The investigation of glomerular diseases, including amyloidosis, relies on the assessment of pathophysiological characteristics, enabling a more comprehensive clinical evaluation and a more accurate determination of therapeutic options (IRIS 2013). Among hereditary renal disorders, amyloidosis is particularly concerning due to its potential for genetic transmission, facilitating the propagation of affected genes across generations.

The clinical manifestations of amyloidosis in dogs are consistent with renal dysfunction and often include azotemia and functional abnormalities. As the disease progresses, chronic kidney disease (CKD) is frequently observed, although nonspecific clinical signs may also be present (Sousa *et al.* 2024). A definitive diagnosis requires histopathological evaluation of affected tissues, revealing amyloid protein deposition in glomerular and/or tubular regions (Sonne *et al.* 2008; Segev *et al.* 2012; Sousa *et al.* 2024). Once diagnosed, amyloidosis is considered incurable, and treatment primarily focuses on managing disease progression and associated complications, though therapeutic interventions often remain ineffective (Sousa *et al.* 2024). This report describes a case of renal amyloid deposition in a female Shar Pei with a familial history of amyloidosis.

CASE DESCRIPTION

A 9-year-old female Shar Pei dog, weighing 25 kg, was presented to a veterinary clinic in the metropolitan region of Belo Horizonte, MG, with a history of vomiting, halitosis, hyporexia, polyuria, and polydipsia. The owner reported that the patient was the daughter and sibling of two other Shar Pei dogs that had died with a definitive diagnosis of renal amyloidosis. On clinical examination, the patient had a heart rate of 132 bpm, a respiratory rate of 24 breaths per minute, and normal mucous membrane coloration; however, reduced brightness and moisture were

noted, consistent with approximately 8% dehydration. The lymph nodes were of normal size, volume, and appearance, with a capillary refill time of 3 seconds and a body temperature of 38.9 ºC. Moderate dental tartar and early-stage periodontal disease were present, along with bilateral necrosis near the lingual frenulum. No significant abnormalities were detected in other examined regions.

Given the strong suspicion of familial amyloidosis, laboratory tests, including a complete blood count, serum biochemistry, and urinalysis, were performed, along with abdominal ultrasonography and a canine visceral leishmaniasis screening test using enzyme-linked immunosorbent assay (ELISA) and total dilution. Laboratory analysis revealed leukocytosis due to neutrophilia without a left shift, significant azotemia, hyperphosphatemia, decreased total protein levels due to hypoalbuminemia, elevated alkaline phosphatase, hypocalcemia, hypercholesterolemia, and increased creatine phosphokinase levels (table 1). The ELISA test and total dilution for visceral leishmaniasis were non-reactive. Urinalysis revealed proteinuria (++), a urine pH of 5, and a specific gravity of 1.018.

Ultrasonographic evaluation (ESAOTE, MyLab X8) showed that both kidneys were of normal size (7 cm × 4 cm × 4.6 cm; 8 cm × 4.8 cm × 5.5 cm) but exhibited structural alterations, including moderate cortical hyperechogenicity and a slight reduction in corticomedullary differentiation, suggestive of CKD. No significant abnormalities were detected in other abdominal structures (figure 1).

TABLE 1. Laboratory findings of the patient in the case described

	Value obtained	Reference values
Leukocytes	17,470 mil/µL	6 a 16 mil/µL
Neutrophils	14,675 mil/µL	3,300 – 12,800 µL
Urea	201.80 mg/dl	15 a 40 mg/dL
Creatinine	6.04 mg/dl	0.5 a 1.5 mg/dL
UPCR	33.41 mg/dl	
Phosphorus	14.40 mg/dl	2.7 a 5.4 mg/dL
Total proteins	4.94 g/dl	5.30 a 7.80 g/dl
Albumin	1.13 g/dl	2.30 a 3.80 g/dl
Alkaline phosphatase	128.80 U/L	10 a 96 U/L
Calcium	8.15 mg/dl	9 a 11.3 mg/dl
Cholesterol	570.40 mg/dl	125 a 270 mg/dl
CPK	333.30 U/L	20 a 200 U/L

Consider: UPCR – urinary protein creatinine ratio; CPK–creatine phosphokinase

Source: own elaboration.

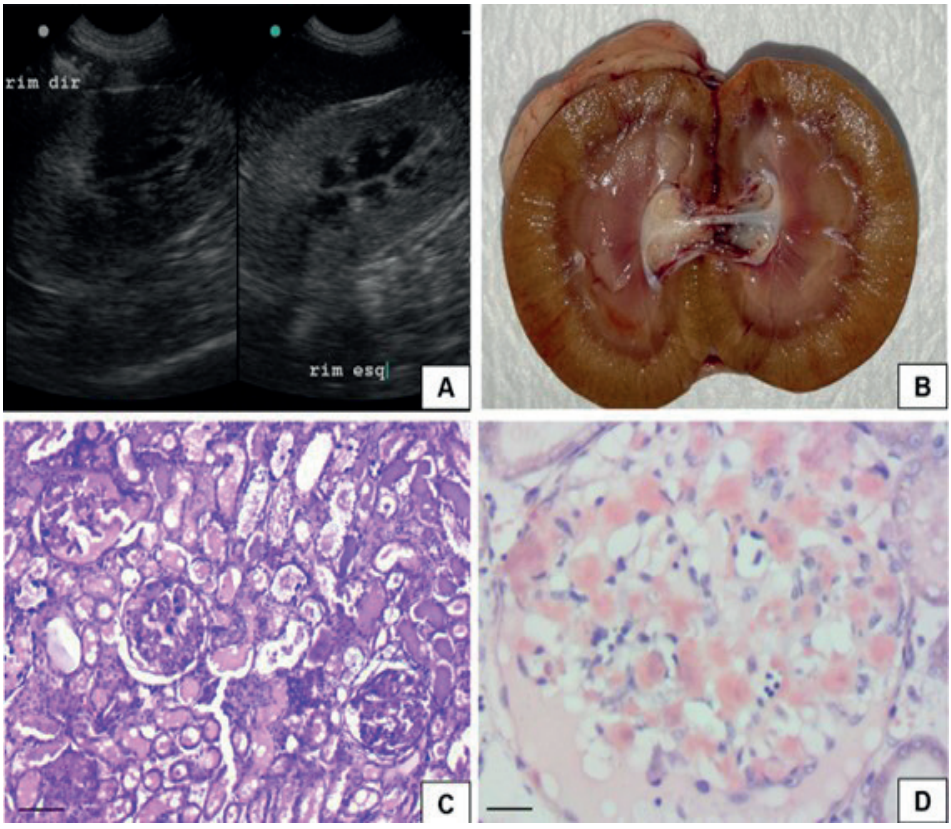

FIGURE 1. A: Ultrasound images of the right and left borders, showing changes in echogenicity. B: Left border, sectioned, for histopathological evaluation, showing mild nephromegaly, loss of the corticomedullary relationship, and dilation of the renal pelvis. C: Photomicrograph of the cortical region, showing the position of the amyloid protein within the glomeruli and tubules. ELE, 20x. D: Glomerulus stained with Congo red, showing the presence of amyloid protein. Congo red, 40x.

Source: own elaboration.

As the patient's clinical condition deteriorated and due to a delay in seeking veterinary care, she arrived in critical condition. Following discussions with the owner, euthanasia was elected. Necropsy was not authorized; however, permission was granted for the removal of the kidneys for diagnostic evaluation.

Fragments of the right and left kidneys, measuring approximately 6.8 × 4.7 × 4.4 cm and 7.2 × 4.0 × 5.0 cm, respectively, exhibited diffuse whitish striations in the cortex, mild dilation of the renal pelvis, and a tense-elastic consistency. These samples were preserved in 10% formalin and submitted to an outsourced laboratory for histopathological analysis.

Histological examination of hematoxylin-eosin-stained sections revealed enlarged glomeruli with extensive deposition of eosinophilic, homogeneous, and slightly fibrillar material. Congo red

staining confirmed amyloid deposition, affecting large areas of the glomeruli and interspersed with mineralized regions. Glomerulosclerosis was also observed. In the tubules, multifocal basophilic, fragmented material indicative of mineralization was noted, along with a marked accumulation of intratubular proteinaceous material, occasionally associated with mild dilation. The interstitial space exhibited a mild multifocal lymphoplasmacytic inflammatory infiltrate.

Based on these findings, a diagnosis of renal amyloidosis was confirmed (figure 1). The owner was informed of the diagnosis and advised against breeding animals from the same lineage, given the hereditary nature of the disease.

DISCUSSION

Renal amyloidosis, as previously described, is characterized by the accumulation of amyloid-type protein in tissues and organs, often in response to inflammatory processes (DiBartola *et al.* 1990; Sousa *et al.* 2024). Inflammatory conditions lead to increased production of amyloid proteins, resulting in their deposition through aggregation of isoforms in organs such as the kidneys (DiBartola *et al.* 1990; Johnson *et al.* 1996). Amyloid protein is insoluble, highly resistant to proteolytic degradation, and composed of fibrils derived from amyloidogenic proteins (Serakides & Silva 2023). The extent of amyloid protein deposition varies depending on factors such as clinical condition and protein composition (Woldemeskel *et al.* 2012). Deposition may involve heterogeneous protein types, including amyloid A (AA), light chain (AL), and familial forms (Serakides & Silva 2023; Giaretta & Barros 2023). The kidneys are among the most commonly affected organs, as demonstrated in this case. However, in this patient, the presence of amyloid deposits in other organs, such as the liver, was not assessed.

This disease is frequently reported in predisposed breeds, such as the Shar Pei, where it is primarily transmitted through familial inheritance, allowing affected individuals to pass the disease to subsequent generations. In the present case, there was a strong suspicion of familial amyloidosis, as the affected patient belonged to a predisposed breed and had direct familial ties to other dogs that had been diagnosed with renal amyloidosis. Previous studies have also reported familial amyloidosis in Shar Pei dogs (DiBartola *et al.* 1990; Lee *et al.* 2007). Given the hereditary nature of the disease, animals diagnosed with renal amyloidosis should be spayed or prevented from breeding, as they are potential carriers of the causative genetic variants (Sonne *et al.* 2008). In this case, the recommendation to neuter affected animals was not followed, as the patient's sire had also been diagnosed with renal amyloidosis, indirectly suggesting that breeding with a diseased or carrier animal had occurred. Spaying affected animals is essential to reduce the risk of genetic transmission, even in cases where they do not exhibit clinical symptoms (Sousa *et al.* 2024).

Owner education is critical in ensuring an understanding of the disease and its associated complications. Additionally, providing emotional support and guidance following the diagnosis of a progressive and life-threatening condition is essential (Sousa *et al.* 2024). Given the severe and irreversible nature of amyloidosis, this stage can be particularly challenging for owners, especially those with a strong

emotional attachment to their pets (Sousa *et al.* 2024). They must be informed that animals diagnosed with amyloidosis, whether clinically affected or identified as carriers, should not be used for breeding to prevent further transmission of the disease (Sousa *et al.* 2024).

In a study conducted on Shar Pei dogs, amyloid protein deposition in the glomerular region was detected in nearly 80% of the animals, highlighting the breed's strong predisposition to renal amyloidosis. However, the disease is not exclusive to Shar Pei dogs and has also been reported in breeds such as Beagle (Jung *et al.* 2014), Weimaraner (Loewen *et al.* 2018), and Italian Pointer (Inman *et al.* 2021). Regarding age-related susceptibility, glomerulopathies tend to manifest more frequently in middle-aged to older dogs (Vaden 2011), with amyloidosis typically reported in animals between four and eight years of age (Segev *et al.* 2012). However, some studies suggest a broader range of one to six years, with Shar Pei dogs potentially developing the disease at an earlier age (Sonne *et al.* 2008; Júnior *et al.* 2011; Loewen *et al.* 2018). In the present case, the patient was diagnosed at nine years of age, which aligns with available data (Vaden 2011). Nevertheless, although clinical signs manifested at this stage, amyloid deposition likely began earlier, with severe impairment of renal function occurring progressively over time. Other dogs from the same familial lineage were diagnosed at three and four years of age.

The clinical manifestations of amyloid deposition vary depending on factors such as proteinuria and the extent of renal dysfunction (Sousa *et al.* 2024). Renal amyloidosis is frequently associated with chronic kidney disease (CKD), as amyloid accumulation induces structural and functional damage in the glomeruli and tubules, impairing nephron function (Greco 2001; Woldemeskel *et al.* 2012). Consequently, the severity and extent of amyloid deposition are expected to correlate with the progression of clinical symptoms due to structural and functional damage in affected renal segments (Greco 2001). Amyloid deposition in the kidneys is directly associated with the frequency of clinical signs, which are commonly indicative of CKD or acute kidney injury, leading to significant organ dysfunction (Sousa *et al.* 2024).

As a result, dogs with renal amyloidosis typically exhibit progressive signs of CKD. The clinical presentation of CKD is often nonspecific in the early stages but becomes more pronounced as the disease advances. Affected dogs may exhibit lethargy, inappetence, vomiting, diarrhea, proteinuria, azotemia, impaired urine concentration and metabolite excretion, and ultrasonographic abnormalities indicative of renal dysfunction (Perondi *et al.* 2020). In the present case, the patient was evaluated due to clinical signs including vomiting, halitosis, hyporexia, polyuria, and polydipsia, which align with descriptions from previous studies on amyloidosis (DiBartola *et al.* 1990; Sonne *et al.* 2008; Júnior *et al.* 2011; Loewen *et al.* 2018; Inman *et al.* 2021). Additionally, the owner reported that other dogs from the same familial generation had exhibited clinical signs suggestive of CKD.

The laboratory characterization of patients with renal amyloidosis is highly variable and closely linked to the severity and progression of chronic kidney disease (CKD) (Sousa *et al.* 2024). Evidence suggests that affected patients may exhibit hematological and serum biochemical alterations, with the most pronounced

findings including azotemia, proteinuria, electrolyte imbalances, and changes in urine specific gravity and the urine protein-to-creatinine ratio, among others (Lee *et al.* 2007; Sonne *et al.* 2008; Júnior *et al.* 2011; Loewen *et al.* 2018; Inman *et al.* 2021; Sousa *et al.* 2024). In this case, the patient exhibited proteinuria (2+), a urine pH of 5, and a specific gravity of 1.018. Patients with amyloidosis typically present with reduced urine specific gravity, approximately 1.015, along with proteinuria (generally exceeding 3+), findings that align with those observed in this case (Lee *et al.* 2007; Segev *et al.* 2012).

Renal amyloidosis is commonly associated with alterations in hematological and serum biochemistry parameters, particularly urea and creatinine levels, hypoalbuminemia, hypoproteinemia, and hyperphosphatemia (Lee *et al.* 2007; Sonne *et al.* 2008; Segev *et al.* 2012; Júnior *et al.* 2011; Loewen *et al.* 2018; Inman *et al.* 2021). In the discussed case, the patient exhibited leukocytosis due to neutrophilia without a shift, significant azotemia (urea: 201.80 mg/dL; creatinine: 6.04 mg/dL), a urea/creatinine ratio of 33.41 mg/dL, hyperphosphatemia, hypoalbuminemia, and hypoproteinemia, findings consistent with those reported in previous studies (Lee *et al.* 2007; Sonne *et al.* 2008; Segev *et al.* 2012; Júnior *et al.* 2011; Loewen *et al.* 2018; Inman *et al.* 2021; Sousa *et al.* 2024). Azotemia is frequently associated with glomerular disorders, occurring in approximately 90% of affected animals, with an average creatinine level of 5.5 mg/dL (Segev *et al.* 2012). Notably in this case, the patient's creatinine level exceeded the values reported in the literature (Segev *et al.* 2012).

In cases involving glomerular pathology, imaging abnormalities may be detected, particularly via abdominal ultrasonography. Chronic glomerular diseases, including amyloidosis, often present with ultrasonographic findings characteristic of CKD, such as irregular renal contours and size, altered cortico-medullary junction and differentiation, cortical and medullary hyper echogenicity, renal infarction, pyelectasia, and mineralization (Perondi *et al.* 2020). According to the International Renal Interest Society (IRIS), ultrasonographic abnormalities in CKD patients are stage-dependent, with the severity of findings increasing in proportion to disease progression (IRIS 2023). In this case, the patient exhibited ultrasonographic evidence of CKD, including increased cortical echogenicity and decreased cortico-medullary differentiation, consistent with previous reports (Perondi *et al.* 2020; Inman *et al.* 2021; Sousa *et al.* 2024).

Although renal amyloidosis is associated with non-specific pathological changes and commonly presents as part of CKD, histological evaluation of renal tissue remains the gold standard for definitive diagnosis. This approach enables the identification of amyloid deposits through specialized staining techniques. Diagnosis is based on the detection of eosinophilic material within glomerular and/or tubular structures, the presence of lymphocyte and plasma cell infiltrates, fibrosis, and, in some cases, protein casts (Sonne *et al.* 2008). Amyloid deposition may be focal or widespread, with the potential for severe systemic involvement (Segev *et al.* 2012). In the present case, histopathological analysis revealed eosinophilic, fibrillar material within the glomerular and tubular regions, as well as calcification and inflammatory infiltrates, findings consistent with those reported in previous studies (DiBartola *et al.* 1990; Lee *et al.* 2007; Sonne *et al.*

2012; Segev *et al.* 2012; Sousa *et al.* 2024). However, investigations for extra-renal amyloidosis and amyloid protein typing were not conducted due to constraints imposed by the patient's guardians.

The clinical presentation of renal amyloidosis was initially suspected based on a combination of clinical findings, the patient's familial history, and previous diagnoses in related individuals. Despite this suspicion, the severity of the disease led to an unfavorable prognosis. Therapeutic management of renal amyloidosis remains challenging, as no effective treatment strategies are currently available to significantly improve disease outcomes or prolong survival (Sousa *et al.* 2024).

CONCLUSION

Renal amyloidosis is a progressive and severe disease often associated with poor clinical outcomes. Characterized by the deposition of amyloid protein, the condition has a strong familial component, particularly in Shar Pei dogs, though it is not exclusive to this breed. In the discussed case, amyloid deposition in renal tissue was confirmed in a Shar Pei dog, a breed predisposed to this condition. Diagnosis was established through a combination of clinical assessment, imaging (ultrasonography), laboratory findings, and histopathological evaluation, supported by specialized staining techniques. The familial nature of renal amyloidosis in this case is strongly suspected, given the history of the disease in closely related dogs (sire and sibling). Future investigations will include immunoperoxidase staining for amyloid AA, kappa, and lambda to further characterize the protein deposits. It is crucial that owners are made aware of the risks of including affected animals in breeding programs, as the disease can be transmitted to subsequent generations.

CONFLICT OF INTEREST

The authors declare no conflicts of interest.

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USE OF ARTIFICIAL INTELLIGENCE

No artificial intelligence was utilized in the diagnosis, treatment, or preparation of this manuscript.

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