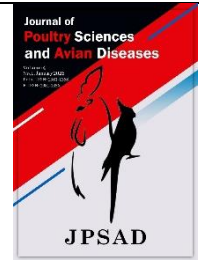


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## Systemic Amyloidosis in Saker Falcon (*Falco cherrug*): A Case Report



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### ABSTRACT

The Saker Falcon (*Falco cherrug*), an endangered species, faces threats from diseases associated with captivity, such as amyloidosis. This study reports the first documented case of systemic AA amyloidosis in a captive Saker Falcon in Iran, linked to chronic pododermatitis (bumblefoot) and visceral gout. A female falcon with a low body score, anorexia, bilateral foot lesions (Grade II bumblefoot), and paralysis died during transport to the veterinary clinic. Postmortem analysis showed urate deposits on the pericardium and gizzard, along with amyloid accumulation in the liver, confirmed by Congo red staining. Histopathology identified eosinophilic hyaline material between hepatocytes and inflammatory infiltrates in the gizzard and heart. Chronic inflammation from bumblefoot and metabolic disturbances from visceral gout were implicated as triggers for amyloidosis, consistent with prior studies linking AA amyloidosis in birds to prolonged inflammation, stress, and poor captive conditions. The liver was the main site of amyloid buildup, suggesting liver failure as a likely cause of death, contrasting with mammals, where the kidneys are usually affected.

**Keywords:** Saker Falcon (*Falco cherrug*), Amyloidosis, Bumblefoot, Visceral gout

### 1 Introduction

The Saker Falcon (*Falco cherrug*) is classified as Endangered due to threats from human activities, particularly the international trade of this species (1). Amyloidosis is the accumulation of soluble proteins in

different organs, classified as localized or systemic. Localized amyloidosis occurs in specific organs, whereas systemic amyloidosis involves amyloid fibrils formed from blood proteins that deposit in multiple organs, especially the visceral organs (2, 3). Avian amyloidosis was first reported in pheasants (4), but it can now affect a wide variety of birds,

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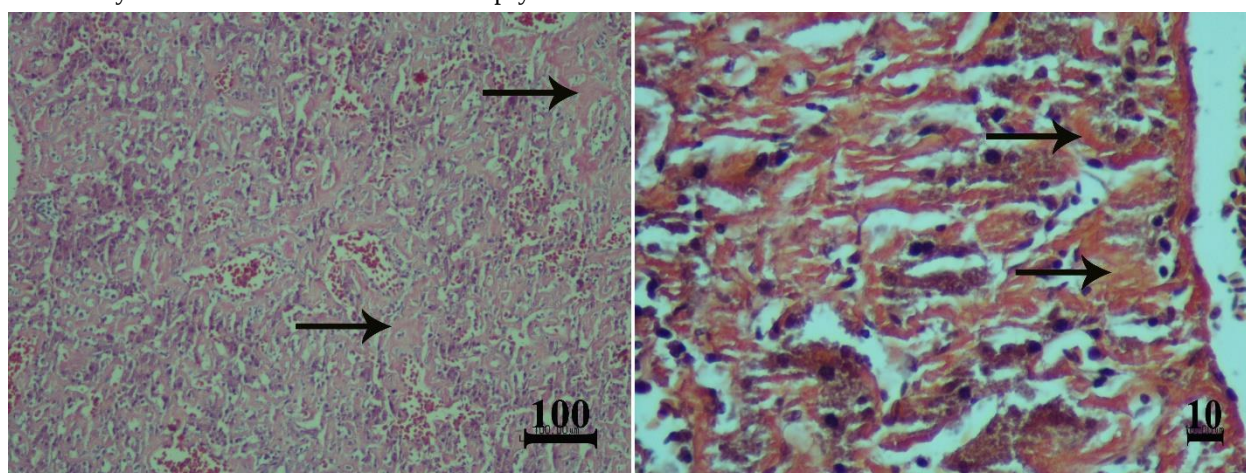
including waterfowl (e.g., swans, geese, and ducks), poultry, and caged wild birds (e.g., doves, finches, and parrots) (5, 6) and limited reports of amyloidosis involvement in birds of prey (7). Chronic diseases, such as aspergillosis, bumblefoot, and tuberculosis, often stimulate amyloid deposition. Nutritional factors may play a significant role, particularly in those who have changed their diet. Captivity and overcrowding are notable risk factors for developing this disease (8). Other risk factors include neoplasia, trauma, infections, inflammatory conditions, and stress. AA amyloidosis can also occur without identifiable causes and may have a genetic predisposition (3, 9, 10). In the current report, we diagnosed amyloidosis using postmortem Congo red stains. Due to the authors' knowledge, this report represents the first documented case of amyloidosis associated with bumble foot and visceral gout in a captive raptor in Iran.

## 2 Case Description

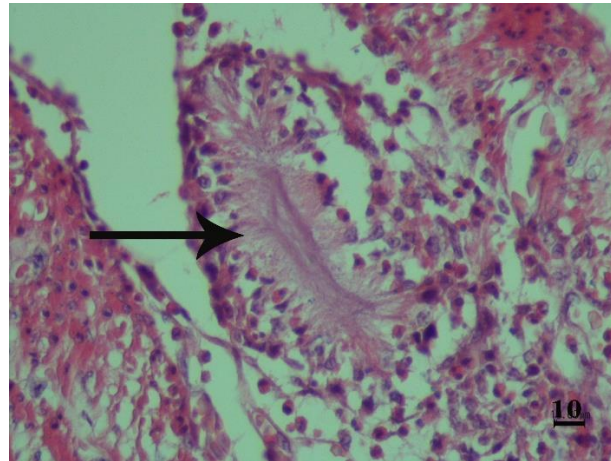
An adult female Saker falcon (*Falco cherrug*) had been housed at the Environmental Rehabilitation Center for six months. Upon her admission to the center, the bird showed no obvious clinical signs. However, after approximately five months, she began to exhibit emaciation, anorexia, bilateral foot inflammation, and the onset of paralysis. The leg paralysis developed gradually as a consequence of bumblefoot. At the time of referral, the bird exhibited clinical signs of a body score under 3, anorexia, bilateral pododermatitis, and paralysis. During its captivity, it was fed chicken meat. The bird succumbed while being transported to the veterinary school for treatment. The necropsy was

performed in the veterinary clinic. Tissue samples such as heart, liver, kidney, spleen, proventriculus, gizzard, duodenum, jejunum, rectum, and pancreas were submitted to the veterinary clinic of the Kerman University for histopathological examination and diagnosis. After the necropsy, tissue samples were collected and fixed in 10% neutral buffered formalin. They were then trimmed and embedded in paraffin wax. The paraffin blocks were sectioned at a thickness of 3  $\mu$ m and stained with hematoxylin and eosin (H&E) for examination under a light microscope.

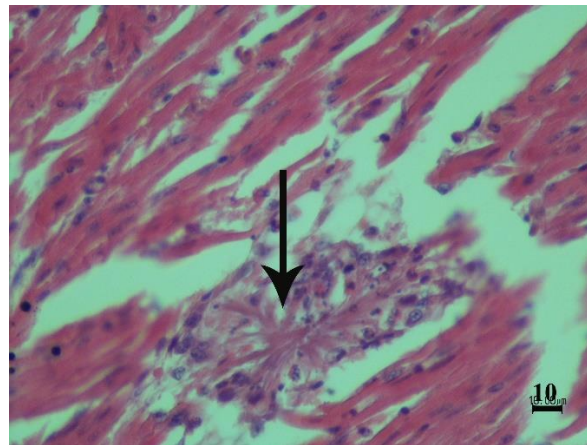
Additionally, special staining, including Congo red stain, was performed on the paraffin-embedded tissue sections from the liver. Gross findings: swelling and ulceration were observed on the central surface of both feet. According to the degree of bumblefoot classification, the foot lesion was classified as Grade II (11). Except for a minor deposition of uric acid observed on the pericardium of the heart, no additional macroscopic signs were noted. Microscopic findings: In the liver, there was a notable deposition of homogeneous eosinophilic hyaline material observed between the hepatocytes. The diagnosis of amyloidosis was confirmed through Congo red staining due to the orange color of these deposits (Fig. 1). In the gizzard, destruction of the muscular layer occurred with infiltration of eosinophils and heterophils among mononuclear inflammatory cells. In some areas, foci of urate crystals were observed, surrounded by infiltration of these inflammatory cells (Fig. 2). Within the heart, there were notable deposits of urate crystals, accompanied by the presence of inflammatory cell infiltration (Fig. 3).



**Figure 1.** Liver; left: amyloid deposition, homogenous eosinophilic material, and atrophic hepatocytes. H & E stain; scale bar, 100  $\mu$ m. Right: Amyloid deposits are stained orange in Congo red staining. Congo red stain; scale bar, 10  $\mu$ m.



**Figure 2.** Gizzard; urate crystal formation arrow in the muscular layer surrounded by mononuclear, eosinophils, and heterophils infiltration. H & E stain; scale bar, 10  $\mu$ m.



**Figure 3.** Heart; urate crystal formation (arrow) in the muscular layer surrounded by mononuclear, eosinophils, and heterophils infiltration. H & E stain; scale bar, 10  $\mu$ m.

### 3 Discussion and Conclusion

To date, most amyloidosis reported in wild and domestic captive birds has been systemic AA-amyloidosis (9). The development of amyloidosis AA in animals is linked to a chronic, long-term infectious or inflammatory process. The incidence of this condition tends to rise with age, likely due to the cumulative effects of ongoing diseases. Furthermore, amyloidosis is often more prevalent in species that are not well-suited to the stress of zoo or farm environments (4, 12-14). Specific clinical signs of systemic amyloidosis have not been observed in wild or domestic captive birds. Since diagnosis is typically made through postmortem autopsy, it is challenging to assess the clinical signs of systemic amyloidosis in birds (8). Avian AA amyloidosis is observed in birds as a consequence of various inflammatory disorders, including tuberculosis, aspergillosis, bumblefoot,

arteriosclerosis, and its associated complications, chronic wound inflammation related to accidental amputation, and chronic peritonitis caused by infection with Gram-negative bacilli (15).

Amyloidosis AA in falcons is associated with inflammatory conditions and chronic infections. Previous studies examining various diseases and inflammatory factors in falcons have demonstrated a significant increase in cases of visceral gout and bumblefoot among those with amyloidosis (7).

Bumblefoot, or pododermatitis, is an inflammatory condition affecting the feet of birds. It may involve both inflammatory lesions and degenerative changes, often resulting from avascular necrosis. Furthermore, secondary amyloidosis related to the inflammatory response observed in pododermatitis has been documented in captive whooper swans in Korea (15, 16).

Visceral gout is a metabolic disorder characterized by the deposition of urates on serosal surfaces. In avian species, common sites for these deposits include the epicardium, pericardium, and the serosal surfaces of the proventriculus, ventriculus, and liver. Morphologically, these deposits present as gray-white in color and exhibit variable shapes. Histopathological examination frequently reveals that many cases are peracute; however, heterophilic infiltrates may be observed in conjunction with the deposits (17).

Different species can involve various organs in avian amyloidosis, with the liver, spleen, kidneys, and small intestines being the most frequently affected organs(17). A study investigating the complications of amyloidosis in falcons has demonstrated that the liver is the organ most significantly impacted by this disease. The pathogen responsible for amyloidosis tends to localize within the liver at an earlier stage and in greater concentrations compared to other organs. Consequently, the liver is identified as the primary target of amyloidosis in falcons (7). In contrast to patterns observed in amyloidosis among humans and other mammals, glomeruli do not serve as the primary target of amyloidosis in falcons. It can be assumed that most falcons die from liver failure (18).

According to both past research and the present study, timely diagnosis and treatment of conditions such as bumblefoot and visceral gout, along with the avoidance of abrupt dietary changes and the reduction of environmental stressors, appear to contribute significantly to the prevention of amyloidosis in falcons.

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## Conflict of Interest

The authors declare no competing interests.

## Author Contributions

H.Sh., contributed to the writing of the original draft, conceptualization, methodology, and review and editing of the manuscript. R.Kh., M.S., and S.h.A., contributed to the development of the study methodology.

## Data Availability Statement

The data used to support the findings of this study are available from the corresponding author upon reasonable request.

## Ethical Considerations

In conducting this study, ethical principles have been fully observed.

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