Veterinary Research Forum
Vol: 2, No: 3, September, 2011, 209 - 213

Case Report

Systemic Amyloidosis and Testicular Interstitial Tumor in a Zebra Finch *(Taeniopygia guttata)*: a Case Report in Iran

Mohsen Nouri [1*]
Farhang Sasani[1]
Mohammad Javad Gharagozloo[1]
Mehrnoush Moeini Jazani[1]

[1]Department of Pathology, Faculty of Veterinary Medicine, University of Tehran, Tehran, Iran

Received: 14 April 2011, Accepted: 30 July 2011

Abstract

Systemic amyloidosis and testicular interstitial tumor are rare conditions in birds and this is the first report in Iran. A male zebra finch was presented because of white diarrhea, anorexia, loss of weight and lethargy. At necropsy, the small intestine was edematous and congested. The spleen appeared pale. The liver was large, firm and brown. One testis was cystic and neoplastic and the remaining testis was atrophic.

Histologically, amyloid materials were seen predominantly in the liver and spleen. Hyaline substances were deposited in the Disse space and in the media of blood vessels of the liver. In spleen, marked deposits thickened the basement membranes of blood vessels and extended into the surrounding parenchyma. In addition, there were lesser degrees of amyloidosis in other organs such as small intestine. Amyloid stained positively with Congo red.

In testis, there was encapsulated unilateral interstitial cell tumor, with multiple foci of necrosis and hemorrhage. The neoplastic cells were round to polyhedral, with small round hyperchromatic nuclei and finely vacuolated cytoplasm. Signs of feminization were observed. The cause of amyloidosis in this study was not conclusively identified.

Key words: Systemic amyloidosis, Finch, Interstitial cell tumor, Leydig cell, Testis

[*]Corresponding author:
Mohsen Nouri, DVM
Department of Pathology, Faculty of Veterinary Medicine, University of Tehran, Tehran, Iran.
E-mail address: mnouri2@yahoo.com

Introduction

Amyloidosis is a disorder that is characterized by extracellular deposition of proteinaceous material between cells in various tissues and organs of the body. Amyloidosis is generally divided into primary and secondary amyloidosis.[1-4] The former (AL) occurs as a consequence of various forms of plasma cell dyscrasias, such as multiple myeloma, and other monoclonal proliferations of B lymphocytes. The latter occurs in association with chronic inflammatory diseases, such as tuberculosis and long-standing suppurative process.[1] Amyloid A (AA) protein, is derived from serum. AA protein synthesized by the liver; Only amyloid A (AA) has been detected in birds.[2,5-7] Amyloidosis is a rare disease, especially in birds except waterfowls.[8-11] There are a few reports of natural amyloidosis in cage birds. [3,12,13]

Testicular tumors are uncommon. Interstitial cell tumors (Leydig cell tumor) are derived from the endocrine cells of the testicular interstitium. They are grouped with Sertoli cell tumors in being derived from tissue of the sex cords or stroma. These neoplasms are almost always benign. They likely begin as regions of nodular hyperplasia. Interstitial cell tumors, which can be cystic and hemorrhagic, are rare, and their behavior is unreported[3,14] and only a few cases have been reported in birds. [15, 16]

There is no published report on systemic amyloidosis and testicular interstitial tumor in finch to the authors' knowledge. In this paper, the behavioral and pathologic characteristics of a systemic amyloidosis and testicular interstitial tumor are described in a zebra finch.

Case Description

In December 2009, a male zebra finch was presented to a private veterinary practitioner with a history of white diarrhea, anorexia, loss of weight, lethargy and unilateral abnormal weight bearing. This case showed feminization and bill color was changed from dark red to orange.

The bird died shortly after presentation and a complete necropsy was performed by the veterinarian.

Gross necropsy revealed edema and congestion on the intestine. The spleen appeared pale and the liver was large, firm and brown. The testes were asymmetric. One testis had an irregular surface, was cystic, soft and orange-white, and measured 3 × 5 mm. The remaining testis was atrophic, and measured only 1 × 2 mm.

Fresh wet smear samples and imprint smear samples from the intestine were stained by the gram methods, and examined microscopically. Many gram-negative bacilli were seen. *Salmonella* were isolated in pure culture. Collected organs were taken in 7.2 % buffered formalin and were sent to the pathology laboratory in Tehran for histopathologic examination. At the laboratory, thin histologic tissue sections were prepared and stained by hematoxylin and eosin (H&E), and Congo red methods.

On microscopic examination, there was generalized deposition of amyloid, appearing as eosinophilic amorphous substances. The degree of amyloid deposition was remarkable in the liver and spleen. Hyaline substances were deposited in the Disse space and in the media of blood vessels. The amyloid effaced much of the hepatic parenchyma (Fig. 1). The hepatic cells showed pressure atrophy by amyloid deposits. The hyaline substances stained positively with Congo red (Fig. 2). In spleen, marked deposits thickened the basement membranes of blood vessels and accumulate around the periarterial sheaths and extended into the surrounding parenchyma (Fig. 3). There was the mild degree of amyloid deposition in the basement membranes of the small intestine. In testes, there were encapsulated unilateral interstitial cell

tumors, with multiple foci of necrosis and hemorrhage. The neoplastic cells were round to polyhedral and large, with small round hyperchromatic nuclei and occasional fine or large vacuolated cytoplasm. The neoplastic cells formed trabeculae and sheets. Mitotic figures were occasionally seen (Fig. 4).

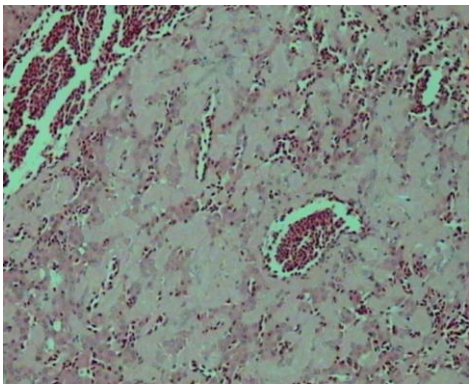

Fig 1. Severe hepatic amyloidosis and congestion; Compression of hepatocytic cords by the accumulation of homogenous material in the diss's space. H&E stains **(**40×**)**

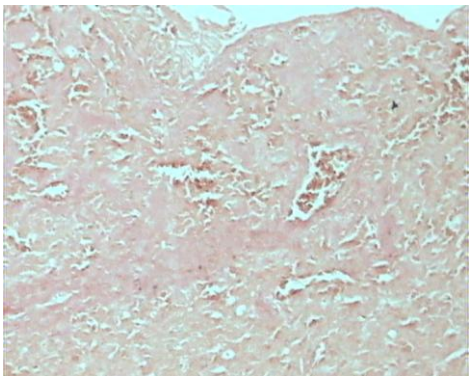

Fig 2. The hyaline substances stained positively with Congo red in liver. Congo red stain. (10×**)**

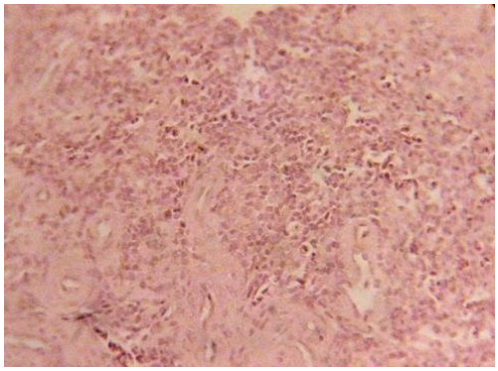

Fig 3. Marked deposits thickened the basement membranes of blood vessels and accumulate around the periarterial sheaths and extended into the surrounding parenchyma in spleen. H&E stains. (40×**)**

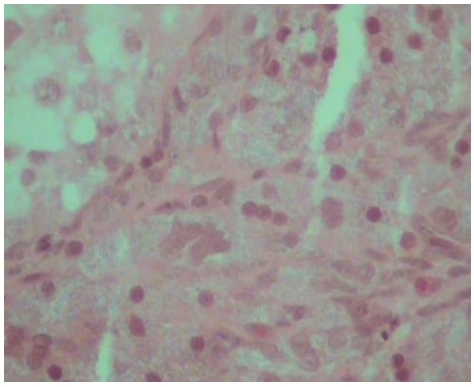

Fig 4. Tubules are effaced by a sheet of neoplastic cells with abundant cytoplasm in testis. The nuclei are regular in size and staining affinity, and mitoses are rare. H&E stains. (100×)

Discussion

Amyloidosis is the general term applied to several diseases characterized by deposition of one of several forms of amyloid.[3] To date in caged wild and domestic birds only systemic AA-amyloidosis has been described, although theoretically other non recognized forms might exist. [5-7,13] AA amyloidosis occurs secondarily from excess SAA produced as a result of chronic antigen stimulation. Inflammation and various stresses induce cytokines (IL-6), the IL-6 stimulates the hepatic cells, and hepatic cells liberate SAA as one of the acute phase proteins. [2,17,18] A few reports of amyloidosis have been made in chickens, although amyloidosis is a well recognized pathological disorder in water birds, such as Anatidae, and swans. [8-11] However, we recently encountered incidental amyloidosis in finch.

No specific clinical symptoms have been associated so far with systemic AA-amyloidosis in caged wild birds and domestic poultry, while the diagnosis is generally performed at postmortem.[2,7,19] The distribution of organ involvement varies from bird to bird. Multiple organs are generally affected in most species; Liver, spleen, kidneys and small intestine are most frequently and severely involved. Less affected are proventriculus, large intestine, heart, gonads and endocrine organs, while rarely affected are brain,

lungs and skin.[2] In this case, the tissue distribution of amyloid was similar to that reported previously in birds. Microscopically, there was generalized deposition of amyloid, appearing as eosinophilic amorphous substances. The degree of amyloid deposition was remarkable in the liver and spleen. Amyloidosis is less common in psittacine birds but, when it does occur, generally involves both the spleen and the kidney.[3] In this case; amyloid deposition was more in the liver and spleen.

Amyloid is differentiated from other acellular eosinophilic material, such as fibrin and immune complex deposition, by positive staining and birefringence with Congo red stain. Avian amyloid, however, does not consistently stain well with Congo red. [2,3]

The predisposing causes in birds were suggested breed, genetic, chronic bacterial infections, inflammatory disease, aging, seasonal variations, social needs, operation, neoplastic diseases, toxicosis, poorly adapted to zoo, farm or home conditions, over-crowded environment and stocking with compatible species. [1-3,6,7,9,10,12,19-21] Thus, adequate management not only focusing on hygiene, but also on stress avoidance is important tools in preventing reactive amyloidosis.

Salmonella infections were noted in this study. *Salmonella* was isolated from the intestine. Amyloidosis can occur before *Salmonella* infections in finch because amyloidosis is a more chronic change than *Salmonellosis*. However, secondary *Salmonella* infections can also accelerate the severity of amyloidosis. Amyloidosis depresses finch immunity; therefore, it might be a factor in the induction of *Salmonella* infection.

Testicular tumors are rare and only a few cases have been reported. [15,16] The histological description of the tumor in the finch was similar to that described in budgerigar. Based on histologic examination, much of testicular parenchyma had been replaced by neoplastic tissue. The clinical signs of testicular cancer such as chronic weight loss and unilateral paresis were similar to that reported previously in birds.[14,22] In budgerigars, interstitial cell tumors cause clinical signs similar to a renal tumor and attributable to a space-occupying mass.[14] But in the present case, there was not any sign of abdominal swelling or a space-occupying mass. Signs of feminization were observed in the case presented here. Both testes were not functional in this adult male bird. Leydig cell are responsible for the production of steroid hormones (primarily testosterone). As in mammals, testosterone must be converted to estraiol in the central nervous system for expression of male sexual behavior. [22]

The cause of amyloidosis in this study was not conclusively identified. This finch might be stressed from the mentioned causes like in other birds. Further study on both internal and external factors is needed to clarify the pathogenesis of testicular interstitial tumor in birds.

Acknowledgments

We are grateful to the owner of finch for allowing us to handle it. Mr. Mirskandari, the Chief Technician in the Baharan Pathology Laboratory, is acknowledged for histologic assistance.

References

1. King N. W, and J Alroy, Amyloidosis. In T. C. Jones, R. D. Hunt, and N. W. King, (eds.): Veterinary pathology, 6th ed. Williams & Wilkins, Baltimore, Philadelphia, London, Paris, Bankok, Buenos Aires, Hong Kong, Munich, Sydney, Tokyo, and Wroclaw. 1996; 50–55
2. Landman W.J.M., Gruys E, Gielkens A.L.J. Avian amyloidosis. Avian Pathol. 1998;27: 437-449.
3. Schmidt RE, Reavill DR, Phalen DN. Pathology of pet and aviary bird, Blackwell Publishing. 2003; 14, 84

4. Snyder PW. Diseases of immunity. In: MD McGavin and JF Zachary, (Eds): Pathologic Basis of Veterinary Disease, 4th Edit. Mosby, St. Louis. 2007; 193-251
5. Zschiesche, W. & Linke, R.P. Immunohistochemical characterization of spontaneous amyloidosis in captive birds as AA-type, using monoclonal and polyclonal anti-AA antibodies against mammalian amyloid. Acta Histo, 1989; 86: 45-50.
6. Nakamura K., Tanaka H., Kodama Y., Kubo M. and Shibahara T., Systemic amyloidosis in laying japanese quail, Avian Dis 1998;42:209-214.
7. Gelis S., The Gouldian Finch (Erythrura gouldiae) in Health and Disease, Sem Avian Exot Pet Med, 2003; 12(4): 215-227
8. Cowan, D. F., Avian amyloidosis. II. Incidence and contributing factors in the family Anatidae. Pathol Vet 1968b; 5: 59–66.
9. Sato, A., T. Koga, M. Inoue, and N. Goto. Pathological observations of amyloidosis in swans and other Anatidae. Jpn. J. Vet. Sci. 1981; 43:509–519.
10. Zschiesche, W. and Jakob, W. Pathology of animal amyloidosis. Pharma and Therap, 1989; 41: 49-83.
11. Hatai H., Ochiai K., Nakamura S., Kamiya T., Ito M., Yamamoto H., Sunden Y. and Umemura T., Hepatic Myelolipoma and Amyloidosis with Osseous Metaplasia in a Swan Goose (Anser cygnoides), J Comp Pathol, 2009; 141, 260-264.
12. Cowan, D. F., Avian amyloidosis, I. General incidence in zoo birds. Pathol Vet 1968a; 5:51–58.
13. Landman, W.J.M. and Grays, E., Amyloid arthropathy in an Indian peafowl. Vet Rec, 1998; 142: 90-91.
14. Reavill D. R., Tumors of pet birds, Vet Clin Exot Anim, 2004; 7: 537–560
15. Beach J.E., Diseases of budgerigars and other cage birds; A survey of post-mortem findings. Vet Rec 1962; 74: 10-14, 63-68,134-140.
16. Petrak M.L., Gilmore C.E. Neoplasms, In: Petrak ML (ed.): Diseases of Cage and Aviary Birds, Lea & Febiger, Philadelphia. 1982; 459-489.
17. Stone, M., Amyloidosis: a final common pathway for protein deposition in tissues. Blood 1990; 75:531- 545.
18. Alsemgeest, S.P., Lambooy, I.E., Wierenga, H.K., Dieleman, S.J., Meerkerk, B., Van Ederen, A.M. & Niewold, T.A., Influence of physical stress on the plasma concentration of serum amyloid-A (SAA) and haptoglobin (Hp) in calves. Vet Quart, 1995; 17: 9-12.
19. Crespo R. and Shivaprasad H.L.: Developmental, Metabolic and other noninfectious Disorders, In Saif Y.M., Fadly A.M., Glisson, J.R., McDougald L.R., Nelan L.K., Swayne D.E. (Eds.): Disease of poultry, 12th edi, Blackwell publishing. 2008:1152-4
20. Karstad, L. & Sileo, L., Causes of death in captive wild waterfowl in the Kortright waterfowl park: 1967-1970. J Wildl Dis, 1971; 7: 236-241.
21. Echols M.S., (2002) Surgery of the Avian Reproductive Tract, Seminars in Avian and Exotic Pet Medicine, 2002; 11:177-195
21. Zekarias, B., W. J. M. Landman, P. C. J. Tooten, and E. Gruys. Leukocyte responses in two breeds of layer chicken that differ in susceptibility to induce amyloid arthropathy. Vet. Immunol Immunop 2000; 77:55–69.
22. Ottinger MA., Bakst MR., Endocinology of the avian reproductive system. J Avian Med Surg 1995; 9:242-250