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Amyloid hepatopathy in a Asian Palm Civet (*Paradoxurus hermaphroditus*)

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Abstract

Wildlife SOS runs wildlife rescue helpline in and around Agra city where the team usually gets call from public for any wildlife rescue. Likewise an injured male Palm civet was rescued and found severe injuries all over the body, so the same was brought to our wildlife hospital for treatment. The animal was highly stressed and had multiple bite marks all over the body. The radiographic examination revealed fracture of maxilla and other bones were intact. The wounds were cleaned and dressed. Necessary antibiotic and anti-inflammatories were administered. We planned the surgery to fix the fracture after stabilizing the health condition. But the animal started showing convulsion and died on second day. Since the animal had multiple bite mark on the body we wanted to rule out rabies. The Post-mortem examination revealed severe internal haemorrhage and contusion. Kidney and spleen were congested. Liver congested and discolored with mosaic appearance, gall bladder is filled with bile. All representative tissue samples were collected from the visceral organ for histopathological examination. However no marked abnormalities noticed in brain macroscopically, sample collected for detail histopathological examination to rule out rabies. The histopathological examination of brain tissue not revealed any negri body thus confirmed no Rabies. But the histopathology examination of liver under hematoxylin and eosin (H&E) staining revealed amyloid deposition in the interstitium thus suggesting amyloidosis. Hence the condition confirmed as amyloidosis induced hepatopathy.

Keywords: Amyloid hepatopathy, Asian Palm Civet, *Paradoxurus hermaphroditus*

1. Introduction

According to IUCN Red List status and Indian Wildlife (Protection) Act, 1972 Common Palm Civets are placed in Lower risk and Schedule II respectively. They are mainly frugivorous, but also eat small vertebrates and invertebrates. They are solitary, nocturnal, and largely arboreal, spending the day in trees and sometimes in buildings. The civet produces a musk (also called civet) highly valued as a fragrance and stabilizing agent for perfume. Both male and female civets produce the strong-smelling secretion, which is produced by the civet's perineal glands. It is harvested by either killing the animal or removing the glands, or by scraping the secretions from the glands of a live animal. The latter is the preferred method today. Common Palm Civets occur in a range of habitats up to 2400 m, including evergreen and deciduous forests (both primary and secondary), plantations, and around human dwellings and settlements [12]. Amyloid is a pathologic proteinaceous substance deposited between cells in various tissue and organs of the body in a wide variety of clinical setting¹. Over 20 different precursor proteins have been identified in the various forms of amyloidosis. However amyloidosis is classified based on the difference in the nature of the precursor protein, all amyloidoses have similar homogeneous eosinophilic histologic appearance when stained with HE and share affinity for certain histologic stains such as Congo red [34]. Amyloidosis is reported in horse [9, 10, 16, 20], cow [15, 32], sheep [8, 17], goats [23], dogs [2, 18], felines [3, 34, 24], birds [6, 19, 29] and macaques [37] by different authors but such report is not available in Palm civets. AA-amyloidosis is the most common type of amyloidosis in mammals including domestic animals and birds and often results in hepatic or renal failure due to physical disruption of the normal cellular and organ processes [34]. In this article we documented a case of amyloid induced hepatopathy in a Common Palm Civet.

2. Materials and Methods

A 3 kg male common palm civet rescued from Agra was brought to wildlife hospital at Agra Bear Rescue Facility, Uttar Pradesh, India (27°0'N;77°45'E). The general examination revealed multiple bite mark on the body of the animal (Figure 1) and the oral cavity examination revealed severe injury on the upper palate. The Radiographic examination of the animal confirmed longitudinal fracture of maxillary bone, and other bones were remaining intact (Figure 2). The wounds were cleaned and dressed with antiseptic ointment, the oral cavity rinsed with chlorhexidine mouth wash solution. Anti biotic and anti inflammatory injection were given and kept the animal under observation for further treatment and care and planed for the surgery to fix the fractured upper palate once the health condition stabilized. But on second day morning the animal started showing convulsion and died. The Post-mortem examination revealed severe internal haemorrhage and contusion. Kidney and spleen were congested (figure 5). Liver congested and discolour with mosaic appearance, gall bladder is filled (figure 4). All representative tissue samples were collected from the visceral organ for histopathological examination in 10% formalin. However no marked abnormality noticed macroscopically in brain, sample from brain tissue were also collected for detail histopathological examination to rule out rabies at Indian Veterinary Research Institute, Izatnagar, Bareilly, India.

3. Result and discussion

The detail histopathology examination of brain tissue samples not revealed any negri body thus confirmed no Rabies. But the histopathology examination of liver section under HE stain suggested amyloid deposition in the interstitium and confirmed amyloidosis (figure 6). Other organs got no microscopic lesion were evident.

Several different pathologic mechanisms and conditions underlie various forms and types of amyloidosis although abnormal proteins with similar staining characteristics are deposited in various organs and tissues of the affected animals [37]. Kidney is the main target organ for the deposition of amyloid in familial amyloidosis of the Abyssinian cat and Shar-Pei dogs, while the amyloid is mainly deposited in the liver in Siamese cats [4, 26]. Deposition of amyloid in the pancreas of cats, non human primates and humans can lead to the development of type 2 diabetes mellitus [31, 11]. The affected organs are often enlarged, moderately firm, and abnormally discoloured [31]. In AA-amyloidosis, the deposition in most species is in the central organs and tissues such as spleen, liver, kidney and the arterial walls [14, 22]. Depending on the extent of the deposition, there may be splenomegaly, Hepatomegaly, and renomegaly as spleen, liver and kidneys are the most commonly affected organs in systemic AA-amyloidosis [37]. In animals, at least eight different amyloid precursors have been described [23]. The precursor proteins in amyloid fibrils may be amyloidogenic mutants as in some familial amyloidosis, whereas other precursors are normal wild-type proteins [7, 35]. The exact mechanisms through which the proteins are converted into amyloid fibrils in vivo are not well known [35]. Amyloidosis is described in association with different chronic disease in captive cheetah (*Acinonyx jubatus*), Siberian tigers (*Panther tigris altaica*), mink (*Mustela vison*), black-footed cats (*Felies nigripes*), black-footed ferrets (*Mustela nigripes*), Dorcas gazelle (*Gazella dorcas*), mountain gazelle (*Gazella gazella*), bighorn and

Dall's sheep, free-living lioness (*Panthera leo*) and in swan and other anatidae [5, 8, 17, 21, 24, 25, 27-30, 34, 36]. Johnson [13] and Terio [33] reported that the chronic inflammation and chronic stress as a predisposing factor for amyloidosis in cheetah. Terio [34] reported that amyloidosis is the inherited trait in black-footed cats.



Figure 1. Multiple bite mark on the body

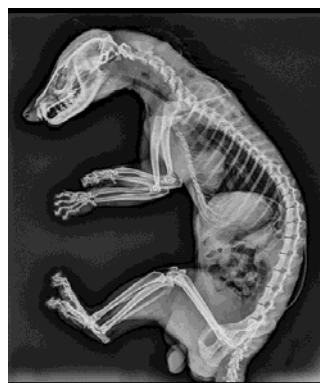


Fig 2: The Radiograph of Civet with longitudinal fracture of maxillary bone.



Figure 3. Hemorrhage and contusion in thoracic and abdominal cavity



Figure 4. Congested and discolored Liver with filled gall bladder .



Figure 5. Congested kidney and spleen .

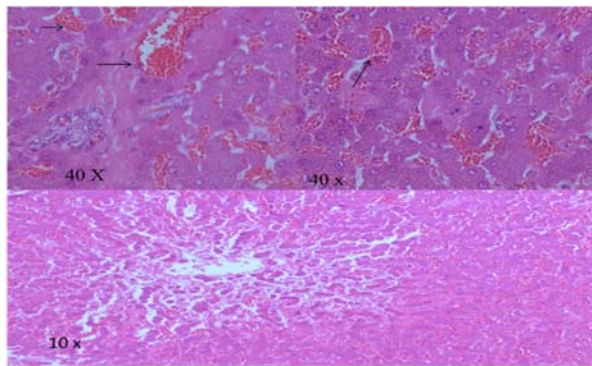


Figure 6 Amyloid deposition (pinkish deposit) in the interstitium of liver(HE staining)

4. Conclusion.

In this case the initial injuries and associated inflammatory process along with the stress might be the predisposing cause for the Amyloidosis.

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6. Reference

1. Abbas A, Kumar V, Abbas AK, Fausto N. Diseases of immunity: amyloidosis, in Pathologic Basis of Disease, Edn 7, Elsevier Saunders, Philadelphia, Pa, USA, 2005, 258-264.
2. Besancon MF, Stacy BA, Kyles AE. Nodular immunocyte-derived (AL) amyloidosis in the trachea of a dog, Journal of the American Veterinary Medical Association. 2004; 224(8):1302-1280.
3. Blunden AS, Smith KC. Generalised amyloidosis and acute liver haemorrhage in four cats, Journal of Small Animal Practice. 1992; 33:566-570.
4. DiBartola SP, Tarr MJ, Webb DM, Giger U. Familial renal amyloidosis in Chinese Shar Pei dogs, Journal of the American Veterinary Medical Association. 1990; 197(4):483-487.
5. Garner MM, Raymond JT, O'Brien TD, Nordhausen RW, Russell WC. Amyloidosis in the black-footed ferret (*Mustela nigripes*), Journal of Zoo and Wildlife Medicine. 2007; 38(1):32-41.
6. Guo JT, Aldrich CE, Mason WS, Pugh JC. Characterization of serum amyloid A protein mRNA expression and secondary amyloidosis in the domestic

7. Gruys E. Protein folding pathology in domestic animals, Journal of Zhejiang University. 2004; 5(10):1226-1238.
8. Hadlow WJ, Jellison WL. Amyloidosis in Rocky Mountain bighorn sheep, Journal of the American Veterinary Medical Association, 1962; 141:243-247.
9. Hawthorne TB, Bolon B, Meyer DJ. Systemic amyloidosis in a mare, Journal of the American Veterinary Medical Association. 1990; 196(2):323-325.
10. Hayden DW, Johnson KH, Wolf CB, Westermark P. AA Amyloid-associated gastroenteropathy in a horse, Journal of Comparative Pathology. 1988; 98(2):195-204.
11. Hoenig M, Hall G, Ferguson D. A feline model of experimentally induced islet amyloidosis, American Journal of Pathology. 2000; 157(6):2143-2150.
12. Ilayaraja S, Arun AS, Sanio J, Yaduraj K, Niraj D. Successful management of a snare wound in a common Palm Civet cat (*Paradoxurus hermaphroditus*). Indian Wildlife yearbook, 11&12, Association of Indian Zoo and Wildlife Veterinarians, Bareilly, India, 2013, 98-100.
13. Johnson KH, Sletten K, Munson L, O'Berin TD, Papendick R, Westermark P. Amino acid sequence analysis of amyloid protein A (AA) from Cheetahs (*Acinonyx jubatus*) with a high prevalence of AA amyloidosis Amyloid; Intl J Exp Clin Invest. 1997; 4:171-177.
14. Johnson KH, Westermark P, Sletten K, O'Brien TD. Amyloid proteins and amyloidosis in domestic animals, Amyloid 1996; 3(4):270-289.
15. Johnson R, Jamison K. Amyloidosis in six dairy cows, Journal of the American Veterinary Medical Association. 1984; 185(12):1538-1543.
16. Kim DY, Taylor HW, Eades SC, Cho DY, Systemic AL. amyloidosis associated with multiple myeloma in a horse, Veterinary Pathology, 2005; 42(1):81-84.
17. Kingston RS, Shih MS, Snyder SP. Secondary amyloidosis in Dall's sheep, Journal of wildlife diseases. 1982; 18(3):381-383.
18. Labelle P, Roy ME, Mohr FC. Primary diffuse tracheobronchial amyloidosis in a dog, Journal of Comparative Pathology. 2004; 131(4):338-340.
19. Landman WJM, Amyloid arthropathy in chickens, Veterinary Quarterly 1999; 21(3):78-82.
20. Linke RP, Geisel O, Mann K. Equine cutaneous amyloidosis derived from an immunoglobulin lambda-light chain. Immunohistochemical, immunochemical and chemical results, Biological Chemistry Hoppe-Seyler, 1991; 372(9):835-843.
21. Linke RP, Hol PR, Geisel O. Immunohistochemical identification of generalized AA-amyloidosis in a mountain gazelle (*Gazella gazella*), Veterinary pathology 1986; 23(1):63-67.
22. Maxie G, Newman SJ, Jubb KVF, Kennedy PC, Palmer N. Urinary system, in Pathology of Domestic Animals, Edn 7, Academic Press, San Diego, Calif, USA 2007; 2:463-465.
23. M'ensua C, Carrasco L, Bautista MJ. Pathology of AA amyloidosis in domestic sheep and goats, Veterinary Pathology 2003; 40(1):71-80.
24. Munson L, Diseases of captive cheetahs (*Acinonyx jubatus*): results of the Cheetah Research Council pathology survey, 1989-1992, Zoo Biology 1993;

12:105-124.

25. Nieto JM, Vázquez S, Quiroga MI, López-Peña M, Guerrero F, Gruys E. Spontaneous AA-amyloidosis in mink (*Mustela vison*). Description of eight cases, one of which exhibited intracellular amyloid deposits in lymph node macrophages, *European Journal of Veterinary Pathology*. 1995; (1):99–103.
26. Niewold TA, Van Der Linde-Sipman JS, Murphy C, Tooten PCJ, Gruys E. Familial amyloidosis in cats: Siamese and Abyssinian aa proteins differ in primary sequence and pattern of deposition, *Amyloid* 1999; 6(3):205-209.
27. Papendick RE, Munson L, O'Brien TD, Johnson KH, Systemic AA. amyloidosis in captive cheetahs (*Acinonyx jubatus*), *Veterinary Pathology*, 1997; 34(6):549-556.
28. Rideout BA, Montali RJ, Wallace RS. Renal medullary amyloidosis in Dorcas gazelles, *Veterinary Pathology*, 1989; 26(2):129-135.
29. Sato A, Koga T, Inoue M, Goto N. Pathological observations of amyloidosis in swans and other Anatidae, *The Japanese Journal of Veterinary Science*. 1981; 43(4):509-519.
30. Schulze C, Brugmann M, Boer M, Brandt HP, Pohlenz J, Linke RP. Generalized AA-amyloidosis in Siberian Tigers (*Panthera tigris altaica*) with predominant renal medullary amyloid deposition, *Veterinary Pathology* 1998; 35(1):70-74.
31. Snyder PW, McGavin MD, Zachary JF. Diseases of immunity: amyloidosis,” in *Pathologic Basis of Veterinary Diseases*, Mosby Elsevier, St Lois, Mo, USA, 2007, 246-251.
32. Taniyama H, Yamamoto S, Sako T, Hirayama K, Higuchi H, Nagahata H. Systemic κ AL amyloidosis associated with bovine leukocyte adhesion deficiency, *Veterinary Pathology* 2000; 37(1):98-100.
33. Terio KA, Marker L, Munson L. Evidence for chronic stress in captive but not wild cheetahs based on adrenal morphology and functions. *J Wildl Dis*. 2004; 51:195-206.
34. Terio KA, O'Brien T, Lamberski N, Famula TR, Munson L. Amyloidosis in black-footed cats (*Felis nigripes*), *Veterinary Pathology* 2008; 45(3):393-400.
35. Westermark P. The pathogenesis of amyloidosis: understanding general principles, *The American Journal of Pathology*. 1998; 152(50):1125-1127.
36. Williams JH, Van Wilpe E, Momberg M. Renal medullary AA amyloidosis, hepatocyte dissociation and multinucleated hepatocytes in a 14-year-old free-ranging lioness (*Panthera leo*), *Journal of the South African Veterinary Association*. 2005; 76(2):90-98.
37. Woldemeskel M. A concise Review of Amyloidosis in animals. *Veterinary Medicine International*, Hindawi Publishing Corporation. Article ID 427296, 2012.