Hemochromatosis (Iron Storage Disease) in Fruit Bats

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Six Egyptian fruit bats *(Rousettus aegyptiacus)* were either found dead or presented with chronic liver disease associated with accumulations of iron (Fe) within the liver as high as 2.3% dry weight. Two other fruit bat species, the Indian flying fox *(Pteropus giganteus)* and the grey-headed flying fox *(Pteropus poliocephalus)*, also had elevated liver Fe levels but without clinical evidence of disease. The bats were fed chopped fruit with a powdered vitamin-mineral supplement, and a gelatinised product containing fruit, meat, and supplements. Analysis revealed that one of the mineral sources used, mono-dicalcium phosphate, contained 11,860 mg/kg Fe dry matter (DM). The Fe content of the complete diet was about 400 mg/kg DM which is close to the toxic range for some domestic animals. The bats also received heavy vitamin C supplementation amounting to an intake of over 7500 mg/kg vitamin C. Ascorbic acid increases iron uptake and enhances Fe toxicity. Pathological findings were similar to those reported in dietary iron overload syndromes in other mammals including man. It is recommended that dietary iron and ascorbic acid levels in diets for frugivorous bats be closely monitored and maintained at required levels only.

Key words: hemosiderosis, bats, Rousettus aegyptiacus, ascorbic acid

INTRODUCTION

Hemochromatosis (HC), or iron storage disease, is a pathological condition associated with excessive iron deposition in the tissues. It has been recognized in several wild animal species in captivity, as well as in man and domestic animals. It is common in some types of birds, notably mynahs and related species, and toucans (Lowenstine and Petrak, 1978; Kincaid and Stoskopf, 1987; Ward et al., 1988). Iron storage disease occurs in lemurs (Benirschke et al., 1985; Gonzales et al., 1984), and hyraxes (Rehg et al., 1978). It has also been seen in birds of paradise in the wild (Ensley and Osborne, 1993). Cattle and horses have been affected (Lavoie and Teuscher, 1993; House et al., 1994) and it is a significant problem in humans (Tavill et al., 1990). No reports were found of the condition in bats.

Iron overload syndromes in humans are grouped into two categories-hereditary (primary) and secondary hemochromatosis. Hereditary hemochromatosis is an autosomoal recessive disorder. Approximately 1 in 200 are homozygous, but only 1 in 5 of those will develop clinical hemochromatosis. Secondary hemochromatosis occurs in people not carrying the HC gene who are exposed to excess quantities of dietary, medicinal, or parenteral iron, and in patients with anemias associated with increased hematopoiesis (Powell and Halliday, 1989; Rubin and Farber, 1994). The term hemochromatosis is usually applied to the condition in which massive deposition of iron is associated with tissue damage in the liver and other organs (Walter, 1989). Hemosiderosis refers to presence of iron deposits in the tissues in the absence of other pathological change.

Hemochromatosis was responsible for the death of six Egyptian fruit bats *(Rousettus aegyptiacus)* (EFB) at the Metropolitan Toronto Zoo (MTZ) between 1992 and 1994. A review of necropsy records of EFB at MTZ revealed a high incidence of tissue hemosiderosis although, in the earlier cases, there was no evidence that iron deposits were directly responsible for death. Two other fruit bat species, Indian flying foxes *(Pteropusgiganteus)* and grey-headedflying foxes *(Pteropuspoliocephalus),* also showed a high incidence of hemosiderosis. Investigation into the cause of the condition revealed that the bats were inadvertently receiving very high levels of dietary iron and ascorbic acid. This report describes the clinical and pathological signs of the disease in EFB and the source of the excessive iron intake.

METHODS

All bats were born in captivity at MTZ, and were kept in a group on public exhibit or in holding cages. The diet was prepared and offered once daily. The bats' diet consisted of approximately 50% by weight of chopped mixed fruit (banana, grape, apple, raisin, orange, pear, etc.) to which a vitamin and mineral powder was added. Temporary additions to the supplements included pollen powder (1991-1992), and shrimp meal (1988-1990). The remainder of the diet was a gelatinised product containing chopped fruit and some meat blended with a vitamin supplement (SA-37, Rogar-STB, London, ON, Canada) and minerals, that is allowed to solidify and then cut into pieces. In the past this had proven a reliable method of ensuring the bats received an adequately balanced diet, although intake has been variable -bats show a definite preference for the fruit component. Dietary ingredients and quantities are listed in Tables 1 and 2.

Blood samples were taken under isoflurane anesthesia from the prepatagial or interfemoral web veins. Biochemical analyses were performed on a Kodak Ektachem analyser (Kodak Canada Inc., Scarborough, ON, Canada). Serum iron levels were measured at a commercial laboratory using a Hitachi 717 analyser. Necropsies were performed on all bats dying in the collection. Tissues were placed in 10% buffered formalin, processed by standard methods, and stained with hematoxylin and eosin (H & E) and Perl's Prussian blue stain. Liver iron levels were measured post mortem on three EFB with clinical hemochromatosis (Cases 3, 4, and 5), and in one EFB, three Indian, and four grey-headed flying foxes with widespread hemosiderosis but without clinically apparent iron storage disease.

RESULTS

Clinical Histories

The bats affected with HC are listed in Table 3. All were adult females. Three of the six, all hand-raised animals, were five years old, one 13 years old, one four, and one just two years old at the time of death. Two bats were found moribund and died shortly thereafter. With the remainder, the initial indicator was one of weight loss. Activity and appetite were usually well-maintained until the later stages of the disease, when separation from the group occurred. The skin, mucous membranes, and urine became icteric as the condition progressed. The livers were palpably enlarged, extending beyond the caudal margin of the sternum, and were firm in texture. Radiography confirmed hepatomegaly. Ascites developed in advanced cases. In the longest surviving case (Case 4), the liver became shrunken and nodular due to extensive fibrosis. The course of the disease varied from three weeks to several months following recognition of the

condition. Progressive deterioration eventually necessitated euthanasia. Case 5 progressed rapidly, due to the development of a hepatic carcinoma. Increases in serum total and conjugated bilirubin were noted in advanced cases, but liver-associated enzyme levels showed no significant differences between icteric and clinically normal animals. Serum iron levels were high in affected EFB (318-480 µg/dl). Clinically normal but siderotic bats showed levels between 150 and 302 µg/dl. Clinical biochemistry and serum iron values are given in Table 4.

Two cases were treated with several courses of the iron-chelating agent deferoxamine (Desferal, CIBA Pharmaceuticals, Mississauga, ON, Canada) 40-60 mg/kg SC once daily for up to three weeks.

Post-mortem Findings

In each bat the tissues were markedly icteric. The livers were enlarged and rounded with an irregular, granular surface and a firm texture. Ascites and hydrothorax were present. In some cases the kidneys were irregular and pitted, and the spleens congested. Microscopically, normal hepatic architecture was disrupted, and in many areas replaced, by extensive periportal and periacinar fibrosis. Large tracts of acellular connective tissue and black pigment granules had replaced normal cellular architecture. Hepatocyte necrosis was evident and in some locations had progressed to areas of coagulation necrosis. Hepatocytes and Kupffer cells adjacent to the bands of fibrosis contained large amounts of hemosiderin. The lungs showed congestion with neutrophils, and accumulation of hemosiderin in macrophages in the alveolar septa. Iron-laden macrophages were seen in other tissues including the spleen, small intestine, lung, and lymph nodes. In Case 5 large areas of the liver had been invaded by a hepatocellular neoplasm. The neoplastic cells contained very little iron but the adjacent parenchymal cells were filled with iron deposits.

A retrospective examination of necropsy results on EFB held at the Toronto Zoo in the since 1974 revealed that in 27/47 (57%) of adult bats examined histologically, hemosiderin was in sufficient quantity in the tissues to warrant note in the report. Extensive hemosiderosis of the liver was seen in three EFB which died of other causes in 1985, and the level of iron in the diet was questioned at that time. Another case with extensive pathology but which did not show clinical disease occurred in 1992 in an EFB housed separately, but concurrently with the problems being seen in the group. The liver was affected in 23/27 (85%) cases and the spleen in 22/27 (81 %). Other sites included the lung (33%) and the heart, small intestine, lymph node and stomach (3% each). No iron was detected in the pancreas or the heart. Within the liver, hemosiderin was seen in either Kupffer cells and hepatocytes, and in most cases, both. In all cases of extrahepatic iron accumulation, hemosiderin was present in macrophages rather than parenchymal cells. Only in the six EFBs were hemochromatosis, or its sequelae, considered the primary cause of death. Case 5 was distinct from the other cases in the development of hepatocellular carcinoma with local metastases. No other cases of neoplasia were seen in the bats with, or without, hemosiderosis.

Results of post mortem liver iron levels are listed in Table 5. All three bats with HC had very high iron levels (mean 20,00 mg/kg dry weight). The clinically normal EFB with hemosiderosis had a liver iron content of 11,000 mg/kg. In contrast, the levels were much lower in both species

of flying fox (mean 3596 mg/kg). None of the latter showed clinical evidence of hemochromatosis although post-mortem examination still indicated substantial liver iron stores.

Dietary Analysis

The original calculated analysis of the whole diet suggested an iron level of about 100 mg/kg but this was based on a presumed iron content for the mineral supplement of only 500 mg/kg. Biochemical analysis of the bat gel in 1991 revealed an iron level of 500 mg/kg, calcium 1.64%, phosphorus 0.68%, zinc 86 mg/kg, and copper 6.2 mg/kg on a dry matter (DM) basis. The pollen, which was eliminated after just a year , contained 200 mg/kg iron. Subsequent laboratory analysis of the ingredients revealed very high iron levels in the calcium-containing supplements used in both the gel and on the fruit portion of the diet. The principal culprit was mono-dicalcium phosphate which contained 11,860 mg/kg iron. Lower amounts were provided by the vitamin supplement (1497 mg/kg), by limestone powder (1206 mg/kg), and steamed bone meal (208 mg/kg). The complete bat supplement powder, added to the fruit portion of the diet to increase the mineral content, contained 3300 mg/kg iron (Table 2). Calculated analysis of the fruit portion of the diet, including the supplement powder , revealed an iron level of 372 mg/kg DM. Feeding trials in 1992 demonstrated that the EFB accepted approximately 65% of the fruit portion of the diet, but less than 20% gel. The total diet consumed therefore contained approximately 400 mg/kg iron. This would amount to a total daily intake for one EFB of approximately 5 mg iron. Results of nutritional analysis of the ration are listed in Tables 1 and 2.

High levels of vitamin C were also added to the diet in response to the requirement of the species for extraneous vitamin C. The vitamin C level of the gel was analysed to be 6140 mg/kg. The powder supplement, which included 5% ascorbic acid powder, contained 32,600 mg/kg vitamin C, providing a calculated total of 7300 mg/kg DM in the fruit portion of the diet (Table 2). The total daily intake of vitamin C for each bat was approximately 90 mg.

DISCUSSION

Initiat evaluation of the diet suggested that the source of the iron was either an ingredient in the bat gel or was the pollen, which had been included for about a year in 1991. Most fresh fruit contains low levels of iron. Subsequent work showed that the high dietary iron values were due to the excessive level of iron in the mineral supplement used both in the gel and on the fruit. This phenomenon has been encountered previously, but in most situations has proven inconsequential, since most livestock species have a high tolerance for iron (McDowell, 1992). Many of the minerals used to supply the calcium and phosphorus needs of animals contain high levels of iron -ground limestone, oyster shell, and many forms of calcium phosphate may contain at least 2000-5000 mg/kg iron. Pigs are tolerant of iron levels up to 3000 mg/kg, cattle and poultry up to 1000 mg/kg, and sheep 500 mg/kg. It has however caused problems when these mineral sources are included in the diets of susceptible species such as mynahs, which will develop hemochromatosis at dietary iron levels of 200 mg/kg or less. Even some commercial primate and bird diets contain levels of iron of 300 mg/kg or more. The daily intake of more than 5 mg iron for a 130g EFB represents 100-200 times the recommended daily allowance for humans on a per kg basis.

Dietary iron levels are not measures of potential toxicity alone since absorption rates vary greatly. The toxicity of high levels of dietary iron depends on its bioavailablity- i.e. the form of iron, and

the presence of other compounds which may enhance or reduce its absorption. For instance, dogs have fed for as long as 18 months on diets containing 1% ferric oxide, while other iron salts have proven toxic at very low intakes (NRC, 1985). The bioavailability of iron in domestic animal feeds varies but may be estimated to be 50-60% (McDowell, 1992), and only a portion of that may actually be taken into the circulation. Heme iron is more readily taken up than iron from plant or mineral sources. Lemurs accumulate large iron stores on diets containing less than 300 mg/kg iron DM. It has been suggested that lemurs in captivity might be susceptible to iron overload as a result of genetic adaptation to a natural diet containing high levels of tannins and other dietary ingredients which bind iron, rendering it unavailable for absorption (Spelman et al., 1989).

Vitamin C is a major factor in dietary iron absorption (Morris, 1987). Ascorbic acid increases the potential toxicity of iron since it chelates iron and promotes the conversion of ferric salts (Fe^{+++}), the usual form of iron in plant material and minerals, to ferrous salts (Fe^{++}), the form in which it is most easily absorbed through the cells of the duodenal mucosa. Excesses of vitamin C will increase the amount of iron absorbed by three to five-fold (Monsen, 1982), and its absence significantly decreases iron uptake (Morris, 1987). Ascorbic acid also enhances the toxicity of iron at the intracellular level. The cytopathological mechanisms of iron overload are not fully understood but it is believed that toxicity principally results from lipid peroxidation and the formation of free-radicals, which damage cell membranes and increase the fragility of cellular lysosomes. Lipid peroxidation was rapid when normal hepatic lysosomes were exposed to iron salts in vitro. At pH 7.4, iron released from hemosiderin could initiate lipid peroxidation only in the presence of ascorbate (Bacon and Brit ton, 1990; Tavill et al., 1990;). The level of vitamin C ingested by the bats was excessive and may have resulted from overenthusiastic efforts to provide this essential nutrient. Typical levels, even in diets for primates, contain a maximum of 3000 mg/kg DM. The bats' diet contained more than twice that.

Iron is an essential nutrient due to its role in hemoglobin and intracellular cytochrome formation. Most iron exists in the body as protein-bound complexes such as hemoglobin. Nonheme iron is also bound in the blood to the proteins transferrin and apoferritin. In normal individuals of most species only 30-40% of the transferrin carries iron, the remainder being known as the latent iron-binding capacity. The level of iron in the plasma varies within individuals on a diurnal basis. Normal serum iron levels in man are < 180 µg/dl. Typical levels are 127 µg/dl for adult males and 113 µg/dl for adult females. Values in animals are similar or slightly higher (Morris, 1987). In cases of hemochromatosis, iron levels are usually > 300 µg/dl representing over 95% of total iron-binding capacity, compared with a normal of around 30% (Morris, 1987; Tavill et al., 1990). The levels in the EFB were very high (up to 480 µg/dl) and there was good correlation between serum iron levels and the development of HC (Table 4). However, since all bats were on the same diet we have no truly normal values for this species.

Up to levels of about 500 mg/kg, iron is stored in the liver equally as ferritin and insoluble hemosiderin, an amorphous compound containing ferric hydroxide and little protein. Above 1000-2000 mg/kg of iron, hemosiderin deposition predominates (Morris, 1987; McDowell, 1992). Normal liver iron levels in man and other animals are usually between 300 and 1800 mg/kg dry weight (Morris, 1987; Tavill et al., 1990). In cases of iron overload, liver iron levels are increased to 10,000 mg/kg and up (Powell and Halliday, 1989; Tavill et al., 1990). The levels found in the EFB were as high, or higher, than those seen in hereditary HC in man. The levels in

the clinically normal EFB and the flying foxes were also elevated and this was reflected in the widespread hemosiderosis in the liver, spleen, and other organs. Results from our limited number of cases would suggest that hepatic storage of iron in bats becomes toxic above 12,000 mg/kg. It is possible that EFBs may be absorb more iron than the other bat species, or that, in view of their smaller size, they were receiving proportionately more iron than the larger species.

Two adult female bats diagnosed ante-mortem were treated with chelation therapy for several months. Serial liver biopsies were taken in an attempt to document a reduction in the level of iron in the liver histologically, as has been done in birds (Loomis and Wright, 1993). Reductions were noted in serum bilirubin and enzyme levels but no reduction in hepatic or serum iron levels were seen during the course of therapy. Repeated phlebotomy is the treatment of choice for human HC and can significantly increase life expectancy. Iron chelation therapy can increase iron loss but is expensive and is less effective for long-term treatment (Crawford and Halliday, 1991). Therapy for the bats was probably not aggressive enough and the disease was probably too far advanced to effect a recovery at that stage. In addition, the source of the iron was not detected or corrected until after death.

In hereditary HC, iron accumulates in the hepatocytes as well as the parenchymal cells of other organs, particularly the pancreas, until the cellular storage capacity is exceeded and cytotoxicity occurs. Overflow of iron deposits to the cells of the reticuloendothelial system occurs as the condition progresses but they are not primarily affected. As hepatocellular damage continues, widespread cirrhosis develops (Powell and Halliday, 1989; Rubin and Farber, 1994). In secondary HC, hemosiderin accumulates in the cells of the mononuclear phagocyte system, particularly the spleen, rather than in the parenchymal tissues, but, when severe, will spillover to the hepatocytes causing cellular death and eventually fibrosis (Walter, 1989).

The histopathology of the disease in the bats closely resembles that described in secondary HC in man, as well as in iron overload syndromes in hyraxes, horses, and some types of birds such as tanagers. Although hepatocytes were affected extensively in the severe cases, there was also a wide distribution in the cells of the reticuloendothelial system. This is in contrast to the distribution seen in mynahs, and in hereditary HC in which hepatocytes and other parenchymal cells are the principal targets (Randell et al., 1981; Ward et al., 1988; Rubin and Farber, 1994). Fibrosis occurs as an end-stage development in both types of HC in man, when iron levels reach close to 20,000 mg/kg dry weight (Bassett et al., 1986). Fibrosis was extensive in most of the bat cases which likely reflects the long duration and high level of iron deposits. Experimental protocols have been unsuccessful at producing fibrosis in laboratory animals, presumably because of the inadequate degree of iron overload achieved (Bacon and Brit ton, 1990). Approximately 15-20% of human patients with hemochromatosis develop hepatocellular carcinoma (Rubin and Farber, 1994), and the incidence of hepatic neoplasia in lemurs with hemosiderosis was considered disproportionately high compared with unaffected primates (Gonzales et al., 1984). One of our six cases in EFBs developed a hepatic neoplasm.

The bats' diet contained very high levels of natural and supplemented Vitamin C. Although the absolute iron levels were still within the range for some foods and forages, iron availability and absorption were probably maximal. It appears that the bats were faced with high levels of iron, rendered more toxic by high levels of vitamin C. There were no other apparent causes for increased iron uptake. Anemia was not present- red cell indices were normal or above normal.

There is no suggestion that any of these fruit bat species are unusually susceptible to iron toxicity when faced with moderate levels of iron intake, in the way that genetic HC patients, and mynahs and toucans are. It is likely, however, that they are more susceptible than many domestic mammals. An intriguing question is why sanguinivorous bats, the vampires, are able to avoid iron overload on a diet containing as much as 3000 mg/kg iron.

This was the second occurrence of nutritional toxicity in the fruit bats at MTZ in recent years. Proliferative bone disease caused by fluorosis was seen in the same three species of fruit bat. High levels of fluoride were detected in shrimp meal, and in the same mono-dicalcium phosphate powder that contributed to the iron toxicity.

CONCLUSIONS

1. Egyptian fruit bats will develop hemochromatosis when fed high levels of dietary iron.
2. Iron absorption and toxicity was likely enhanced by excessive vitamin C intake.
3. Pathologic findings in bats resembles secondary hemochromatosis in man.
4. Toxicity may result from the use of food supplements which contain high concentrations of iron or other minerals.

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TABLE 1. Iron and vitamin C levels in MTZ diet for one Egyptian fruit bat in 1992

Ingredient	Quantity g	Iron mg/kg DM	Vitamin C mg/kg DM
Bat Gelatin	65	500	6140[1]
Banana	15	21	292[2]
Grape	15	21	132[2]
Orange	15	20	3571[2]
Apple	7.5	17	219[2]
Pear	7.5	16	229[2]
Melon	7.5	22	1833[2]
Peach	7.5	42	573[2]
Figs	6	27	67[2]
Mixed dried fruit	4	21	84[2]
Pollen powder	2	207	205[1]
Bat supplement[4]	2	3940	32,600[3]

[1] Analysed values
[2] From Adams, 1975
[3] Calculated from analysis of ingredients
[4] See Table 2

TABLE 2. Iron and vitamin C levels in the ingredients of MTZ bat supplement powder

Ingredient	%	Iron mg/kg	Vitamin C mg/kg
Mono-dicalcium phosphate	34	11,000	0
Vitamin-mineral supplement	29	1497	6,000
Steamed bone meal	23	208	0
Limestone powder	6	1206	0
Ascorbic acid powder	5	1	1,000,000
Vitamin E powder	3	713	0

TABLE 3. Egyptian fruit bats (*Rousettus aegyptiacus*) affected with clinical and pathological hemochromatosis at MTZ

Case No.	Bat No.	Sex	Age
1	22807	F	5 yr
2	22049	F	5 yr
3	26065	F	2 yr
4	25647	F	4 yr
5	14739	F	13 yr
6	25336	F	5 yr

TABLE 4. Serum biochemistry values from Egyptian fruit bats in 1992

Bat No.	ALPL IU/l	LDH IU/l	AST IU/l	ALT IU/l	T.Bil mmol/l	C.Bil mmol/l	Fe µg/dl
26065[1]	209	1335	213	149	150	-	480
25647[1]	428	2076	100	41	<1	3.1	479
14739[2]	369	1710	124	52	3.4	1.7	150
25336[2]	354	>2500	316	170	-	-	184
15262	511	1070	146	55	<2	-	-
22261	364	1139	103	74	-	-	302
25436	287	947	97	51	<2	0.6	289
27219	814	1088	88	57	<2	0.5	236
27222	925	1487	130	57	<2	1.0	168

[1] Clinically affected at time of sample
[2] Developed hemochromatosis subsequently

TABLE 5. Liver iron levels in three species of bat at MTZ

Species	Bat No.	Case No.	Pathology	Iron mg/kg
Egyptian fruit bat	26065	3	HC	23,300
Egyptian fruit bat	25647	4	HC	17,500
Egyptian fruit bat	14739	5	HC	19,600
Egyptian fruit bat	15262		HS	11,100
Grey-headed flying fox	22012		HS	1468
Grey-headed flying fox	22013		HS	2341
Grey-headed flying fox	22014		HS	655
Grey-headed flying fox	22902		HS	4979
Indian flying fox	11144		HS	8274
Indian flying fox	14306		HS	5480
Indian flying fox	25188		HS	1972