MORTALITY OF CAPTIVE GIRAFFE (GIRAFFA CAMELOPARDALIS) ASSOCIATED WITH SEROUS FAT ATROPHY: A REVIEW OF FIVE CASES AT AUCKLAND ZOO

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MORTALITY OF CAPTIVE GIRAFFE (GIRAFFA CAMELOPARDALIS) ASSOCIATED WITH SEROUS FAT ATROPHY: A REVIEW OF FIVE CASES AT AUCKLAND ZOO

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Abstract: Five giraffe (Giraffa camelopardalis) died peracutely within an 8-yr period. The giraffe were maintained in an outside enclosure during the day and moved under shelter at night. All the deaths occurred in winter. All the dead giraffe had serous fat atrophy at postmortem. The giraffe were fed good quantities of browse, together with alfalfa hay and commercial supplements. Retrospective analysis of the dietary ingredients showed that the diets were energy deficient. Subsequent additional high-energy feeds have caused a marked increase in surviving giraffe body weights although energy levels consumed were at the lower end of current recommendations. The relationship between low-energy reserves, high-energy demand in colder temperatures, and the possibility that hypoglycemia is a credible cause of the collapse of giraffe in these circumstances, is postulated to be the likely pathogenesis of giraffe deaths, previously reported elsewhere under the generic term “peracute mortality syndrome.”

Key words: Giraffe, Giraffa camelopardalis, nutrition, peracute mortality syndrome, serous fat atrophy, cold, energy, hypoglycaemia.

INTRODUCTION

Captive giraffe (Giraffa camelopardalis) often die suddenly without a history of illness or ill thrift. Because the death in these cases is usually unexpected, the term “peracute mortality syndrome” was introduced when this phenomenon was first fully described in 17 animals in 1978. On postmortem, there is serous fat atrophy, particularly around the heart, and frequently gastrointestinal ulceration, pulmonary inflammation, and edema. A second survey in 1993 revealed 11 more cases; a literature review in 1998 collated 12 other literature reports on giraffe deaths with similar findings, the earliest of which dated from 1854; and additional cases have been reported since. Poor nutrition, particularly inadequate protein levels in the diet, diets with a high proportion of grass or alfalfa hay and a low proportion of browse, chronic energy malnutrition, intake problems because of worn teeth, environmental stress, and increased energy demands because of cold have all been suggested as contributing factors to the peracute deaths.

The actual physiologic process that causes the giraffe to collapse and die is not fully understood. Hypotheses have included cholinergic bradycardia, hypovolemic shock or adrenal exhaustion, toxicoses, mineral imbalances, and hypoglycaemia. After the sudden death of a mature female giraffe at Auckland Zoo New Zealand in July 2001, a review was made of four other sudden deaths in giraffe at the Zoo over the previous 8 yr.

CASE REPORTS

A brief summary of the giraffe referred to in this study, together with the different dietary regimes from 1990 to 2003, is given in Table 1. All five giraffe that died with serous fat atrophy died in the (southern hemisphere) winter months of July or August. All were captive born. Average winter temperatures during these 2-mo ranged from overnight lows of 6°C to daytime highs around 15°C (Auckland Weather Office Data). The giraffe remain outside during the day and are housed overnight in a giraffe house that provides shelter from wind and rain but little in the way of heating.

On postmortem examination, all five giraffe had serous atrophy of pericardial and other fat deposits, and each animal had an acute pneumonia associated with the inhalation of ruminal contents after the animal had collapsed. In addition, giraffe C had histopathologic evidence of a chronic abomasitis associated with internal parasitism that was thought unlikely to be responsible for the animal’s death, although it may have contributed to loss of protein. Apart from a mild rumenitis in giraffe D, there were no other significant findings in any other giraffe from either gross or histologic examination at postmortem (the tissues that underwent histologic examination in each giraffe included lung, heart,
Table 1. Summary of dietary and clinical history of the Auckland Zoo giraffe in relation to serous fat atrophy deaths.

<table>
<thead>
<tr>
<th>Diet fed and years</th>
<th>Diet ingredients</th>
<th>Giraffe in Zoo</th>
<th>Date of Giraffe deaths</th>
<th>Age at death</th>
<th>Serous fat atrophy at necropsy?</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>B, born 1991</td>
<td>B: Jul 1994</td>
<td>2.5 yrs</td>
<td>Yes</td>
</tr>
<tr>
<td></td>
<td></td>
<td>C, born 1992</td>
<td>C: Jul 1994</td>
<td>1.5 yrs</td>
<td>Yes</td>
</tr>
<tr>
<td>Diet II: 1995–1998</td>
<td>Alfalfa hay, browse + 3.5 kg calf pellets</td>
<td>D, E, F, G</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>E: Jul 2001</td>
<td></td>
<td>17 yrs</td>
<td></td>
</tr>
</tbody>
</table>

*The diet was in transition from January 2002–June 2002.

Liver, kidney, lymph nodes, abomasum, rumen and/or reticulum, and large bowel; brains were examined in giraffe D and E.

Deaths in four of the giraffe occurred overnight and were discovered when the giraffe house was opened at the start of the day, whereas giraffe C died during the day in an outside enclosure. After Giraffe C’s collapse, blood samples were taken from her before death. She had a low serum albumin (17 g/L; reference range (RR) 20–44 g/L) contributing to a total protein of 44 g/L (RR 68–108 g/L), a mild hypocalcemia (1.76 mmol/L; RR 1.68–2.65 mmol/L) and a hyperkalemia (7.1 mmol/L; RR 3.4–6.5 mmol/L) (ISIS reference values20). Other serum chemistry values were normal, although blood glucose was not analyzed. Giraffe C also had a nonregenerative, normocytic, hypochromic anemia with a hematocrit of 25.2% (RR 24–49%). She was treated with an unrecorded volume of i.v. electrolytes together with dextrose after she collapsed but died within 2 hr of her collapse. Blood tests of three other adult giraffe at that time revealed one (giraffe D) with a serum albumin in the low normal range (23 g/L) and a hematocrit of 28.7%. Her blood glucose was normal, as were all the serum chemistry and hematology results from two other giraffe.

Giraffe at Auckland Zoo had traditionally been fed a diet of alfalfa (*Medicago sativa*) hay together with quite large amounts of browse (mainly *Pittosporum* spp., *Coprosma* spp., *Meryta sinclairii*, *Metrosideros excelsa*) (diet I). After the initial three deaths and the later discovery of giraffe D with hypoalbuminemia in 1994, calf grower pellets (NRM Grower Plus, NRM New Zealand, Private Bag 98-927, Newmarket, Auckland, New Zealand), at a level of 3.5 kilograms (kg) per day per animal, were added to the diet to increase energy and protein levels in the diet (diet II). This supplementation continued until 1999, when the calf pellets were replaced with a commercial food, Chaffhage (The Great Hage Company, RD2, Reporoa, NZ). Chaffhage had claimed energy and protein levels equivalent to the calf pellets and was considered to be more suitable for giraffe because it had an alfalfa silage base (diet III). Up to 3.5 kg per animal per day of Chaffhage was fed, together with ad lib. alfalfa hay and browse. In addition, a multimineral supplement (Feramo H, Bomac Laboratories Ltd, NZ) was given daily and a vitamin E supplement
Table 2. Nutritional analysis of Giraffe food ingredients fed in 2001.

<table>
<thead>
<tr>
<th>Analysis</th>
<th>Alfalfa</th>
<th>Chaffhage</th>
<th>Grower pellets</th>
<th>Browse (average)</th>
<th>Browse (range)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dry matter (%)</td>
<td>82</td>
<td>43</td>
<td>85</td>
<td>26</td>
<td>20–42</td>
</tr>
<tr>
<td>Protein (% DM)</td>
<td>14</td>
<td>19</td>
<td>21</td>
<td>9</td>
<td>7–12</td>
</tr>
<tr>
<td>Digestible organic matter (g/100 g DM)</td>
<td>51</td>
<td>52</td>
<td>81</td>
<td>66</td>
<td>47–76</td>
</tr>
<tr>
<td>ME (MJ/kg DM)</td>
<td>8.2</td>
<td>8.2</td>
<td>13.1</td>
<td>10.6</td>
<td>7.6–12.2</td>
</tr>
</tbody>
</table>

*Twelve commonly fed browse species were placed into four separate groups selected on similarity of leaf type. All were evergreen New Zealand native species, and only leaves and edible twigs were analyzed.

a Digestible organic (DOMD) matter was estimated by the pepsin cellulose method.29

b ME, metabolic energy.

c ME was estimated as 0.016 DOMD.3

(White E Powder, Bomac Laboratories Ltd) twice weekly from 1999. The giraffe were all conditioned to stand in a crush chute daily, using small amounts of vegetables and fruit as an incentive. This conditioning food, which was also used during public “giraffe” encounters, accounted for 3% of the total dry matter (DM) in the diet.

The deaths of giraffe D in July 2000 and then giraffe E exactly a year later led to a further diet and husbandry review. Samples of alfalfa hay, browse, Chaffhage, and the previously used calf grower pellets were sent to a laboratory (Alpha Scientific, Hamilton, New Zealand) for nutritional analysis. The results are summarized in Table 2.

The commercial food Chaffhage had an energy level well below that claimed by its manufacturers, which resulted in the actual total energy supplied to the giraffe being overestimated by about 25%. In addition, the lower than claimed DM content of the Chaffhage food reduced the total amount of protein fed to the giraffe. The alfalfa hay also had a lower than expected nutrient content, which compounded the energy and protein deficit.

Once the food analysis became available, a retrospective calculation was made to get an estimate of the average total nutrient intake, including the Chaffhage, on the basis of intake measurements performed on the remaining giraffe (group average) for a week during which food offered and left over was quantified (Table 3).

Table 3. Nutritional content consumed per giraffe per day from 2001 to 2004. (For diets, see Table 1).a

<table>
<thead>
<tr>
<th>Food</th>
<th>Fresh weight (kg)</th>
<th>DM (kg)</th>
<th>Protein (kg)</th>
<th>Energy (ME MJ)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Diet III: 1999–Jul 2001</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Alfalfa Hay</td>
<td>2.5</td>
<td>2.05</td>
<td>0.29</td>
<td>16.81</td>
</tr>
<tr>
<td>Browse</td>
<td>3.5</td>
<td>0.91</td>
<td>0.08</td>
<td>9.65</td>
</tr>
<tr>
<td>Chaffhage</td>
<td>3.5</td>
<td>1.51</td>
<td>0.29</td>
<td>12.34</td>
</tr>
<tr>
<td>Total</td>
<td>9.5</td>
<td>4.47</td>
<td>0.66</td>
<td>38.80</td>
</tr>
<tr>
<td>(14.8% DM)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Diet IV: Aug 2001–Dec 2001</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Alfalfa Hay</td>
<td>1.5</td>
<td>1.23</td>
<td>0.17</td>
<td>10.08</td>
</tr>
<tr>
<td>Browse</td>
<td>3.5</td>
<td>0.91</td>
<td>0.08</td>
<td>9.65</td>
</tr>
<tr>
<td>Calf pellets</td>
<td>7.5</td>
<td>6.38</td>
<td>1.34</td>
<td>83.51</td>
</tr>
<tr>
<td>Total</td>
<td>12.5</td>
<td>8.52</td>
<td>1.59</td>
<td>103.24</td>
</tr>
<tr>
<td>(18.7% DM)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Diet V: Jun 2002–Apr 2004</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Alfalfa Hay</td>
<td>5.0</td>
<td>4.10</td>
<td>0.70</td>
<td>33.60</td>
</tr>
<tr>
<td>Browse</td>
<td>2.6</td>
<td>0.68</td>
<td>0.06</td>
<td>7.16</td>
</tr>
<tr>
<td>Calf pellets</td>
<td>3.0</td>
<td>2.48</td>
<td>0.54</td>
<td>33.45</td>
</tr>
<tr>
<td>Linseed*</td>
<td>1.0</td>
<td>0.90</td>
<td>0.29</td>
<td>9.46</td>
</tr>
<tr>
<td>Total</td>
<td>12.1</td>
<td>8.66</td>
<td>1.46</td>
<td>83.70</td>
</tr>
<tr>
<td>(17.8% DM)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

a Note: there are insufficient records for a nutritional analysis of diets before 1999.

b National Research Council values.30
Immediately after this intake study, up to 7.5 kg per day per giraffe of calf pellets was reintroduced to the giraffe diet to replace Chaffhage as a temporary measure to prevent potential problems with the remaining three animals (diet IV). This high level of concentrate feeding was continued until early summer. Browse and alfalfa hay were continued ad lib., although the giraffe reduced the amount of alfalfa actually eaten once they were receiving high levels of concentrate, as assessed by a subsequent 1-wk intake study. Although there were concerns that the amount of concentrates now being fed to each remaining giraffe could result in ruminal acidosis, it was considered that this was a lesser short-term risk than maintaining their low-energy status until the risk of colder weather had passed. No diarrhea or other potential problems associated with high concentrate levels in the diet were observed.

At the time of the death of giraffe E in July 2001, Auckland Zoo had no means of weighing its giraffe. Weighing scales were installed 2 mo after her death at which time the two remaining adult female giraffe (F and G) weighed 612 and 738 kg, respectively. These weights were taken after the animals had been on the higher energy diet (diet IV) for 7 wk. The increased energy intake on diet IV resulted in an average 8% increase in body weight (BW) in these two giraffe over a period of 4 mo from late August 2001 until late December.

Over the first 6 mo of 2002, the amount of pelleted food in the diet was gradually reduced from 7.5 to 3.5 kg daily per adult giraffe. Over this period, linseed was added to the diet, and the availability of a much higher quality alfalfa hay led to a greatly increased intake by the giraffe of this food which continued to be available ad lib.. Giraffe weights during this diet-transition period continued to increase, and in March 2002, giraffe F weighed 704 kg and giraffe G 858 kg. Since June 2002, the diet has remained relatively constant (diet V) although the quality of the alfalfa hay, and thus the intake, declined in the winter and early spring months each year as the hay aged. The daily metabolizable energy (ME) intake on diet V was estimated at 83.7 MJ.

Both female giraffe raised healthy calves that were born in November 2002. Giraffe F, which was the smaller of the two remaining adult females, was euthanized after fracturing a humerus in May 2003. Her BW then was 706 kg. Because the cause of death was euthanasia, no specimens were sent for histopathology, but fat deposition was evident in her abdomen and in the coronary band of the heart during gross necropsy. At that time, giraffe G weighed 892 kg.

**DISCUSSION**

The dietary requirements of giraffe are not fully described. A minimum protein content of 15% of a total daily food consumption of 1% to 2% of the animal’s BW, and a gross energy input of about 150 MJ, equivalent to 75 MJ ME for an adult giraffe has been suggested.17 The American Zoo Association Nutritional Advisory Group Fact Sheet on the feeding of large ungulates recommends a hay/pellet intake of 2% of BW and a protein content of 15–19%. However, experimental determination of protein requirements in several wild ruminants has not identified a single species to date that has maintenance protein requirements in excess of 10% of DM.9 It is apparent that energy intake, rather than dietary protein levels, should be the major focus of attention in giraffe nutrition. In a species with such an enormous BW range as the giraffe (with recorded BWs in adult specimens ranging from 680 to 1,400 kg), a single absolute value for energy intake should not be used as a guideline. Rather a value relative to (metabolic) BW should be calculated.

Malnutrition in the form of an energy deficiency is a major problem in captive giraffe. This was noted in the first survey on giraffe with serous fat atrophy,13 which found that mainly thin animals were affected. The initiating cause of the peracute deaths in giraffe at Auckland Zoo has undoubtedly been nutrition, with inadequate energy input as the primary factor.

In feeding trials with captive adult giraffe, ME intakes vary from 67 to 108 MJ11 and 63 to 90 MJ9 per animal per day were estimated. For the purpose of these studies, a maintenance energy requirement of 0.39–0.59 MJ ME/kg0.75 was assumed. For a 706-kg giraffe (giraffe F), this would translate into a ME requirement of 53.4–80.3 MJ per day. Given the fact that giraffe F maintained BW for more than a year, including birth and lactation, on a diet providing an estimated 84 MJ ME per day, the actual energy requirement of this giraffe is likely to have averaged over this period about 0.61 MJ ME/kg0.75 per day. Giraffe G also did not lose BW (but actually gained weight from 858 to 892 kg) in the same period, on an average energy intake somewhat less than 0.52 MJ ME/kg0.75 per day.

Estimating energy intake from DM intake can be difficult. DM intakes in giraffe often do not reach the recommended 1.8–2.0% of BW in captivity.11,23 In the case of Giraffes F and G, DM intake was estimated at 0.61–0.73% BW on diet III, peaking
at 1.15–1.39% BW on diet IV (on which they gained weight), and stabilized at 0.92–1.17% BW on the (energetically adequate) diet V.

Although many browsing or grazing ruminants can thrive on solely grass hay or alfalfa hay diets, giraffe cannot. This is not because of alfalfa hay having a particularly low-energy content but because animals often do not ingest enough of this item. Because alfalfa hay was the predominant diet item before the three deaths with serous fat atrophy in 1993 and 1994, it can be assumed that the giraffe were energy deficient during that period and that low winter temperatures exacerbated this condition. Evidently, raising the energy content of the diet (diet II) prevented further cases until, when the diet was changed as a result of incorrect information from the “Chaffhage” manufacturer (diet III), the energy supplied to the animals was reduced again. Once more, it was evident that the giraffe did not adequately compensate for the energy deficit in their food ration by an increased intake of the ad lib. alfalfa hay. After another increase in dietary energy supply with diets IV and V, no further cases of serous fat atrophy were noted, and in contrast, the one animal that was euthanized because of an inoperable bone fracture was found, on necropsy, to have normal body fat stores.

A major factor that will determine the amount of energy derived from the roughage source is the quality of the alfalfa hay. The palatability of the hay at Auckland Zoo has a major effect on the amount they are willing to consume. For giraffe, only high-quality alfalfa hay should be used. It has been recommended that giraffe, along with other ruminants, should ingest a ration of roughage/concentrate of 60:40 (DM basis) to prevent the development of a chronic but usually subclinical acidosis in the rumen. However, a review has shown that this recommendation is often not met, and this is most likely a consequence of the animals’ reluctance to ingest larger proportions of the ad lib. roughage source. In the case of the Auckland giraffe, the roughage/concentrate ratio (DM basis) was 66:33 on the energy-deficient diet III (c.f. Table 3), and 60:40 on the energetically adequate diet V but was very low at 25:75 on diet IV. High concentrate levels fed in diets such as diet IV are very likely to induce acidic rumen conditions which can predispose to rumenitis. As a contrast, if the alfalfa hay is of high quality, Hummel et al. demonstrated that it is possible to maintain giraffes on a diet with a roughage/concentrate ratio of 70:30.

A connection between different DM intakes and the amount of internal body fat has been observed in a study involving two separate giraffe facilities: one with a case of serous fat atrophy where a relatively low DM intake was reported and one in a facility where giraffe had a higher DM intake and copious amounts of body fat were noted. Because energy deficiency is not a sudden event but the result of a medium-term to chronic process, the term peracute mortality syndrome probably does not reflect the pathogenesis of the problem but rather its human perception. Wild animals usually mask diseases or weakness as long as possible. To detect chronic malnutrition, Auckland Zoo now regularly weighs giraffe and also uses a body condition score. In addition, periodic measurements of food intake are also desirable.

One common factor in the five deaths is that they all occurred during the coldest months of the year. There has been speculation about the increased energy demands of giraffe after prolonged cold exposure. An incidence of high mortalities in free-ranging giraffe after a period of exceptionally cold and wet weather has been reported from Southern Africa.

As previously mentioned, at 6–15°C, Auckland winters are not particularly cold, although night temperatures can fall below a temperature of 13–16°C, which is recommended as a minimum husbandry temperature by one author and far below the recommended indoors temperature range of 21–22°C by some others. Low night temperatures do occur in the natural habitat of giraffe, but these do not persist for several months. Because giraffe are partially obligatory heterothermic, with body temperatures dropping close to 36°C at an ambient temperature of 16°C, continuing exposure to low temperatures will pose an additional energy demand to maintain the normal body temperature of 37.5–38.8°C.

A thin appearance (low—body condition score) and serous fat atrophy at necropsy have been described in other captive wild ruminant species. In another browsing ruminant species, the moose (Alces alces), it was found that body condition correlated with the amount of trough feeds offered. When available trough feed was inadequate, these animals did not compensate by ingesting a higher proportion of the ad lib. hay offered.

Serous fat atrophy in okapi (Okapia johnstoni) has been described but exclusively in coincidence with old age or excessive tooth wear (or both). This observation is in accord with the suspicion that excessive tooth wear might be a major contributing factor to serous fat atrophy in captive giraffe. However, in giraffe, serous fat atrophy at necropsy is not only observed in aged individuals but also occurs regularly in young animals, such
as giraffe B and C. Two incidences of serous fat atrophy have been reported in eland (Taurotragus oryx) after either a prolonged period of social stress, with restricted access to feeding troughs, or a very long transport procedure, with frequent vehicles changes lasting over a week. In a third eland, death after long loss of body condition was avoided when the animal was kept individually, with unrestricted access to its feeding station, emphasizing that one should consider questions of food accessibility and acceptance as well as those of nutrient composition.32

The exact physiologic processes that occur during the sudden collapse of affected giraffe are not fully understood. However, because the serous fat atrophy observed in all affected giraffe is a strong indication of inadequate energy reserves, it may be linked with the ability of the animals to maintain adequate levels of blood glucose in the final stage. Two giraffe that died in Florida with serous fat atrophy at necropsy were both recorded with hypoglycaemia shortly before death.2 The physiologic effects of acute hypoglycaemia in many other species is well understood, leading to weakness, collapse, coma, and death. If hypoglycaemia is the primary physiologic mechanism of the collapse, it does, however, not appear to be associated with ketosis. There are no reported cases of ketosis in giraffe, and neither giraffe D or giraffe E had ketones in their urine at the time of death. In domestic cattle, ketosis mainly occurs in animals of good body condition that experience a sudden dramatic increase in energy requirements (such as the onset of lactation), which is not met by the diet.5,18,27 The absence of ketone bodies in collapsed giraffe could therefore be another indication that the serous fat atrophy syndrome is not the result of a peracute episode but rather that at the end of a chronic process.

On the basis of this review of cases, we recommend that to reduce the risk of serous fat atrophy in captive giraffe, provision of adequate levels of energy in the diet would appear to be the most effective preventative step. In temperate climates, where temperatures may drop below 13°C, a diet consisting of a roughage source alone will not meet their requirements. If possible, the energy should be supplied in a formulation (with a maximum of 40% concentrates on a DM basis) that does not induce acidosis. For this purpose, alfalfa hay of only the highest quality should be fed, so that as much energy as possible can be provided with the required roughage intake. Persistent exposure to temperatures below 20°C should be avoided unless the energy intake can be substantially increased. Treatment of weak or collapsed giraffe with fluids containing glucose or dextrose, supplemented with calcium and magnesium, may offer the best chance of a successful response.

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LITERATURE CITED


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