Successful management of Heinz body hemolytic anemia associated with leek (*Allium ampeloprasum*) ingestion in a South American coati (*Nasua nasua*)

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Abstract

Objective **-** To describe the diagnosis, management and outcome of Heinz body hemolytic anemia in a South American coati (*Nasua nasua*) secondary to suspected leek (*Allium ampeloprasum*) toxicosis.

Case summary – A South American coati presented with Heinz body hemolytic anemia following addition of leeks to its diet for two to five days prior to initial presentation. Administration of a whole blood transfusion from an animal of the same species (conspecific) and supportive care resulted in immediate improvement in clinical signs. Normal behavior fully returned within six days of transfusion. Hematological evidence of anemia resolved by four weeks and there were no significant features of oxidative injury present by eight weeks following initial presentation.

New information provided **–** This is the first reported case of Heinz body hemolytic anemia, suspected leek toxicosis and administration of a blood transfusion in this species.

Key words

Zoo animal, *Allium* spp. toxicosis, blood transfusion

Abbreviation list

CPDA = Citrate-phosphate-dextrose-adenine

IM = intramuscularly

IV = intravenous

RI = reference intervals

Introduction

Heinz body hemolytic anemia is a rare but potentially severe condition in domestic and wild animals.[1-5] It is caused by the formation of denatured hemoglobin aggregates (Heinz bodies) within erythrocytes following exposure to an oxidant and subsequent erythrocyte lysis.[6] Oxidants include certain drugs, chemicals, heavy metals and plants, including *Allium* spp. such as garlic, leeks and onions.[6] Clinical signs may comprise anorexia, vomiting, diarrhea, lethargy, pale or icteric mucous membranes, hemoglobinuria and death.[1-5] Heinz bodies are identified on blood smears as rounded structures which are pale pink if Wright's stain is used, or dark blue with new methylene blue stain, within the erythrocyte cytoplasm, protruding from the cell membrane, or as free bodies in the smear background.[6] Other oxidative changes to erythrocytes include eccentrocytosis, pyknocytosis, and ghost cells.[6] Treatment involves oxidant removal, if possible, and supportive care.[5] Blood transfusion is indicated if clinical signs of anemia are significant.[5,7]

The aim of this report is to describe a case of Heinz body hemolytic anemia in a South American coati (*Nasua nasua*) associated with leek ingestion, managed with a whole blood transfusion and supportive care. To the authors' knowledge this is the first reported case of Heinz body hemolytic anemia, suspected leek toxicity and blood transfusion in this species.

Case Description

A 3 year 4 month old, 4.1 kg, male, South American coati (*Nasua nasua*) presented for a post-operative check 24 hours after partial digit amputation due to a phalangeal fracture. Clinical examination at the time of surgery had revealed dorsolateral displacement of digit four of the right forelimb, with purulent discharge at the nail bed, and moderately pale mucous membranes. Surgery and anesthesia had been unremarkable. A blood sample had been collected for hematology and biochemistry analysis and full body radiographs showed no abnormalities other than the phalangeal fracture. Meloxicam[a] (0.3 mg/kg, subcutaneously), buprenorphine[b] (0.01 mg/kg, intramuscularly

(IM)) and amoxicillin-clavulanate[c] (15 mg/kg, subcutaneously) had been administered intra-operatively and meloxicam[d] (0.1 mg/kg orally, once every 24 hours) and clindamycin[e] (11 mg/kg, orally, once every 24 hours) dispensed to continue from 24 hours post-operatively. At the post-operative check, the coati was slightly quiet and had regurgitated. Soaked pellets were offered 3-4 hours later, which was followed by vomiting. The coati became progressively quieter and 3-4 hours later presented lethargic, dyspneic and tachypneic, in a sitting position with the head lowered in the corner of its enclosure. Blood results from the previous day became available and were compared to global reference intervals (RI) derived from serum or plasma samples collected from between 15 and 100 zoo-housed South American coatis which were recorded with a normal health status at the time of blood sampling.[8] Hematology revealed marked anemia (PCV 0.110 L/L (11.0%); RI 0.277 – 0.508 L/L (27.7 – 50.8%)) with moderately hemolyzed plasma (Table 1). On blood smear examination (Modified Wright's stain), numerous erythrocytes contained Heinz bodies, which were typically large (1 - 2 μm), protruding, and often multiple per cell (Figure 1); brilliant cresyl blue staining demonstrated Heinz bodies in 100 % of erythrocytes, with up to 15 per cell (Figure 2). Ghost cells and eccentrocytes were present in moderate numbers, often with attached Heinz bodies (Figure 1). No polychromasia was noted. Complete blood count performed using a hematology analyzer[f] revealed a mildly increased mean corpuscular hemoglobin concentration (487 g/L (48.7 g/dL); RI 282 – 391 g/L (28.2 - 39.1 g/dL)). Mild neutrophilia (18.95 x 10^9 cells/L (18.95 x 10^3 cells/μL); RI 1.796 – 14.715 x 10^9 cells/L (1.796 – 14.715 x 10^3 cells/μL)) was observed, although the neutrophil count was similar to values reported in wild South American coatis with a low parasite load and may be within normal limits.[9] Moderate numbers of neutrophils displayed mild cytoplasmic foaminess, occasional basophilia, and low numbers of Döhle bodies (Figure 1). Moderate numbers of reactive lymphocytes were present. Biochemistry performed on heparinized plasma using a biochemistry analyzer[g] revealed mildly increased bilirubin (18.0 μmol/L (1.05 mg/dL); RI 0.70 - 12.0 μmol/L (0.00 – 0.70 mg/dL)) and urea (11.4 mmol/L (31.9 mg/dL); RI 1.90 - 9.30 mmol/L (5.40 – 26.0 mg/dL)).

Glutamate dehydrogenase was also elevated compared to RI in other domestic carnivores (56.0 U/L; dog RI < 6.0 U/L; cat RI 0.0-0.4 U/L; ferret RI 0.0-2.5 U/L).[10-12]

A thorough husbandry review with keepers revealed that between five and ten grams of leeks (*Allium ampeloprasum*) had been added to the diet of this coati and one other animal of the same species (conspecific) which shared its enclosure up to four times daily for two to five days prior to initial presentation, defined as the day when partial digit amputation was performed and the initial blood sample was collected. The maximum total leek intake was therefore 200 grams, equivalent to 48.8 g/kg body weight. There was no known exposure to other oxidants associated with Heinz body anemia.[6] Mean corpuscular hemoglobin concentration was likely falsely increased due to hemoglobinemia and the presence of Heinz bodies, while hyperbilirubinemia was also a reflection of increased bilirubin production in the face of hemolysis.[13] Neutrophilia with mild to moderate toxicity and reactive lymphocytes indicated an inflammatory response and antigenic stimulation, likely associated with the nail bed infection. Mildly increased urea was suggestive of pre-renal azotemia or protein catabolism.

The coati was placed in an oxygen chamber. Tachypnea and dyspnea improved but worsened when oxygen therapy was discontinued. Given the severity of anemia and associated clinical signs, a blood transfusion was performed. A young, healthy, vaccinated, male neutered, full sibling conspecific with no history of ill health, nor access to leeks, was selected as a blood donor. Both were anesthetized, the recipient induced with 8% sevoflurane[h] delivered in 100% oxygen via facemask, intubated and maintained with 4-5% sevoflurane[h] in 100% oxygen, and the donor induced with medetomidine[i] (0.05 mg/kg, IM) and ketamine[j] (3.92 mg/kg, IM), intubated and maintained with 2% isoflurane[k] in 100% oxygen. The PCV of the recipient and donor was 0.100 L/L (10%) and 0.300 L/L (30%) respectively. The blood collection and administration sites were aseptically prepared. Intravenous (IV) catheterization was initially unsuccessful in the donor coati therefore medetomidine was reversed with atipamezole[l] (0.25 mg/kg, IM) to resolve presumed medetomidine-

induced hypotension. Subsequently fifty milliliters of blood were collected from the donor coati via IV catheters in the lateral saphenous and jugular veins into syringes containing citrate-phosphate-dextrose-adenine (CPDA) in a 1:7 ratio of anticoagulant to whole blood. A combination of 2.5 ml syringes containing 0.31 ml of CPDA and 2.19 ml of whole blood, 5 ml syringes containing 0.63 ml of CPDA and 4.37 ml of whole blood and 10 ml syringes containing 1.25 ml of CPDA and 8.75 ml of whole blood were collected. These were transfused to the recipient via an IV catheter in the lateral saphenous vein in boluses at an initial rate of 1 ml/kg/hour, increasing to 3 ml/kg/hour after 15 minutes then doubled every 15 minutes to reach a maximum rate of 24 ml/kg/hour. Respiratory rate, heart rate and body temperature were monitored continuously and were stable. A crystalloid solution[m] (10 ml/kg/hour, intravenously) was administered via a separate IV catheter in the cephalic vein throughout the procedure. The recipient vomited once on recovery from anesthesia; otherwise the donor and recipient recovered uneventfully.

Provision of leeks was discontinued and a conspecific which shared an enclosure with the coati examined visually. No clinical signs of anemia were observed. During the subsequent 24-hour period, the recipient coati was isolated in an enclosure with minimal furniture to reduce activity levels. The coati was brighter than prior to transfusion, its mucous membranes were less pale and it was no longer dyspneic or tachypneic at rest; however, the respiratory rate increased from 20 to 40-50 breaths per minute on exertion. The coati appeared comfortable using its right forelimb therefore meloxicam was discontinued. Over the subsequent 24 hours, the coati was bright, eating small amounts and showed no dyspnea or tachypnea. Respiratory rate was stable at 28 breaths per minute when mixed with conspecifics in its normal environment. A free-catch urine sample was collected. No abnormalities were observed on dipstick[n] or microscopy and the specific gravity was 1.005. On days three and four following transfusion, the coati was brighter and climbing normally, with no dyspnea or tachypnea. Mucous membranes remained slightly paler than those of conspecifics. The coati was eating a normal amount, although slightly slower than usual, and was sleeping lower down

in the enclosure than normal. On day six following transfusion, the coati had returned to normal behavior and the digit amputation site had healed, therefore antibiotics were discontinued.

Four weeks after initial presentation, a blood sample was collected under general anesthesia to reassess anemia. Anemia had resolved (PCV 0.34 L/L (34%)) (Table 1). There were occasional eccentrocytes (< 1 per 100 x hpf), indicating minimal residual oxidative injury. Mild macrocytosis (approx. 1 – 10 per 100 x hpf) and moderate codocytosis (approx. 11 – 20 per 100 x hpf) were present, consistent with erythrocyte regeneration. Moderate numbers of schistocytes (approx. 3 – 8 per 100 x hpf) and low numbers of keratocytes (approx. 3 – 5 per 100 x hpf) were noted for the first time, indicating erythrocyte fragmentation. Neutrophilia and neutrophil toxicity had resolved; however reactive lymphocytes were still present, suggestive of mild antigenic stimulation. Serum biochemical analytes were within the RI for zoo-housed coatis.[8]

Eight weeks after initial presentation, a health examination was performed under general anesthesia to reassess erythrocyte fragmentation. There were very rare Heinz bodies present, indicating minimal residual oxidative injury, and mild macrocytosis (approx. 1 - 10 per 100 x hpf) and codocytosis (approx. 5 – 10 per 100 x hpf) were observed, consistent with erythrocyte regeneration (Table 1). On serum biochemistry profile, glutamate dehydrogenase was elevated compared to the initial blood sample and compared to RI in other domestic carnivores (99.0 U/L; dog RI < 6.0 U/L; cat RI 0.0-0.4 U/L; ferret RI 0.0-2.5 U/L).[10-12] However, there are no published RI for coatis, other hepatic parameters were within normal limits and no clinical signs of hepatic disease were evident. Echocardiography and abdominal ultrasound examination showed no abnormalities. One year following initial presentation, the coati remains clinically well.

Discussion

To the authors' knowledge, this is the first reported case of Heinz body hemolytic anemia in a South American coati (*Nasua nasua*). *N. nasua* is a member of the Family Procyonidae, Superfamily

Canoidea and Order Carnivora therefore the ferret and dog represent its closest domestic relatives.[14] Heinz body hemolytic anemia has previously been described in dogs, cats, horses, ruminants, and a ferret, and has rarely been reported in captive or free-ranging wild animals, including Atlantic puffins, common marmosets, cotton-top tamarins, herring gulls, koalas, murres, a red panda and a river otter.[1-3,6,15-18] Heinz bodies have been described in the erythrocytes of healthy cats and several wild mammals, but not in healthy coatis.[1,6,19-21] Heinz bodies can cause hemolytic anemia via: 1) trapping and lysis of affected erythrocytes in the spleen as they are less deformable; 2) lysis of affected erythrocytes within blood vessels as they are fragile due to binding of Heinz bodies to the cell membrane or oxidative damage of membrane proteins; and 3) phagocytosis of affected erythrocytes by splenic or hepatic macrophages as antigens form on their cell membrane when hemichromes attach to erythrocyte membrane band 3 proteins.[6]

Leeks (*Allium ampeloprasum*) are considered the likely oxidant which caused Heinz body formation in this case. *Allium* spp. contain organosulfur compounds which are absorbed across the gastrointestinal tract and metabolized to form highly active oxidants.[5] They are not described in the wild diet of *N. nasua*, nor are they offered as part of the captive diet in zoos.[14,22] In two previously reported cases of Heinz body hemolytic anemia associated with *A. ampeloprasum* ingestion in a domestic shorthair cat and cattle, *A. ampeloprasum* was offered for eight or ten days; however, the amount consumed was unknown.[23,24] The toxicity of onions (*Allium cepa*) in dogs and cats has been evaluated. Consumption of 5 g/kg of onions in cats or 15-30 g/kg in dogs can cause clinically important hematologic changes and ingestion of 0.5% of an animal's body weight in onions at one time is consistently associated with toxicity.[5] A high single dose or smaller doses over several days may result in Heinz body anemia.[5] Up to 48.8 g/kg body weight of leeks had been added to the diet over two to five days prior to initial presentation therefore the dose of leeks offered to the coati may be associated with toxicity. However, species susceptibility is highly variable, dogs and particularly

cats being highly susceptible, mice, rats, goats and sheep far less susceptible and humans being more resistant.[5]

Other oxidants associated with Heinz body hemolytic anemia include drugs (paracetamol [acetaminophen], aspirin, benzocaine, cetacaine, ecabapide [DQ-2511], methylthioninium chloride [methylene blue], phenacetin, phenazopyridine, phenothiazine, propofol), heavy metals (copper, zinc), other chemicals (methionine, naphthalene, oil, phenylhydrazine, propylene glycol, skunk musk, vitamin K_3), and plants (*Brassica* spp., wilted red maple (*Acer rubrum*) leaves, possibly other types of maple and other *Allium* spp. including Catalan spring onions, Chinese chive, garlic and onions).[4,6,15,17,25] Increased Heinz body formation and reduced PCV has also been associated with skeletal muscle myopathy due to vitamin E, selenium and protein deficiency in common marmosets, and secondary to diabetes, hyperthyroidism and lymphoma in cats.[6,16] No exposure to these oxidants, nor evidence of these diseases, were identified in this case.

Most of the clinical signs in this case were similar to those previously associated with Heinz body anemia.[1-5] Gastrointestinal signs tend to occur first after ingestion of *Allium* spp., and clinical signs associated with anemia take several days to develop, with the most severe being 5-6 days post-ingestion.[5,26] As expected, the most severe signs in the coati were observed 2-5 days after leeks were added to the diet. However, several typical clinical signs were not noted, such as anorexia, diarrhea, icterus and hemoglobinuria.[1-5] Absence of hemoglobinuria and icterus was likely due to hemolysis not being sufficiently severe. An unusual feature of this case was that Heinz body anemia was identified on hematology performed during assessment of a phalangeal fracture. It is possible that the coati was experiencing weakness associated with anemia, which may have predisposed it to falling and fracturing a digit; however, we suspect that the phalangeal fracture and Heinz body anemia were unrelated as it is unlikely that there would have been sufficient time for purulent discharge to develop at the nail bed in association with the fracture if the fracture occurred following development of anemia.

The clinicopathologic features of Heinz body anemia in this case were similar to those previously described, including anemia, hyperbilirubinemia, Heinz bodies, eccentrocytes and ghost cells.[6] However polychromasia and reticulocytosis were absent, likely due to insufficient time for development of a regenerative response or dampening of regeneration by the concurrent inflammation.[6] Furthermore, reticulocytes may have been missed on the brilliant cresyl blue stained smear due to the overwhelming presence of Heinz bodies hindering a thorough search for similarly staining aggregates of ribosomal material. Bilirubinuria, hemoglobinemia and hemoglobinuria were also absent, likely due to hemolysis not being sufficiently severe.[6] Methemoglobinemia is often observed in Heinz body anemia cases; however, methemoglobin levels were not measured in this case.[6] Heinz bodies may be identified on a fresh blood smear stained with Romanowsky stains; however, they are best seen on blood smears stained with new methylene blue or brilliant cresyl blue stain.[9] Heinz bodies vary in size, from 1-2 μm in dogs, guinea pigs and rabbits to one third of erythrocyte diameter in cats, and the total mass of precipitated hemoglobin is indicative of the severity of the toxic injury.[13] Heinz bodies in the coati of this report were large and numerous, consistent with a severe toxic insult.[13] More frequent monitoring of erythrocyte parameters and physical examination could have provided useful information on the resolution of Heinz body hemolytic anemia in this case; however, this was not pursued as anesthesia would have been required for each follow-up examination and sample collection due to the non-domesticated nature of this animal.

Management of Heinz body hemolytic anemia involves removal of the oxidant, if possible, and supportive care. If oxidant ingestion occurred within 2 hours of presentation, unlike in this case, vomiting can be induced and activated charcoal administered following emesis.[5] Intravenous fluid therapy is recommended to prevent hemoglobin nephrosis and replace losses from vomiting and diarrhea.[5] This would not have been tolerated conscious therefore it was only performed during the anesthetic period. Supplemental oxygen therapy may be required if anemia is severe and a blood

transfusion is indicated if the PCV is less than 0.100 L/L (10%), if the PCV has dropped rapidly to less than 0.200 L/L (20%) in dogs or 0.150 L/L (15%) in cats or if the animal is showing significant clinical signs of anemia, as observed in this case.[5,7]

A donor coati was chosen following several of the criteria for donor dogs and exotic small mammals, being young (3 years 4 months old), neutered, male, fully vaccinated, in good body condition, with no signs of ill health or history of infectious disease, and of the same genus and species as the recipient.[27,28] The donor was also chosen as it had had no access to leeks. The donor coati was 5 kg which is slightly higher than the median body weight derived from 146 zoo-housed South American coatis aged 3-4 years old and its PCV measured under anesthesia immediately prior to transfusion was 0.300 L/L (30%) which is within the RI derived from 100 healthy zoo-housed South American coatis (0.277 – 0.508 L/L (27.7 – 50.8%)).[8,29] It is unknown whether erythrocyte antigens exist in coatis; cross-matching prior to transfusion might have provided useful information and is recommended for exotic small mammals due to lack of knowledge regarding blood types.[28]

Approximately 10% of total blood volume can be collected from donor dogs with no expected adverse effects, therefore 50 ml of whole blood was obtained from the 5 kg donor coati in this report.[27] Assuming coatis have a similar blood volume to dogs, 50 ml of whole blood with a PCV of 0.300 L/L (30%) should increase the recipient PCV from 0.100 L/L (10%) to approximately 0.141 L/L (14.1%).[7] CPDA was used as this is the anticoagulant of choice for small animal blood transfusions and a gradual increase in the transfusion rate from 1 ml/kg/hour with continuous monitoring performed to reduce the risk of transfusion reactions.[27]

Other than vomiting on recovery from anesthesia, no acute adverse transfusion reactions were noted.[27] The blood transfusion was successful at immediately resolving clinical signs of lethargy, dyspnea and tachypnea at rest. Complete clinical recovery to normality was also more rapid than that described in dogs with experimentally-induced onion toxicity which did not receive a whole

blood transfusion.[26] Resolution of anemia was complete by four weeks after initial presentation, as described in dogs with experimentally-induced onion toxicity.[26] The appearance of schistocytes and keratocytes at four weeks, indicative of erythrocyte fragmentation, is unexplained. Causes in dogs and cats include vascular abnormalities, turbulent blood flow, microvascular injury, disseminated intravascular coagulation, dyserythropoiesis, hepatic disease, glomerulonephritis, hemangiosarcoma, myelofibrosis and chronic doxorubicin administration.[30] Formation of microthrombi in the whole blood used for transfusion and subsequent microvascular damage may have been responsible.[27] Use of a blood administration set and/or blood filter is recommended for blood transfusions in human and veterinary patients to remove microthrombi and could have reduced the post-transfusion RBC morphologic changes seen in this case.[27]

Conclusion

The authors describe successful management of Heinz body anemia in a South American coati (*Nasua nasua*) secondary to suspected leek (*Allium ampeloprasum*) toxicosis. Administration of a whole blood transfusion from a conspecific resulted in rapid resolution of clinical signs and the coati made a full recovery.

Footnotes

[a] Metacam® 5 mg/ml solution for injection for dogs and cats, Boehringer Ingelheim Vetmedica GmbH, Ingelheim am Rhein, Germany.

[b] Buprecare® 0.3 mg/ml solution for injection for dogs and cats, Animalcare Ltd, York, United Kingdom.

[c] Amoxycare LA Suspension for injection 15% w/v, Norbrook Laboratories Limited, Newry, Northern Ireland.

[d] Metacam® 1.5 mg/ml oral suspension for dogs, Boehringer Ingelheim Vetmedica GmbH, Ingelheim am Rhein, Germany.

[e] Antirobe® Capsules, Zoetis, London, United Kingdom.

[f] ADVIA 2120i, Siemens Healthcare, Surrey, United Kingdom.

[g] ILab600, Instrumentation Laboratory, Munich, Germany.

[h] SevoFlo® 100% w/w Inhalation vapor, liquid for dogs, Zoetis, London, United Kingdom.

[i] Sedastart, Le Vet B.V., Oudewater, The Netherlands.

[j] Vetalar® V 100 mg/ml Solution for injection, Zoetis, London, United Kingdom.

[k] Isocare 100% w/v Inhalation vapor, liquid, Animalcare Ltd, York, United Kingdom.

[l] Sedastop, Le Vet B.V., Oudewater, The Netherlands.

[m] Aqupharm 11 (Hartmann's) Solution for Infusion, Animalcare Ltd, York, United Kingdom.

[n] Multistix® 10 SG Reagent Strips for Urinalysis, Siemens Healthcare Diagnostics Inc., Camberley, United Kingdom.

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345

346 Table 1. Peripheral blood erythrocyte parameters in a South American coati (*Nasua nasua*) with
347 severe Heinz body hemolytic anemia at initial presentation (Day 0) and at follow up examinations
348 27 and 62 days following initial presentation.

Erythrocyte parameter	Reference intervals	Test result		
		Day 0	Day 27	Day 62
RBC count, x 10^{12} cells/L (x 10^{6} cells/μL)	3.88 - 8.10 (3.88 - 8.10)	2.68 (2.68)	5.35 (5.35)	5.96 (5.96)
Hemoglobin, g/L (g/dL)	88.0 - 151.0 (8.8 – 15.1)	65.0 (6.50)	109.0 (10.90)	110.0 (11.00)
MCV, fL	39.7 - 71.2	49.7	55.2	59.4
MCH, pg	13.6 - 24.7	24.3	20.4	18.5
MCHC, g/L (g/dL)	282 – 391 (28.2 – 39.1)	487 (48.7)	371 (37.1)	312 (31.2)
RDW, %	N/A	21.1	19.8	17.1
PCV, L/L (%)	0.277 - 0.508 (27.7-50.8)	0.11 (11)	0.34 (34)	0.34 (34)
Heinz body presence	-	Numerous	Absent	Very rare
Eccentrocytosis	-	3 – 8 per 100 x hpf	< 1 per 100 x hpf	Absent
Ghost cells	-	3 – 8 per 100 x hpf	Absent	Absent
Schistocytosis	-	Absent	3 – 8 per 100 x hpf	Absent

Keratocytosis	-	Absent	3 ‑ 5 per 100 x hpf	Absent
Codocytosis	-	Absent	11 - 20 per 100 x hpf	11 ‑ 20 per 100 x hpf
Macrocytosis	-	Absent	1 – 10 per 100 x hpf	1 – 10 per 100 x hpf
Hemolysis	-	Moderate	Mild	Absent

MCV = mean cell volume, MCH = mean corpuscular hemoglobin, MCHC = mean corpuscular hemoglobin concentration, RDW = red cell distribution width, N/A = not available. Values for RBC count, hemoglobin, MCHC and PCV are given in Système International units followed by conventional units in parentheses. Hemolysis was assessed based on the color of the plasma. Reference intervals are derived from serum or plasma samples collected from between 15 and 100 zoo-housed South American coatis which were recorded with a normal health status at the time of blood sampling.[8]

356 Figure legends

357 Figure 1. Blood smear from a South American coati (*Nasua nasua*) with severe Heinz body hemolytic
358 anemia. Note the large, protruding Heinz bodies (thick arrows), eccentrocytes (thin arrows),
359 eccentrocyte with attached Heinz body (double arrow), ghost cells with attached Heinz bodies
360 (arrowheads), and a neutrophil containing Döhle bodies. Modified Wright's stain, 100x objective, oil
361 immersion. Bar = 10 μm.

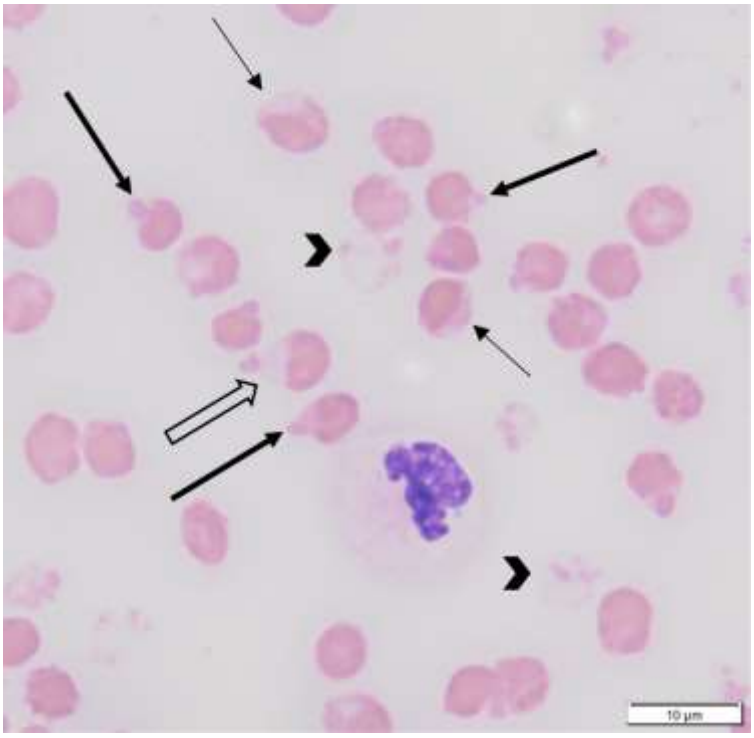

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Figure 2. Blood smear from a South American coati (*Nasua nasua*) with severe Heinz body hemolytic anemia. Note multiple, dark blue staining Heinz bodies within and protruding from erythrocytes (thick arrows) and attached to ghost cells (thin arrows). Brilliant cresyl blue, 100x objective, oil immersion. Bar = 10 µm.