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CRITICAL CARE FOR A HYPOTHERMIC AND HYPOGLYCEMIC WHITE RHINOCEROS (*CERATOTHERIUM SIMUM SIMUM*) CALF

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Abstract: A 3-day-old, 57.3-kg, male white rhinoceros (*Ceratotherium simum simum*) neonate presented laterally recumbent with comatose mentation, cold extremities, and severe hypothermia, hypoxemia, and hypoglycemia. Critical care support was initiated with aggressive fluid and warming support as well as dextrose and oxygen supplementation. After initial stabilization, additional complications arose in subsequent weeks including intermittent diarrhea, dry skin with loss of epidermal layers, urticaria on the head, and superficial wounds on the face, ears, feet, and penis. There is a lack of critical care information for rhinoceros calves. However, due to similarities to other Perissodactylids, some general guidelines for critical equid neonatal care were implemented. The calf was hand-raised until weaning and is now a subadult white rhinoceros with no abnormalities.

Key words: *Ceratotherium simum simum*, critical care, hypoglycemia, hypothermia, neonate, white rhinoceros.

BRIEF COMMUNICATION

A 3-day-old, 57.3-kg, male white rhinoceros (*Ceratotherium simum simum*) neonate presented laterally recumbent with comatose mentation, cold extremities, and severe hypothermia (27.8°C, adult reference interval 36.6–37.2°C).[3] The calf appeared dehydrated with tacky mucous membranes and decreased skin turgor. The mucous membranes were a deep reddish-purple color. The dam was multiparous and gestation length was normal. The parturition was during the night or early morning in January and was not observed. The calf had been seen nursing in days prior and nursing or attempting to nurse on the morning of presentation. Hematology, serum biochemistry, and arterial blood gas (auricular artery) evaluation revealed several values outside of the normal range reported for adult or subadult white rhinoceroses (Table 1). Considering the severe abnormalities, the calf was given critical care over the following 72 hr.

Hypothermia was addressed using a water-heating pad (37°C), a forced-air warmer (Bair-Hugger®, Arizant Healthcare Inc., Eden Prairie, Minnesota 55344, USA), blankets, and warmed isotonic intravenous fluids (37°C, 2.5% dextrose in NaCl 0.45%, and 2.5% dextrose in lactated ringer's solution) supplemented with 20 mEq potassium chloride/L bolused at approximately 833 mL/hr. After approximately 6 hr, rectal temperature was 37°C. The calf, however, was not thermally stable and required continual thermal support (including warmed ambient air 32.2°C using forced air heaters) for the ensuing 48 hr. Subsequently, the rhinoceros was slowly weaned from thermal support but was housed indoors (20–23°C).

Oxygen support was used to correct the hypoxemia. Preputial pulse oximetry readings ranged from 90–95% and 87–90% with and without oxygen supplementation, respectively (100% oxygen, administered via mask). Oxygen was supplemented for approximately 12 hr before it was no longer required (monitored by pulse oximetry) or tolerated. Sternal recumbency was maintained when possible. After the first 12–24 hr, the calf began ambulating, although ataxic and lethargic, and was more alert.

Initially, hypoglycemia was controlled using 5% dextrose in water, 2.5% dextrose in NaCl 0.45%, and 2.5% dextrose in lactated ringer's solution intravenously with intermittent boluses of 50% dextrose in water every 1–2 hr during the first 6 hr (blood glucose ranged 14–64 mg/dl) with empirical antibiotic therapy (ceftiofur 11 mg/kg intravenously every 12 hr for 6 days, amikacin 5 mg/kg intravenously or subcutaneously every 12 hr for 8 days). Because of persistent hypoglycemia, 5–10% dextrose in NaCl 0.45% was continued at approximately 83 mL/hr for the subsequent 12 hr (rate variable due to calf position and activity). Under general anesthesia, a nasogastric feeding (NG) tube was placed. Endotracheal intubation was performed for airway protection and for facilita-

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Table 1. Hematologic, chemistry, and arterial blood gas data from a critical neonatal white rhinoceros (*C. s. simum*).

Blood parameters	Presentation	Day 2	Day 3	Day 4	Day 9	Day 15	Adult-subadult reference range[3,8]
PaO_2 (mm Hg)	55	—	—	—	—	—	90.2–108.6
$PaCO_2$ (mm Hg)	48	—	—	—	—	—	44.4–53.7
pH	7.43	—	—	—	—	—	7.346–7.382
Hematocrit (%)	58.5	52.0	50.0	51.5	46.5	33.9	27.2–47.4
White blood cells (10^3 cells/μl)	10.32	14.60	17.24	15.16	15.10	6.66	4.86–13.87
Mature neutrophils (10^3 cells/μl)	7.43	12.56	14.83	12.73	10.27	4.28	0.88–9.03
Band neutrophils (10^3 cells/μl)	1.14	1.31	1.21	0.76	0.30	0.20	0.00–0.19
Band neutrophils (%)	11	9	7	5	2	3	N/A
Blood glucose (mg/dl)	11	43	25	18	—	—	48–149
Total protein (g/dl)	8.2	6.6	6.9	5.2	—	—	4.8–8.8
Globulins (g/dl)	5.1	4.1	4.5	2.8	—	—	N/A
BUN (mg/dl)	38.7	39.2	44.3	28.9	—	—	2.0–22
Creatinine (mg/dl)	0.36	0.46	0.45	0.37	—	—	N/A
Sodium (mEq/L)	131.4	138.8	—	—	—	—	125–139
Potassium (mEq/L)	3.74	2.85	—	—	—	—	3.5–4.9
Chloride (mEq/L)	97.3	103.4	—	—	—	—	87–98

tion of NG tube placement. A lateral radiograph confirmed appropriate placement and also revealed a mild, mixed alveolar to interstitial pattern in visible lung fields. Milk formula (27 : 9 : 1 : 1 nonfat cow's milk : low fat cow's milk : dextrose : - water at 8–15% body weight/day gradually increased)[1,15] and electrolyte solution (Calf Quencher, Vedco Inc., St. Joseph, Missouri 64507, USA) (5% body weight/day and gradually weaned over 1 wk) was given for fluid and nutritional support. At 48–72 hr the calf became more responsive, with less-frequent hypoglycemia (18–109 mg/dl—measured with hand held glucometer Accu-Chek Advantage [Boehringer Mannheim, Indianapolis, Indiana 46256, USA] using Accu-Chek Comfort Curve test strips [Roche Diagnostics, Indianapolis, Indiana 46256, USA]). At 72 hr, the calf began bottle-feeding voluntarily; intravenous fluids and nasogastric feedings were discontinued.

After initial stabilization, additional complications arose in subsequent weeks including intermittent diarrhea, dry skin with loss of epidermal layers, urticaria on the head, and superficial wounds on the face (Fig. 1), ears, feet, and penis. The diarrhea was treated with di-tri-octahedral smectite (Equine Biosponge®, Platinum Performance, Inc., Buellton, California 43427, USA) orally every 12 hr, as well as Probios (Vets Plus, Inc., Menomonie, Wisconsin 54751, USA) gel orally every 24 hr. Trimethoprim-sulfamethoxazole (20 mg/kg orally every 12 hr for 10 days) was started as broad spectrum coverage following parenteral antimicrobials for possible pneumonia or sepsis. Skin and penis wounds were treated topically with neomycin-polymyxin B-bacitracin and vitamin A and D ointments (applied every 8 hr) and resolved without complication. The urticaria (unknown cause) resolved with 4 days of diphenhydramine (2 mg/kg orally every 8 hr). During the first week, the calf gained 4 kg and continued to gain weight at approximately 0.5 kg/day for the first month. The calf was hand-raised until weaned and is now a subadult white rhinoceros with no abnormalities.

The paucity of critical care information for rhinoceros calves made implementation of care challenging. Limited reports of white rhinoceros

Figure 1. Facial wounds and epidermal loss in a neonatal white rhinoceros (*C. s. simum*).

neonates include those of patent urachus, decubitus ulcers, disseminated intravascular coagulation, and neosporosis.[5,10,17,23] However, as Perissodactylids, they share similar features and are best compared with members of that order, notably the domestic horse.[15] For example, rhinoceroses are herbivorous, hind-gut fermenters with well-developed ceca. Hematologic and serum chemistry values are also similar to domestic horses. Some general guidelines for critical equid neonatal care were implemented in this case. Adult and subadult blood values were used for comparison, as no specific reference intervals for neonates of this species exist. For this reason it is difficult to interpret the blood values and note should be taken that, for other Perissodactylids (i.e., domestic horses), extensive research has found significant differences between neonate and adult reference intervals.

A cold environment, prematurity, sepsis, and metabolic disease such as hypoglycemia can predispose equid neonates to hypothermia,[6,14,18,20] with large surface area to volume ratio and minimal insulation exacerbating this predisposition.[6,7,18] Although not studied, white rhinoceros calves likely have similar predispositions to heat loss, with relatively large body size (approximately 50 kg) at birth. Unexpectedly low environmental temperatures (mean of 3.8°C from means of 11.1°C in the days prior to birth) around the time of birth likely contributed to the neonate's hypothermia. Mean temperature in January in our geographic area is approximately 11.5°C. White rhinoceroses can be born throughout the year in southern Africa, so temperatures can vary widely. White rhinoceroses are adapted to tropical climates,[15] which may diminish their ability to withstand lower ambient temperature.

The dam was found to have vesicular lesions and swelling of her teats, making it difficult for the calf to nurse, which had also occurred during previous lactations. Diagnostics (e.g., virus isolation, PCR) have failed to determine an etiologic agent. The authors suspect this factor contributed to hypoglycemia, hypothermia, and dehydration. Globulins were elevated, suggesting adequate intake of colostrum and maternal antibody transfer. However, further testing to assess passive transfer of immunity was not done in this case. Although not commonly performed in rhinoceros neonates, protein electrophoresis has been reported in a black rhinoceros (*Diceros bicornis*) neonate to assess passive transfer of immunity.[19] Qualitative and quantitative tests for failure of passive transfer of immunity have also been used in white rhinoceros neonates but have not been validated.[5]

In domestic large and small animals, hypothermia causes respiratory depression with hypoventilation leading to hypoxia and respiratory acidosis.[6,16,20] As body temperature increases and shivering recommences, muscle greatly contributes to oxygen consumption and hypoxia.[9] Shivering was observed in this neonate during the rewarming period. This, combined with lateral recumbency, may have contributed to hypoxia due to decreased chest wall expansion, as seen in foals.[14] The mixed alveolar to interstitial pattern seen on radiographs may have indicated pneumonia, pulmonary edema, or atalectasis from hypoventilation or recumbency. Hypothermia can be a cause of pulmonary edema in other species.[16,18] Hypothermia also decreases immune function with increased incidence of infection.[6,9,16] Leukocytosis characterized by neutrophilia with a left shift is indicative of an inflammatory leukogram which could indicate pneumonia or other systemic inflammation. Follow-up radiographs were not performed in this case due to response to therapy and an inability to perform radiographs without chemical restraint because of physical limitations.

Hypothermic foals are prone to hypoglycemia due to limited energy reserves, a condition exacerbated by exposure to low environmental temperatures.[6,14] Isotonic fluids with 5–10% dextrose administered as a constant rate infusion (CRI) are recommended for treatment of hypoglycemia.[2,4,13,14,22] Some sources recommend using 50% dextrose,[4] but risks include hyperglycemia with rebound hypoglycemia, phlebitis, and perivascular tissue damage if extravasation occurs.[12,13,21] Because of the severity of hypoglycemia in this case, 50% dextrose boluses followed by 2.5–10% dextrose CRI were used. Extravasation of dextrose and cold exposure–frostbite may have contributed to the rhinoceros' skin wounds, as auricular and radial veins were catheterized. The highly vascular makeup of rhinoceros skin may make them more prone to ischemic lesions.[5] Further research in normal rhinoceros neonates is warranted for determination of normal glucose values and best methods of dextrose supplementation.

Hemodynamic shock in hypothermic domestic large animal neonates may result in severe anoxic changes in the bowel wall and cause severe diarrhea, sloughing of gastrointestinal mucosa, or clostridial overgrowth in the bowel.[20] Diarrhea may be caused by bacterial or parasitic infection, stress, or formula intolerance in hand-reared rhinoceros calves or domestic foals.[1,11] Feces were evaluated for parasites (via direct fecal smear and fecal flotation with sodium nitrate and zinc sulfate) in this rhinoceros calf, but no abnormalities were noted and the calf was responsive to treatments used.

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