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STOMACH RUPTURE AND TORSIO COLI IN AN OLD MALE WHITE RHINOCEROS (*Ceratotherium simum simum*)

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Abstract

An old white rhinoceros (*Ceratotherium simum simum*) suffered from exfoliative dermatitis during his live. The last months he lost weight despite of good appetite. At the end of July he showed signs of pain and was treated with analgesics. He died from the consequences of peritonitis due to a ruptured stomach and a torsio coli. The animal showed extensive lesions of the teeth, correlated with old age.

Key words: Rhinoceros, *Ceratotherium simum simum* , stomach rupture, peritonitis, dental disease.

Clinical history

A captive born, male, white rhinoceros (*Ceratotherium simum simum*) imported from South Africa (Pretoria National Zoo Gardens), and exhibited in the Antwerp Zoo since 1974, developed skin lesions in 1993. Malassezia pachydermatitis and Candida paraspilosis turned out to be responsible for an exfoliative dermatitis, which was successfully treated with natamycin. Another female white rhino living together with this animal never showed any symptoms of this exfoliative dermatitis (1).

The male animal with the name Balthazar moved to Safari Beekse Bergen, Hilvarenbeek, The Netherlands, in November 1995. There he lived together with ten other white rhinoceroses. Only he developed periodically signs of this specific exfoliative dermatitis, which every time was successfully treated using topical cod oil with enilconazole. It was at that time postulated that this animal could be immune-depressed in some sort of way. In the years following he fathered three young. One male offspring also developed skin lesions and was treated in several ways, including different antibiotics. This young animal finally died 6 months old due to an endocarditis, probably caused by haematogenic transport of bacteria out of the extensive skin lesions. The other two offspring's, both female, never showed any skin problems.

Besides the skin problems Balthazar was a healthy normal white rhinoceros. Tetanus vaccinations and occasional slight hoof problems with treatment are the only remarks in his medical report until the summer of 2001. Fecal examinations were always negative and reproduction behavior occurred. In August 2001 he developed a unilateral nasal discharge. The discharge was clear but sometimes contained some blood. Early September the animal was treated orally for five days with trimetoprimsulfamethoxazole. Bacteriological examination of the discharge revealed beta-hemolytic streptococci, which were not susceptible for this antibiotic.

Blood examination showed no irregularities. The animal was treated with 4 grams of ceftiofur once a day i.m. by dartgun for 24 days. On day 14 the animal showed no longer signs of

discharge. In October and November the same year 2001 the nasal discharge occurred again and the animal was treated with 4 grams ceftiofur respectively for 5 and 14 days.
Until July 2002 the animal was fine. At that time there was only clear discharge reported for a few days. In September however the discharge occurred again and contained blood. Beta-haemolytic streptococci, sensitive for ceftiofur were cultured. The animal was again treated for nine days with ceftiofur and recovered.
June 2003 the animal was reported to loose weight. Faecal- and blood examination revealed no abnormalities. In the next two months his weight loss progressed. Despite good appetite and apparently enough food-intake the animal was deteriorating and at the end of July it showed pain. No nasal discharge was seen at the time. The animal was treated with 6 grams of ceftiofur and 50 cc of finadyne for the two consecutive days before he died.

Post mortem
At necropsy, based on fat stores and muscle reserves, the animal was in a moderate body condition.
Multiple superficial lesions of the skin were present. Locally on the right leg a wrath-like lesion was seen. On the right thigh, at the site of the trochanter tertius a vesicular lesion with connective tissue and proliferation of bursal epithelium was present.
Inspection of the oral cavity revealed irregularly eroded teeth covered with large amounts of plaque, chronic gingivitis, periodontitis, numerous loose teeth, and exfoliation of a molar. Food impaction between the teeth was evident.
Petechial hemorrhage was present in the heart muscle, chronic filamentous pericarditis on the right auricle, and irregular atrio-ventricular valves.
The lungs showed chronic filamentous, adhesive pleuritis, and focally alveolar emphysema. The bronchial lymphnodes were enlarged.
The abdominal cavity was filled with approximately 100 liter of turbid yellow and red fluid with food elements. The omentum was hyperemic and covered with fibrin depositions.
In the esophageal zone of the stomach a 7-cm long rupture was evident, furthermore multiple, large and irregular ulcers were present.
The stomach contained a lot of food and sand.
The small intestines were overfilled with watery contents.
The contents of the ascending colon were poorly digested, very dry, and contained much sand. The left dorsal and ventral colon was rotated 180 °along the long axis.
The liver and the kidneys were enlarged and pale.
Tissue samples from the heart, lung, liver, stomach, intestine, pancreas, adrenal glands, and skin lesions, were fixed in 4% phosphate-buffered formalin, embedded in paraffin, cut at 4 µm sections and stained with hematoxylin and eosin (H&E), and Perls' Prussian blue reaction for ferric Iron.
Aerobic bacterial culture of the liver and colon contents was performed using standard techniques.
Histologically, the myocardium was hemorrhagic. The lung showed alveolar emphysema.
The liver was hyperemic, and kupffer cells and to a lesser extent hepatocytes were filled with iron pigments. There was focal ballooning and necrosis of the hepatocytes.
The esophageal zone of the stomach showed ulceration of the epithelium, with locally many bacteria, and little inflammatory reaction. Elsewhere, more prominent mixed granulocytic and lymfo-plasmacytic inflammatory reactions, with necrosis, and angiogenesis with fibrosis in the submucosa were present.
The intestinal serosa was covered with a thick fibrinous exsudate with bacteria and plant material. The muscle layer showed superficial necrosis.
The pancreas had multiple hyperplasias of exocrine pancreas tissue.
The adrenals showed multiple hyperplasias of medullar cells.
The kidney showed interstitial fibrosis with calcium depositions.
The superficial lesions of the skin showed aggregations of bacteria in the superficial keratin layer. In some areas the superficial keratin layer had disappeared. There was no inflammatory reaction.

The process on the trochanter tertius was a proliferation of connective tissue with numerous vessels.
No bacteria were cultured from the liver. A mixed population of bacteria was cultured from the colon.

Discussion

The animal died of the consequences of the rupture of the stomach and subsequent peritonitis due to food substances in the abdominal cavity.
Equidae, rhinoceroses and proboscides in nature often die at old age because of teeth problems. The fact that they can't eat or can't digest properly anymore induces emaciation and finally death.
This animal had severe changes in the mouth. Varying from gingivitis, loose teeth, and alveolitis and heavy plaque formation. We think that the animal tried to eat enormous amounts of food, in order to cope with his energy needs, without being able to properly chew the food.
Sand and impaction of the colon with maldigested food provoked torsion of the left dorsal and ventral colon. Stomach impaction with non-chewed material and sand, subsequently evoked a rupture of the stomach. Resulting in a massive peritonitis. Colic signs weren't that obvious, but the animal must have died with bitter pain.
In the clinical case described above we think that the animal finally died as a consequence of the described lesions of the teeth correlated with old age. Due to altered food intake, it finally resulted in the stomach rupture and the torsio coli.

References

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