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SEVERE NECROTIZING ENTERITIS IN A SUMATRAN RHINOCEROS (*DICERORHINUS SUMATRENSIS*)

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SUMMARY A severe necrotising enteritis in a captive Sumatran rhinoceros is described. The lesions were highly suggestive of a *Salmonella* infection. Attempts to isolate the bacteria were however unsuccessful.

INTRODUCTION Health problems of captive Sumatran rhinoceros (*Dicerorhinus sumatrensis*) commonly comprise lesions of the integument and diarrhoea associated with stress and endoparasitism mainly nematodes (Z. Zainal-Zahari, unpublished data). Gastrointestinal problems in the Black rhinoceros (*Diceros bicornis*), White rhinoceros (*Ceratotherium simum*) and the Indian rhinoceros (*Rhinoceros unicornis*) have been reported (Windsor and Ashford 1972; Williamson *et al.*, 1973; Ensley and Bush, 1976 and Jones, 1979). No similar reports are however available for the Sumatran rhinoceros.

This report describes a severe intestinal disease in a Sumatran rhinoceros resembling that caused by a *Salmonella* infection.

CASE REPORT

History and clinical findings A subadult (4 to 5 years old) female Sumatran rhinoceros, captured a year earlier, had a severe shooting bloody diarrhoea. She was inappetence, dull and inactive and her rectal temperature was 38.5 C. Signs of pain and restlessness was apparent. On several occasions flatulence was observed. The faeces were mixed with frank blood, necrotic tissues and mucus and had a fetid odour.

Profuse salivation and froth were apparent from the mouth. She was panting and her respiration rate was 80 per minute. Mouth breathing was observed on several occasions. A short inspiration and a hard, coarse expiration was apparent. During episodes of panting, she would remain on her sternum or lateral recumbency. Teeth grinding was also apparent. Her heart rate was 76 beats per minute.

She was immediately isolated into a holding pen and given 60 mls of Trimethoprim/Sulphamethoxypyridazine (Septotryl, Vetoquinol S.A. Lure, France) and 20 mls of Vitamin B-Complex intramuscularly. A long acting spasmolytic and analgesic (Buscopan compositum, Boehringer Ingelheim Ltd. Berks) was also given intramuscularly. Vitamins and electrolytes supplements (Stress Pak, Camplex, Canada) were provided in the drinking water, which she drank excessively. The diarrhoea however persisted and she died three days later. An autopsy was carried out within two hours of the death.

The dead rhinoceros was one of seven kept in a paddock. Their daily feed were leaves of *Ficus* spp plants, long beans, sweet potatoes, papaya and bananas *ad libitum* and concentrate consisting 50% pig starter and 50% dairy conditioner at 3 kg/animal. Drinking water was from the nearby lake and provided in a mosaic trough and replaced daily. Water from the lake was also used to clean the night stall and the animals.

Two months earlier the same animal had suffered from a similar clinical disease but of lesser severity and shorter course. It responded well to similar line of treatment and recovered fully.

The remaining six animals, aged one to five years old, were healthy. Upon the death of the sick animal, they were treated prophylactially with Amphicillin intramuscularly for five days at 25 mls per day for the young and 50 ml per day for the adult.

Laboratory Findings Autopsy revealed significant lesions mainly in the digestive tract. The mucosal surface of the small intestine, colon and caecum was congested and had large areas covered with yellow loose diphtheritic membranes. Other areas of the mucosa were haemorrhagic and had ulcers of various sizes. In the small intestine, the ileum was affected the worst. The intestinal contents were bloody. The gastric mucosa and the serosal surface of the intestine were markedly congested. Other changes consisted of marked congestion of the liver, kidneys, spleen and lungs as well as petechiations subepicardially and subendocardially.

Microscopically the intestinal mucosa exhibited large and deep areas of necrosis and diphtheresis and thrombosis of capillaries of the lamina propria. In addition, there was severe congestion and oedema of the submucosa.

Samples of tissues and intestinal contents submitted for bacterial isolation did not yield any significant findings eventhough special attention was given to isolating *Salmonella*.

DISCUSSION The clinical signs and the gross and microscopic lesions resembled that caused by an infection with *Salmonella*. That bacteriological isolation for Salmonella was negative, was likely due to the suppression of the bacteria by the antibiotics administered orally and parenterally. Following the death of three young elephants in the same zoo with a severe necrotising gastroenteritis, *Salmonella* was only isolated from one of them (Chooi and Zainal-Zahari, 1987).

The isolation of *Salmonella* from samples of water from the lake and the soil from within the rhinoceros paddock soon after the death of the rhinoceros (Z. Zainal-Zahari, unpublished data) is suggestive that the enteritis was associated with a *Salmonella* infection. This was further supported by the death of two horses in the adjacent paddock of septicaemia with *Salmonella blockley* two weeks later (Z. Zainal-Zahari, unpublished data).

The disease in the rhinoceros occurred at the onset of the rainy season. This was similar to the outbreak of *Salmonella blockley* infection in Asian elephants in the Zoo the year before (Chooi and Zainal-Zahari, 1987).

Faecal analysis indicated that there were carriers among the rhinoceros excreting *Salmonella* spp. contaminating the paddocks. The source of Salmonella was likely the humans. Water from the lake was polluted by human excretas and also other wastes from the nearby hotels.

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