TREATMENT OF OSTEOMYELITIS IN A GREATER ONE-HORNED RHINOCEROS *(RHINOCEROS UNICORNIS)*

Flach EJ[1], Walsh TC[2], Dodds J[1], White A[1], Crowe OM[2]

[1]Zoological Society of London, Veterinary Department, Whipsnade Wild Animal Park, Dunstable, [1]Beds. LU6 2LF, UNITED KINGDOM

[2]Royal Veterinary College, Royal College Street, London NW1 0TU, UNITED KINGDOM

Summary

Osteomyelitis of the second phalanx of the third (middle) digit of an adult greater onehorned rhino's forefoot was diagnosed by clinical signs, radiography and bacterial culture from a discharging sinus. The causative organism, Streptococcus equisimilis, is frequently associated with rhinos and was probably introduced by a penetrating/tracking infection, or localised in the bone following trauma to the foot. Medical treatment did not cure the infection, but surgical exploration and curettage of necrotic bone, plus local and systemic antibiosis was ultimately successful in achieving a clinical cure, although the affected bone fractured in the process.

Introduction

The greater one-horned, or Indian, rhinoceros *(Rhinoceros unicornis)* is commonly affected by foot lesions (VON HOUWALD, 2001, 2002). Chronic cracks between the middle toe (digit III=D3) and the digital cushion or pad of one or both hind feet, leading to separation with deep fissures and hypertrophy of adjacent tissues, are commonly reported in older animals, especially males (STRAUSS and SEIDEL, 1985; VON HOUWALD and FLACH, 1998), but in addition there are reports of vertical cracks in the hoof wall and ulceration of the pad (VON HOUWALD, 2002), and a case of laminitis was seen in an adult with chronic renal failure (JONES, 1979). The skin of the feet and lower limbs may be affected by exudative or pustular dermatitis (VON HOUWALD, 2002). As far as we are aware, there have been no previous reports of osteomyelitis involving the feet of rhinos.

Case Report

"Roopa", a 29-year-old female greater one-horned rhinoceros presented in November 1999 with sudden onset lameness in the right foreleg. The animal was born at Delhi zoo, but had been imported to Whipsnade in 1973 and had bred successfully, having seven pregnancies from which four calves were reared. Previous clinical history included severe wounds from the transportation to Whipsnade, an endometritis following the delivery of a dead foetus (from which *Streptococcus equisimilis* and a *Klebsiella* species were isolated), two incidents of exudative dermatitis (one of which yielded *Streptococcus equisimilis),* three incidents of keratoconjunctivitis and 18 separate incidents of lameness and/or foot lesions. The right foreleg was affected eight times; the lameness resolved without treatment three times, there was one report of solar cuts caused by sharp stones (flints), pus discharged twice from the coronary band and/or inter-digital skin of D3, and on two occasions there were vertical cracks in the lateral wall of D3 in conjunction with bruising and/or splitting of the digital pad. The most recent occurrence of these cracks was in May 1998. Body-weight (BW) at the time of movement to new housing in January 1998 was 2200 kg, and this was used for all drug doses.

Roopa had access to a heated house with concrete flooring partially covered with rubber mats, concrete and woodchip yards and a grass paddock. She was often mixed with two younger females. At presentation the foot was warm, and there was pain in D3. There was a remnant vertical crack in the lateral wall, but the inflammation was not associated with it. Initial treatment consisted of 75 g co-trimazine (Uniprim for horses, Cheminex Laboratories Ltd., approximate dose 34 mg/kg BW) and 2 g flunixin meglumine (Finadyne granules, Schering-Plough Animal Health, 0.9 mg/kg BW) daily in food for five days. Lameness decreased while Roopa was on the course, but gradually increased afterwards, and appeared to be worse on the lateral side of the foot. Inflammation was still centred on the distal part of D3, but now with hyperaemia of the lateral aspect of the coronary band.

Roopa was examined whilst conscious, but lying down, and the crack in the lateral hoof wall was opened up and suspect tracks were followed until they ran out. However, the lameness was unchanged. The course of flunixin was repeated twice with a day in between, but at a dose of approximately 1.1 mg/kg (2.5 mg total dose), and again there was a clinical improvement whilst on treatment.

Roopa's lameness increased markedly over-night in December. She was reluctant to move and only walked on three legs. It was suspected that one of the juveniles had trodden on her foot during the night, so they were kept separate thereafter. The whole of the distal part of D3 was inflamed, including the coronary band and extending on the dorsal surface of the leg as far as the proximal interphalangeal joint. She was chemically immobilised with approximately 1.7 µg/kg BW etorphine hydrochloride and 6.8 µg/kg BW acepromazine maleate (1.5 ml Large Animal Immobilon, Vericore) administered intramuscularly (i/m) by remote injection (Dan-inject rifle and darts) to allow radiographical examination of her affected foot, and the etorphine was then reversed with 2.7 µg/kg BW deprenorphine hydrochloride (1.8 ml Large Animal Revivon, Vericore). Radiographs were taken by a commercial radiographer (Mobile Vet X-rays) and details of the equipment and exposures used are not available. The radiographs revealed that the third phalanx of D3 was misshapen with a ragged outline and multiple areas of radiolucency. Routine haematology revealed slightly low red cell parameters (red blood cell count, haemoglobin concentration and haematocrit) compared to normal values at Whipsnade (Lynx database, BENNETT et al., 1991), slightly raised white cell, and neutrophil counts and a raised fibrinogen concentration (Tab. 1). The only abnormal biochemical results were a slightly low total protein concentration and an increased creatinine phosphokinase concentration. Treatment with co-trimazine and flunixin was restarted.

Over the following week the swelling on the dorsal and medial aspects of the foot, proximal to the coronary band, increased markedly, and then the skin in the centre of the swelling sloughed and revealed yellow necrotic tissue in the centre of which were two sinuses (Fig.1).

Two swabs from these sinuses yielded mixed, non-specific bacterial growths, and there was no clinical improvement with local cleaning and flushing with diluted povidone iodine (Pevidine, C-Vet), followed by instillation of antibiotics (in consecutive order: cloxacillin sodium 'Orbenin LA intramammary', Pfizer, cephalonium 'Cepravin eye ointment', Schering-Plough and enrofloxacin 'Baytril 10 %', Bayer) in conjunction with oral antibiotics (co-trimazine, then approximately 7.3 mg/kg cephalexin 'Ceporex Veterinary Tablets 250 mg, Schering-Plough for 10 days, and finally 4.5 mg/kg enrofloxacin = 100 ml 'Baytril 10 % oral solution' for 18 days). Therefore, the rhino was immobilised again with etorphine and acepromazine (as above) to allow further investigation of the sinus and radiography. The main sinus in the middle of the depression in the dorsal swelling was probed and was found to extend deeply to bone at an approximate depth of 6 cm. A biopsy of the surrounding tissue comprised degenerating granulation tissue with associated inflammation, and a pure growth of *Streptococcus equisimilis* was cultured.

Radiographs revealed a discrete lucent region in the distal third of the second phalanx (P2) and an enlarged joint space between P2 and P3, in addition to the misshapen P3 (Fig. 2). Haematological and biochemical parameters were similar to the previous results (Tab. 1). Osteomyelitis of P2 was diagnosed, with arthritis of the distal inter-phalangeal joint.

Tab. 1: Haematological and biochemical parameters.

Parameter		**December 1999**	**February 2000**	**September 2000**	**Reference range**
					(n=6)
RBCC	$x10^{12}$/l	6.02	6.06	5.05	6.1 – 8.46
Haemoglobin	g/dl	12.5	12.7	11.1	13.6 – 18.7
PCV	l/l	0.343	0.34 1	0.29	0.4 – 0.49
MCV	fl	57.0	56.3	57.4	52.5 – 65.6
MCH	pg	20.8	21.0	22.0	18.8 – 23.8
MCHC	g/dl	36.4	37.2	38.3	34.0 – 38.1
WBCC	$x10^9$/l	10.3	6.6	5.4	5.4 – 9.9
Neutrophils	$x10^9$/l	8.96	5.48	4.54	3.19 – 6.43
Lymphocytes	$x10^9$/l	1.24	0.59	0.76	1.12 – 2.67
Monocytes	$x10^9$/l	0.1	0.26	0.0	0.0 – 0.3
Eosinophils	$x10^9$/l	0.0	0.26	0.11	0.0 – 0.72
Basophils	$x10^9$/l	0.0	0.0	0.0	0.0 – 0.09
Platelets	$x10^9$/l	Clumped	148	84	124 – 302
Fibrinogen	g/l	6.0	6.0	ND	2.87 – 3.66
Total protein	g/l	74.0	73.0	67.6	76.3 – 99.5*
Albumen	g/l	30.2	28.5	24.6	18.6 – 29.8*
Globulin	g/l	43.8	44.5	43.0	41.8 – 76.6*
Urea	mmol/l	3.0	4.6	2.9	1.8 – 5.1*
Creatinine	µmol/l	74.2	84.1	107.7	33.2 – 117.5*
Total bilirubin	µmol/l	1.7	2.1	1.9	1.0 – 10.0*
Alk.Phos	IU/l	36	25	ND	25 – 187*
AST	IU/l	82	74	49	72 – 204*
CPK	IU/l	564	217	149	218 – 497*
Sodium	mmol/l	132.0	136.0	134.0	122.0 – 134.6*
Potassium	mmol/l	4.2	3.66	ND	3.77 – 5.29*
Phosphorus	mmol/l	0.46	0.81	1.28	0.5 – 1.76*
Calcium	mmol/l	2.89	2.77	ND	1.86 – 3.5*

* Biochemical reference range for black rhinoceros *(Diceros bicornis)* n=35

ND = not done

Roopa was anaesthetised on 8th March 2000 in order to investigate and treat the distal digit surgically. She was darted with 4.16 mg etorphine hydrochloride and 17 mg acepromazine (1.7 ml Large Animal Immobilon, Vericore) and then, once recumbent, 200 mg ketamine (Vetalar V, Pharmacia & Upjohn) and 10 mg diazepam (Diazepam injectable, CP Pharmaceuticals) were injected intravenously to deepen anaesthesia for intubation with a 30 mm cuffed tube. Anaesthesia was maintained with 3 % isoflurane (Isoflo, Schering-Plough) in oxygen delivered by a to-and-fro rebreathing system with a 35 L bag. Anaesthetic details have been reported previously (SELISKAR et al., 2000).

Two elliptical incisions on the dorsal aspect of D3 were extended deeply through the swollen tissue to the dorsal aspect of P2. Soft bone was found in the midline and this was curetted and removed for bacteriological culture. Gentamycin-impregnated methyl-methacrylate beads (Septopal, Biomet Merck Ltd.) were inserted in the cavity, and then the deficit on the dorsal surface was filled with a concentrated sugar paste and a dressing was applied to the foot. The dressing was then protected with a cast comprising an initial shell of synthetic casting tape (Vetcast Plus, 3M), a 40 cm length of car tyre on the palmar surface held with further strips of Vetcast and finally a covering of hexalyte veterinary bandage (Vet-lyte, Runlite S.A.) (Fig. 3). The etorphine was reversed with 7.5 mg diprenorphine (Revivon, Vericore), but recovery was slow due mainly to the animal becoming profoundly hypothermic (minimum rectal temperature 32.3 ºC) during the procedure, despite being covered with blankets. Recovery was quickened when 400 mg doxpram (Dopram-V, Fort Dodge) was administered i/v.

Streptococcus equisimilis was cultured from the necrotic bone fragments, and their histological appearance was of degenerate bone with haemorrhagic fibrovascular tissue and heavy exudates of neutrophils and macrophages. Consequently the animal was kept on oral antibiotics (cephalexin for a further two weeks and then enrofloxacin for two weeks) and flunixin meglumine (six weeks of five days on treatment, two days off).

The cast was replaced one week later, under etorphine/ acepromazine immobilisation, and again one week afterwards. There was good granulation tissue at the base of the surgical wound after one week, but after the second week the gentamycin beads were expelled from the wound and the original sinus had reformed in the new granulation tissue. The third cast only lasted two days, so the foot was left open and the wound cleaned daily, and the sinus packed with a gauze swab soaked with 3 g benzylpenicillin (Crystapen, Schering-Plough) and 250 mg gentamycin (5 ml Pangram 5 %, Bimeda). The swelling around the sinus slowly reduced and one month after the operation Roopa was able to walk without a limp. The sinus itself closed approximately two months after surgery, shortly after a piece of necrotic bone was expelled. By three months the swelling had decreased to approximately one third of the size at the time of surgery. However, radiography revealed a sagittal, midline fracture in P2 with lateral displacement of both fragments (Fig. 4). There were no radiological lesions consistent with active infection, so it was decided not to attempt any further surgical treatment.

To date (January 2003) there has been no recurrence of lameness and the swelling on the dorsal aspect of the foot has slowly reduced, although the shape of the middle toe is still abnormal. Unfortunately, the requirement to keep Roopa restricted to her house and yard for long periods caused severe deterioration in chronic splits between D3 and the digital pads of both hind feet, and she has had to undergo extensive and frequent treatments since 2000 for these lesions. The worst foot, the left hind, was placed in a cast in September 2002 and this cast lasted six weeks. The lesions had improved considerably and there has been no lameness since.

Fig. 1: Swelling on the dorsal aspect of D3, right forefoot, with erosion of the skin. The two sinuses are not clearly visible in this view.

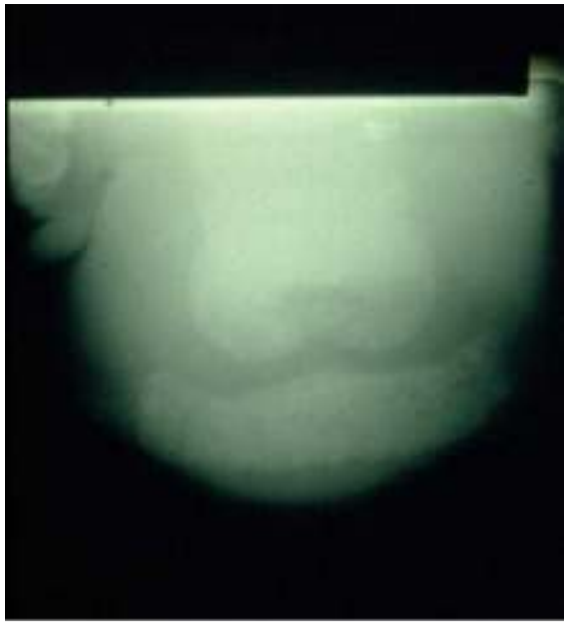

Fig. 2: Dorso-palmar radio graph of terminal D3, right forefoot. There is a spherical area of bone lucency in distal P2 and diffuse lucency and irregular outline of P3, particularly the medial aspect. The distal inter-phalangeal joint space is enlarged.

Fig. 3: Roopa with the first cast, 24 hours after surgery.

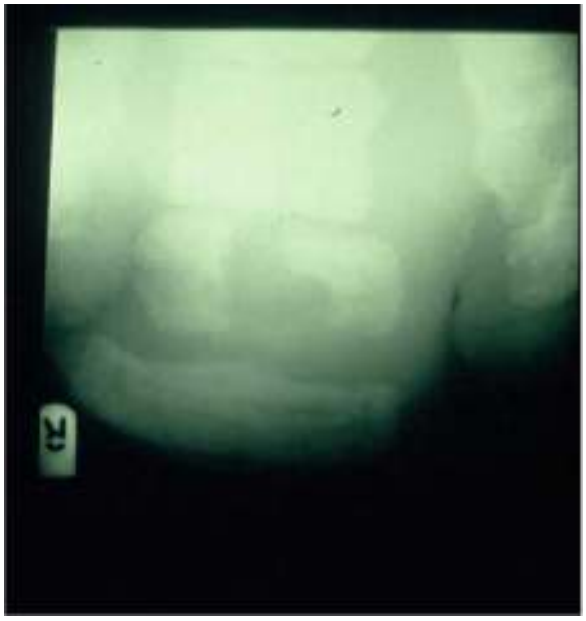

Fig. 4: Dorso-palmar radiograph of D3, right forefoot, 3 months after surgery. The second phalanx has split sagitally and the two fragments are displaced medially and laterally. The distal interphalangeal joint space is still enlarged, whereas the proximal joint appears normal laterally, but enlarged medially. The third phalanx retains a diffuse radiolucency and irregular outline.

Discussion

The causative organism of the osteomyelitis in this case, *Streptococcus equisimilis,* was isolated in pure culture from curetted bone fragments. It is commonly isolated from the skin and mucous membranes of greater one-horned rhinos at Whipsnade and was associated with two previous pathological conditions in Roopa. However, the disease pathogenesis is less clear. Infection may be introduced locally, may spread from an infectious arthritis, or may settle in bone following a bacteraemia. All three could have occurred in this case; firstly, there had been earlier incidents of penetration wounds and tracking infections which may have introduced bacteria to P2, however, there were no recent lesions on the palmar aspect of the foot. Secondly, there was radiological evidence of enlargement of the joint space of the distal inter-phalangeal joint, indicating an arthritis, but thirdly bacteria circulating in the blood stream tend to settle in areas of de-vitalised bone and there was circumstantial evidence of trauma to the affected foot immediately prior to the worsening of the condition.

The history and clinical signs suggested an acute process; a fracture, an infectious arthritis or osteomyelitis, and radiography and bacterial culture were diagnostic. Medical and topical treatment was palliative, but did not cure the condition, whereas surgical debridement of necrotic, infected bone and local application of antibiotics was ultimately successful. It is unlikely that all of the infected parts of P2 were removed, and none of P3 was debrided, so it seems likely that the osteomyelitis was restricted to P2 (despite the radiological changes in P3 which were initially interpreted as osteomyelitis) and that debulking of the necrotic areas was sufficient, in conjunction with the other treatments. Surprisingly, Roopa improved clinically in spite of P2 fracturing vertically. The fracture had not closed when radiographed at the end of 2002, but local fibrosis has presumably stabilised the fragments.

The foot was protected by casting for 2½ weeks and this allowed granulation tissue to fill the deficit left after surgery. Each cast lasted between two and nine days, and other foot casts on rhinos have also lasted only a few days. However, the recent hind foot cast stayed on, and remained intact, for six weeks and so casting should be considered more often for the treatment of rhino foot conditions.

Roopa has been immobilised 18 times since 1994 using etorphine and acepromazine. The dose rate of etorphine for induction has had to be increased over time, from 1.7 µg/kg (1.5 ml Large Animal Immobilon) to 2.6 µg/kg (2.3 ml). However, a single dose has been suitable for minor examinations and non-painful procedures lasting up to 1½ hours. For longer, or painful, procedures ketamine has been given i/v in supplemental doses (between 200 and 1200 mg total dose), and 10 mg diazepam has been injected i/v on the four occasions when she was intubated. We have not had to use detomidine in the induction combination, although ATKINSON et al. (2002) found it to be useful.

Acknowledgments

We should like to thank everyone who helped at any time, especially the keepers on the Asia section at Whipsnade, Alenka Seliskar, Stelio Luna and Richard Eastwood from the University of Cambridge who kindly helped with the anaesthesia for the surgical procedure, students on the MSc course in Wild Animal Health (1999-2000), final year veterinary students at both Cambridge and the Royal Veterinary College, plus veterinary colleagues at both colleges. Bacteriological investigations were carried out by Shaheed Macgregor, Institute of Zoology and Idexx Laboratories Ltd., We the rby, provided histopathological examinations.

In particular we should like to pay tribute to Roopa for her patience and tolerance with all that was done to her.

Colm Walsh died tragically on 12th June 2000. We should like to pay tribute to his skill and infectious enthusiasm and good humour.

References

ATKINSON MW, HULL B, GANDOLF AR, BLUMER ES (2002): Repeated chemical immobilization of a captive greater one-horned rhinoceros (Rhinoceros unicornis), using combinations of etorphine, detomidine and ketamine. J. Zoo Wildl. Med. 33, 157-162.

BENNETT PM, GASCOYNE SC, HART MG, KIRKWOOD JK, HAWKEY CM (1991): Development of LYNX: a computer application for disease dia gnosis and health monitoring in wild mammals, birds and reptiles. Vet. Rec.128, 496-499.

CORNICK-SEAHORN JL, MIKOTA SK, SCHAEFFER DO, RANGLACK GS, BOATRIGHT SB (1995): Isoflurane anesthesia in a rhinoceros. J. Am. Vet. Med. Assoc. 206, 508-511.

JONES DM (1979): The husbandry and veterinary care of captive rhinoceroses. Int. Zoo Yearb. 19, 239-250.

SELISKAR A, FLACH EJ, LUNA SPL, EASTWOOD R (2000): Isoflurane anaesthesia in an Indian rhinoceros (Rhinoceros unicornis). Proc. 7th World Cong. Vet. Anaesth. Sept. 20-23 2000, Berne. pp. 147-148.

STRAUSS G, SEIDEL B (1982): Pododermatitis purulenta beim Panzernashorn (Rhinoceros unicornis) – ein Fallbericht. Verh.ber. Erkrg. Zootiere 24, 177-181.

VON HOUWALD F (2001): Foot problems in Indian rhinoceroses (Rhinoceros unicornis) in zoological gardens: Macroscopic and microscopic anatomy, pathology, and evaluation of the causes. Vet. Med. Diss., Zurich Univ. Switzerland.

VON HOUWALD F (2002): Health. Chapter 5. In: GULDENSCHUH G, VON HOUWALD F (Eds), Husbandry Manual for the Greater One-horned or Indian Rhinoceros Rhinoceros unicornis Linné, 1758. Basel zoo, Switzerland.

VON HOUWALD F, FLACH EJ (1998): Prevalence of chronic foot disease in captive greater onehorned rhinoceros (Rhinoceros unicornis). Proc. Europ. Assoc. Zoo Wildl. Vet. 2, 323-327.