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Rotaviral enteritis in an okapi calf

GIRAFFIDAE

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Brigitte Lord outlines how rapid diagnosis and aggressive treatment given to an immature okapi allowed it to recover from a dangerous condition.

A TWO-MONTH-OLD, male okapi (Okapia johnstoni) calf presented with acute onset watery diarrhoea.

The calf was housed with the dam in a heated shed. The environmental temperatures ranged between 20°C and 25°C. Its birth had been uneventful. Meconium passage occurred at 42 days of age, with 30 to 70 days old being considered normal for okapi calves.

Presentation

On presentation the okapi calf was quiet but alert, continuing to nurse and drink water from a bucket. It was in good body condition and weighed 55kg. It was producing large volumes of pale-coloured watery faeces, but no blood or mucus was present. Defaecation frequency remained normal at once to twice daily, indicating the diarrhoea was of small intestinal origin. On the fourth day the calf was lethargic, depressed and anorexic. Physical examination revealed tacky mucous membranes. Dehydration was estimated at six to eight per cent. Rectal temperature was initially monitored daily and remained normal, between 38.4°C and 38.9°C. The rest of the physical examination was normal.

Differential diagnoses

The differential diagnoses for okapi calf diarrhoea include:

- Viral enteropathogens
  - Rotavirus
  - Coronavirus
- bovine viral diarrhoea virus
- Bacterial enteropathogens
  - Salmonella species
– *Escherichia coli*
– *Campylobacter* species
– *Clostridium perfringens*
– *Yersinia* species

• Parasitic enteropathogens
– *Cryptosporidia* species
– *Giardia*
– strongyle-types
– *Capillaria* species
– *Coccidia* species

• Non-infectious
– nutritional associated
  – ruminal acidosis

**Diagnostic investigation**

Diagnostic investigation included faecal bacterial culture and antibacterial sensitivity testing, viral enteropathogen testing and parasitology. Blood samples were obtained aseptically from the ear veins. Regular monitoring of haematology, electrolyte and acid base analysis and routine urine analysis was carried out to allow therapy to be adjusted as required (Tables 1 to 3). An insufficient blood sample was obtained for full serum biochemistry. Dehydration, metabolic acidosis, hypernatraemia and hypokalaemia were found on blood analysis.

Faecal bacteriology isolated a heavy growth of non-haemolytic *Escherichia coli* and *Enterococcus*. *Salmonella* and *Campylobacter* were not isolated on selective growth. Antibacterial sensitivity testing confirmed sensitivity to amoxicillin. No parasite ova or protozoa were found on faecal parasitology. *Rotavirus* antigens were found in the faeces via a lateral flow immunoassay (immunochromatography). Both dam and sire were screened and were found to be negative for *Rotavirus*.

**Treatment and management**

Treatment with oral rehydration solution and parenteral antibiotic therapy was initiated – amoxicillin intramuscular injection of 15mg/ kg every 48 hours for 10 days.

On further deterioration, intravenous fluid therapy was initiated. An intravenous catheter was aseptically placed into one of the superficial auricular veins on each ear (Figure 1). The calf’s calculated fluid deficit was 4,400ml and maintenance fluid requirements were 3,300ml/day. It was given compound sodium lactate at a rate of 650ml/hr over six hours a day for six days. It was only separated from the dam for this six-hour treatment period daily to encourage nursing from her and avoid maternal rejection. Potassium chloride was supplemented as indicated by the calf’s electrolytes.
After the first day of intravenous fluid therapy, the calf started to nurse and drink small amounts of oral electrolytes from a bucket. It was then maintained on milk replacer and oral rehydration fluids administered by stomach tube twice daily for three days. All clinical signs subsided within 14 days. Close monitoring continued and the calf rapidly started to regain weight (Figure 2). A povidone-iodine foot bath was used to maintain biosecurity.

Discussion

Rotaviruses, virions with double-stranded RNA genome, are very resistant and can persist in the environment. At least five antigenically distinct groups of rotaviruses have been established. Group A, which has been separated into serotypes, has been found in all examined mammals, including okapi, and has an important role in domestic neonatal calf diarrhoea.

More often, atypical rotaviruses are found in diarrhoeic faeces from okapi and gerenuk calves\(^4\). Prevalence of Rotavirus in normal and diarrhoeic faeces from 15 exotic animal species was found to be 57 per cent (20 of 35 animals)\(^5\). Okapi were not included in the study. Young animals excrete rotaviruses in their faeces, clinically or sub-clinically, as passive maternal antibody wanes in the gut.

The source of infection in this case was not determined. The dam and animals in adjacent exhibits were not clinically affected. Transmission is faecal-oral and may have been via personnel, fomites or rodents. Intermittent shedding from the asymptomatic dam cannot be ruled out as the source of the virus. The footbath at the door of the enclosure and protective clothing (overalls and boots) were important in containing the disease to one area and avoided zoonotic transmission.

The clinical signs of small intestinal diarrhoea and anorexia with normal rectal temperature are typical of those seen in other reports of Rotavirus infection in okapi. The 14-day duration of clinical signs was longer than the previously reported 24 hours to five days in okapi of 10 weeks to 11 years of age\(^6\). Secondary opportunistic bacterial infection may have complicated this case. Co-infection of Rotavirus and an encapsulated E. coli was associated with a mortality rate of 50 per cent, affecting neonatal non-domestic hoof-stock\(^7\). Nonpathogenic, non-haemolytic E. coli and Enterococcus were isolated on faecal bacteriology in this case.

Antibiotic therapy was considered appropriate as the bacterial flora of nursing okapi calves is primarily gram-positive in contrast to more gram-negative bacteria being found normally in adults\(^8\). Parenteral use avoided disruption of the start of rumination at 42 days.
Another stressor is the dependency on high environmental temperatures – 19°C to 26°C – as okapi calves do not thermoregulate well until they are about 51 to 60 days old\(^1\). Failure of sufficient passive transfer of maternal antibodies cannot be ruled out in this case.

*Rotavirus*-induced diarrhoea is mediated by malabsorption of electrolytes and nutrients from the cranial portion of the small intestine. Severe metabolic acidosis due to bicarbonate loss was seen as a result in this case. Decreased intake and depletion from intracellular stores lead to hypokalaemia. The high packed cell volume, total protein and hypernatraemia all reflected the dehydration noted on the clinical examination.

The diagnosis of *Rotavirus* associated diarrhoea in this case was made on detection of group A rotaviral antigens on lateral flow immunoassay (LFT), the clinical presentation and lack of detection of other specific causative agents.

Electron microscopy, although considered the gold-standard assay, as it has the potential for rapid diagnosis of enteropathogenic viruses – including atypical *Rotavirus* – lacks sensitivity where a minimum of 10⁶ complete viral particles per gram of faeces must be present for detection. Damage to the virions during sample processing can lead to false interpretations.

LFT, latex agglutination and enzymoimmunoanalysis (ELISA) are rapid and very sensitive, but are specific for un-complexed group A *Rotavirus* antigens. LFT has the advantage over ELISA and latex agglutination in its simplicity and the formation of permanent lines, allowing interpretation of the results at any time\(^10\). PCR may detect more symptomatic cases of *Rotavirus* infection, but this is not commercially available.

Both intravenous and oral fluid therapy, as well as potassium supplementation, were crucial to the management of this calf’s diarrhoea. Intravenous fluid therapy was limited to large boluses given over six hours daily to try to limit maternal rejection and trauma, which has been reported in calves up to four months of age\(^6\). Nutritional support was felt necessary, as calves with extensive villous atrophy can have protracted malabsorption, clinically seen by a reduction in weight gain. Okapi calves should double their bodyweight in four weeks and triple it by seven weeks of age\(^1\). This calf was slightly under weight prior to the diarrhoeal disease and was only just over double his first week birth weight of 26.5kg at seven weeks of age\(^6\). However, on recovery from the *Rotavirus* infection, a good growth rate curve can be seen in Figure 2.
Prevention of *Rotavirus* infection is multifactorial, including local gut immunity by sufficient maternal passive transfer. Good sanitation and a warm humid environment will decrease survivability of *Rotavirus*. Vaccination of the dam or calf has been used in some non-domestic hoof stock, but is not recommended in okapi (Dr F Vercammen, personal communication).

**Conclusion**

This case demonstrates the typical presentation of *Rotavirus* in a non-domestic ruminant, okapi calf. Rapid diagnosis and aggressive treatment proved successful.

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