CLINICAL MANAGEMENT OF SEVERE NECROTIC LAMINAR DISEASE IN AN EASTERN BLACK RHINOCEROS *(Diceros bicornis michaeli*) ASSOCIATED WITH AN UNDETERMINED ETIOLOGY

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Abstract

Recently, a syndrome seen in the black rhinoceros of Zimbabwean origin only, (at the time of this writing) has been suggested as resembling purpura hemorrhagica in the domestic horse.[4] Whether or not the case presented in this paper is a definitive representative of this syndrome has yet to be determined. One aspect of the purpura hemorrhagica like syndrome includes lesions of the feet similar to laminitis. All four feet were affected in this rhinoceros. The lesions appeared to develop initially as horizontal, linear directed, weeping ulcers along the coronary band lesions of varying feet. The length and depth of the ulcer, which foot was affected, as well as the location along the coronary band area also varied over the years. However, in the spring of 1996, they were noted to have been more severe in appearance. It is speculated that the lesions indicated a primary site which then lead to ischemic necrosis of much of the distal aspects of the feet. Most of the soles were sloughed leaving the corium exposed as the weight bearing surface. Both horns were also lost during the initial phases of the disease syndrome. An immune mediated response as seen in Type III hypersensitivity leading to antigen:antibody complexes with resulting vasculitis was suspected. Since the time of the initial tentative diagnosis listed, the purpura hemorrhagica like syndrome has been suggested. Frequent foot trimmings were required beginning in July 1996 for successful therapy. In addition to the surgical management of severe necrotic laminitis, pharmaceutical prescriptions included phenylbutazone (usually 6 g p.o., s.i.d. × 5 days post-immobilizations), trimethoprim-sulfa (#16 960 mg tablets p.o., b.i.d. × 13 wk), and Metronidazole (#50 500 mg tablets p.o., b.i.d. × 7 wk). Minor trimmings are still necessary in the spring of 1998 due to what would appear to be pockets being formed secondary to the more rapid wall growth over the slower growing underlying corium. To date, what was once considered potentially hopeless or at best chronic now appears to carry a prognosis of returning to a fully normal foot without the obvious waving of the nail wall as seen in the domestic horse with chronic laminitis.

Introduction

Purpura hemorrhagica has been associated most often with an earlier respiratory infection caused by *Streptococcus equi* in the domestic horse although an association has been noted with equine influenza as well. There are a few other less common associations. Although the actual cause of the syndrome is not known for sure, it has been strongly suggested that a hypersensitivity to streptococcal antigens leading to antigen:antibody complexes which would then produce the primary histologic lesion of aseptic vasculitis associated with hemorrhage and edema, the classic signs of

purpura in horses.[2,6] This edema is most apparent in the distal extremities of all four limbs, the head, and the ventral midline. Leakage of serum may occur. When purpura occurs in the horse it is usually 2-4 wk post respiratory infection. Affected horses tend to retain their appetites. Treatment involves ridding any underlying bacterial infections, reducing the edema, and inhibiting the immune response. Penicillin and corticosteroids are the pharmaceuticals of choice.

Laminitis in the domestic horse is most commonly associated with a carbohydrate overload (i.e., grain overload). It is suggested that lactase-producing bacteria proliferate in the cecum releasing large volumes of lactic acid. This sudden drop in cecal pH kills the enterobacteria, releasing endotoxins. The combination of lactic acidosis and endotoxemia leads to the inflammation of the pedal laminae. Although blood flow actually increases in the feet, blood is shunted by arteriovenous shunts and therefore no blood reaches the laminae. Thus ischemic necrosis of the laminae occur. There are other conditions which have been noted to lead to the development of laminitis in the horse such as retained placentas, excessive water consumption following exercise, too much exercise, excessive trimmings, pastures with lush grass, and probably others. Treatment includes phenylbutazone for pain and improved blood flow into the corium, cold water soaks to reduce oxygen demand in the laminae and for pain, and corrective shoeing and trimming in the horse. Corticosteroids are contraindicated due to the inhibition or at least the delay of keratin synthesis. Antibiotics prevent secondary infections which could lead to abscesses. Exercise is to be limited to short walks or limited to small pens which encourages blood flow to the laminae while at the same time limiting excessive pressure on the same structures.[1,3,5] It is further suggested that if aggressive daily foot care is unavailable then euthanasia should be considered.[5]

Case Report

A 7.5-yr-old male eastern black rhinoceros (*Diceros bicornis michaeli*) was immobilized for multiple foot trimming to remove necrotic material involving the nail, sole, and corium of all four feet. It had been noted that the animal had experienced coronary band lesions off and on for several years, especially in the spring and summer months. These typically were weeping, crusty, linear directed ulcerated areas extending for various lengths along the coronary bands of varying feet every year. In the early summer of 1996 these lesions were noted to be more severe than noted during fly seasons from earlier years. Topical administration of a chlorhexadine acetate ointment (Nolvasan, Fort Dodge) was chosen to avoid topical corticosteroids in a black rhinoceros even though the minute amount present topically may have rendered this concern as unwarranted. In late July 1996 the first of a series of immobilizations were initiated to first evaluate the reason for recent lameness, why tarsal joints bilaterally and acutely felt hot to the touch the day before, and to determine the extent of coronary band involvement. Radiographs were unremarkable. It was during this initial immobilization that we discovered that a finger could be inserted deep into the foot through one of the ulcers. A Group C *Streptococcus* was grown from this wound but was dismissed as a contaminant. Subsequent immobilizations were conducted to begin the process of removing necrotic material from all four feet. Although, initially we attempted to leave the sole intact to provide a surface to walk on, much of it was necessarily removed from each foot due to the amount of necrotic corium that had to be removed underneath. This meant that the animal was forced to walk on

exposed laminar tissue which allowed for the introduction of foreign material such as pebbles, sand, urine, hay and other feed materials, etc. The decision to avoid bandaging was made due to the cost and difficulty of changing four bandages at least daily with the added risks of even more immobilizations. Feet trimmings were accomplished using X-Acto blades (Bob Corey Associates, PO Box 73, Merrick, NY 11566) which proved to be ideal for rapid, aggressive removal of dead tissue. We use these for routine elephant foot trimmings and consider them indispensable. The blades are available in a multitude of shapes and sizes which allow for precise excision. Corium, sole, and nail tissue were removed to the point of bleeding. Tincture of iodine was applied to cauterize and harden the exposed tissues. Multiple immobilizations allowed for staged excisions as the need arose as well as for the visualization of progress. Spooning of the exposed softer corium tissues was at times so pronounced that one could only imagine an end result of a spooned foot similar in appearance to a Shetland pony with chronic laminitis.

Further, early on in the disease process the animal had developed a number of severe decubital ulcers, especially over the bony prominences. The animal also had developed a severe ventral midline moist, necrotic dermatitis. One may now look back and wonder if ventral edema was first present, although none was noticed, or simply had developed secondary to staying moist and contaminated from sternal positioning and decreased activity. This ventral midline dermatitis responded well to firm scrubbings with iodine, scraping away loose necrotic tissue, and antibiotic coverage (trimethoprim-sulfa (#16 960 mg tablets p.o., b.i.d. for 13 wk), and metronidazole (#50 500 mg tablets p.o., b.i.d. for 7 wk). Nolvasan ointment was applied topically later when the wound was no longer moist. Management of the skin lesion complications progressed from the use of wood chips for cushioning (was beneficial, but contaminated the wounds) to making huge wood chip pillows out of sheets (impossible to move out when it was wet and acted as a constant source of contaminated moisture). We found a satisfactory solution in the use of public donated water bed bladders. These allowed pressure on the feet and skin lesions to be minimized but would pool surface fluids at times. The animal was visibly much more comfortable on these and even appeared to be euphoric at times.

We began supplementing the animal's diet with gelatin to encourage nail growth. This is made into "brownies." Later we added an essential fatty acid supplement to the "brownie," which is flax seed based (Missing Link, Designing Health, Inc. 28310 Avenue Crocker, Unit G, Valencia, CA 91355 USA). It was hoped that adding these micronutrients might help in healing the skin lesions as well. I feel that browsers fed pelleted diets and processed hays would benefit from this addition.

Immobilizations were accomplished using carfentanil (1.2 mg i.m.) or etorphine (3 mg i.m.). Reversal was utilized using naltrexone (100 mg i.v. or 300 mg i.v., respectively). Hand injections in the exhibit with the animal were possible initially. Hiding behind poles with a Telinject dart system while someone distracted the animal was necessary as the animal felt better later on. Weight was estimated at 1000 kg. The animal was typically placed on phenylbutazone (usually 6 g p.o., s.i.d.) for 5 days following immobilizations to control pain. Further, as a nonsteroidal antiinflammatory blood flow to the corium is improved with phenylbutazone due its ability to decrease platelet aggregation. However, concern for gastrointestinal ulceration and the worry of

1000 kg being too comfortable on exposed corium clouded the decision to utilize phenylbutazone more fully.

Discussion

The purpura hemorrhagica like syndrome in black rhinos, which has recently been brought to our attention, does have characteristics that are similar to purpura in horses.[4] Feet lesions that appear similar to laminitis have been noted to occur early in the disease but it has been suggested that these similarities are probably related to pressure associated with the edema. However, histopathology performed on affected areas in the rhino reveal a lymphocytic perivascularitis unlike the purpura in horses, which includes a vasculitis. Further, this syndrome in the black rhinoceros has been associated with a very low PCV (13) and low serum phosphorus. The rhino in this case maintained a PCV of 53 early, slowly dipped to 39 around 6 mo later, and steadily rose back to 53, 1 yr later. Serum phosphorus wavered from 3.2 and 4.8 mg/dl, normal values,[7] throughout the same period. During the initial workup an IgG level was requested but no anti-rhino IgG was available then. A request for IgT was also requested but no reagent for rhino serum was available. Serology was negative for bovine viral diarrhea, equine herpes I, equine viral arteritis, eastern and western equine encephalitis, equine infectious anemia, and bluetongue were all negative. Leptospira serology indicated the following titers approximately 6 yr post single leptospira vaccination: bratislava 1:25, canicola 1:25, ictero 1:200, pomona 1:25, tarassovi 1:25. One year later the animal demonstrated an elevated gamma globulin consistent with chronic inflammation and a decrease in alpha 1 proteins indicating immunosupression or immunodeficiency in this specific group of acute phase proteins (Lisa Michelle Tatum, DVM).

Conclusion

Although it is yet undetermined as to the definitive cause for the necrotic laminitis in this rhinoceros, clinical management proved successful when proper surgical management was combined with appropriate pharmaceutical coverage and an environment which promoted good nursing care and animal comfort. Concerns at this point include recurrence of the disease syndrome: a) the purpura hemorrhagica like syndrome, which has already occurred in at least one institution, resulting in death, and b) the laminitis, which commonly reoccur in horses due to now being predisposed. Unforeseen complications during regrowth such as foreign bodies within the previously exposed laminar tissue or open spaces deep to the wall due to different regrowth rates of the various tissues of the foot are also expected. Finally, one should take and store plenty of serum for future reference should this disease syndrome begin to demand serologic evidence not anticipated earlier.

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