TRYPANOSOMIASIS (SURRA) IN THE CAPTIVE SUMATRAN RHINOCEROS (*Dicerorhinus sumatrensis sumatrensis*) IN PENINSULAR MALAYSIA

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Abstract

Five captive Sumatran rhinoceros (*Dicerorhinus sumatrensis sumatrensis*) housed in a facility in Selangor Malaysia died in a biphasic epidemic that spanned 18 days. Four of the five rhinos had been wild-caught in peninsular Malaysia and translocated into captivity; one was the only offspring of a female that had been pregnant at the time of capture. Clinical signs included initial depression and anorexia followed by rapidly progressing incoordination, muscle tremors, nasal hemorrhage, recumbency and labored breathing, followed by death. Despite broad-spectrum antibiotic and supportive therapy, all five rhinos succumbed. Trypanosomes identified as *Trypanosoma evansi* were detected in blood smears taken just prior to death from the last two animals. Gross pathology was nonspecific; however, histopathologic examination revealed multi-systemic disease compatible with historic reports of surra in other animals. Three animals had intralesional trypanosomes and extravascular hemolysis; three of four animals for which spleen was available had unique and characteristic splenic lesions consisting of marked enlargement of periarteriolar sheaths with lymphoid depletion. Trypanosomes were identified in the brain of one animal in association with endothelial hypertrophy. Immunohistochemistry was performed on tissue samples to further characterize the disease. A herd of buffalo located adjacent to the Sumatran Rhino Conservation Center shared a common fence with the 10-acre reserve where the first animal had been housed for the 2 wk immediately prior to its death. This outbreak represents the first report of surra in the Sumatran rhinoceros.

Introduction

The Sumatran rhinoceros (*Dicerorhinus sumatrensis*) is considered the most endangered rhinoceros species, even though the estimated 300 animals outnumber the rarer Javan species (*Rhinoceros sondaicus*). The Sumatran rhino remains the most threatened of the five extant species largely due to poaching for the rhino's horn, compounded by forest degradation. Following the tragic loss of five animals in Peninsular Malaysia, just eight Sumatran rhinos survive in captivity worldwide, four in zoological facilities in the USA and four in sanctuaries in Southeast Asia.

Wild populations increasingly face risk of emerging diseases, especially where domestic animal reservoirs exist near populations of captive or wild species that may not share similar disease ecology. Recent examples of diseases that are infecting new species or crossing environmental barriers include BSE, CWD, West Nile virus, highly pathogenic avian influenza, monkey pox and ebola virus, and the list continues to grow. Zoonoses are also becoming more prevalent as more people and their livestock move into new environments.[7,16,17] Many of these diseases, including surra, are emerging directly or indirectly because of mankind's exploitation of the earth's resources for food, fuel, medicines, and agriculture - problems compounded by the modern trend towards globalization.[16,17]

Methods

The epidemiology and pathology of an acute epidemic mortality event involving the last captive Sumatran rhinoceros in Peninsular Malaysia was investigated. In addition to extensive necropsy and laboratory testing conducted in Malaysia, partial pathologic tissue sets collected from each animal by the attending veterinarian or the Universiti Putra Malaysia were imported into the USA for further examination. Epidemiologic data were reviewed. This included assessment of chronologic and demographic information; local staff interviews; evaluation of scientific reports and laboratory analysis of samples taken during the outbreak and of testing of domestic animals surrounding the reserve; and review of the literature.

Results and Discussion

Five Sumatran rhinoceros (four females and one male) housed in a species conservation facility in Selangor Malaysia died in an epidemic that was a significant setback to an already struggling captive propagation program. Four of the five rhinos had been wild-caught in peninsular Malaysia and translocated into captivity; one was the only offspring of a female that had been pregnant at the time of capture. The biphasic epidemic spanned just 18 days in October and November of 2003, during the peak of the rainy season.

The first animal died on October 30, within 24 hr of being moved from the adjacent forested reserve back to the barn. This death was followed ten days later by the first clinical signs in the male. Despite broad-spectrum treatment that included antibiotics, fluid therapy, anthelminthics, anti-inflammatory medications and other supportive measures, the animal died. Clinical signs

included lethargy, depression, anorexia, and weakness that progressed to posterior paresis, labored respirations, recumbency and death, which are compatible with those seen in surra in other species.[1,6,20] The next day another female was affected with similar signs, and soon succumbed. The remaining two animals, which became ill about the same time, appeared to be recovering with treatment. However, 6 days later severe clinical signs recurred and death followed. Blood samples collected during the event showed mild anemia and monocytosis, but an otherwise unremarkable leukocyte count.

An initial diagnosis of trypanosomiasis was made by identification of trypanosomes on thick and thin blood smears at the Universiti of Kebangsaan in Kuala Lumpur, Malaysia.[4] The organisms were classified as *Trypanosoma evansi* based on size and morphology. Unfortunately, this diagnosis was not made until after all of the animals had succumbed to the disease. Additional diagnostic tests, including enzyme-linked immunuosorbent assays (ELISA), card agglutination test for trypanosomaiasis (CATT), mouse inoculation test (MIT), immunohistochemistry (IHC), and polymerase chain reaction (PCR), have been developed to help improve detection of *T. evansi*, surveillance for infection, and diagnosis of surra.[11,12,14,15,18,23,24] These methodologies often are used to monitor for recurrent parasitemia post-treatment.[24]

A unique lesion was observed in three of the four spleens examined microscopically. The splenic white pulp was greatly expanded by histiocytes, with central lymphoid depletion, a pathologic lesion observed in both natural and experimental surra infections and classified as depletion of periarteriolar lymphoid sheaths.[3] Anemia, circulating hemosiderophages and marked splenic hemosiderosis were documented, suggesting extravascular hemolytic disease. One animal had evidence of disseminated intravascular coagulation, a condition attributed to trypanosomiasis in humans.[2] Histopathology revealed presumptive trypanosomes in the brain of one animal, in association with endothelial hypertrophy, and in multiple other organs. Further testing of frozen and formalin-fixed tissues, including reproductive organs, for trypanosomes was facilitated by immunohistochemistry. Infertility has been associated with trypanosome infections, which disrupt spermatogenesis in domestic boars and rams.[13,19]

The salivarian trypanosomes, such as *T. evansi*, are readily transmitted by tabanid flies and other diptera.[6,8,22] Tabanids were abundant at the rhino center, with increased numbers prevalent at the time of this epidemic, likely because of the rainy season conditions.[9] Transmission would likely be efficient in the captive environment because of the concentration of susceptible animals exposed to infected vectors, and the unique nature of trypanosome biology.[22] A herd of buffalo located adjacent to the Sumatran Rhino Conservation Center shared a common fence with the 10-acre reserve where the first animal had been housed for the 2 wk immediately prior to its death, and may have been a reservoir of infection for local tabanids.

Perissodactylids as a group appear to be highly sensitive to trypanosomiasis, with high mortality reported in domestic horses.[6,20,21] Trypanosomes have been associated with disease in African black rhinoceros (*Diceros bicornis*), although that species appears relatively resistant to disease, unless stressed by translocation, presumably because of innate resistance.[5,10] Such tolerance of

trypanosome infection would provide an adaptive advantage for a species like the black rhinoceros that has co-evolved with these blood parasites. Since *T. evansi* is a relative newcomer to Southeast Asia,[6] Sumatran rhinos probably have had little opportunity to adapt to infection with this agent. Hence, surra might be expected to appear as an epidemic in a group of susceptible animals exposed to infection at a common site over a short period.

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