

STREPTOCOCCAL ENDOCARDITIS IN A CAPTIVE SOUTHERN WHITE RHINOCEROS (*CERATOTHERIUM SIMUM SIMUM*)

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Abstract: Postmortem examination of a 43-yr-old male southern white rhinoceros (*Ceratotherium simum simum*) revealed gross lesions and histopathologic findings consistent with endocarditis. The animal was born in Umfolozi National Park, South Africa, and then it was moved at 2 yr of age to two successive European zoologic collections. For several weeks prior to death, the animal was increasingly recumbent or assuming a dog-sitting position. Postmortem examination revealed cutaneous pressure sores and multiple rough nodular structures on the mitral valve and left ventricular endocardium. Histopathologic examination revealed vegetative endocarditis, myocardial and hepatocellular degeneration, hepatic fibrosis, and chronic nephritis. Bacterial culture from the oral cavity, trachea, lung, skin, and heart isolated beta hemolytic *Streptococcus dysgalactiae* subsp. *equisimilis* and *Streptococcus ovis*. The cause of death was acute cardiopulmonary failure due mainly to endocarditis and moderate myocardial degeneration. Streptococcal infections are not uncommon causes of morbidity and mortality in rhinoceros. This is the first detailed report of streptococcal endocarditis in a rhinoceros.

Key words: *Ceratotherium simum simum*, endocarditis, nephritis, *Streptococcus*, white rhinoceros.

BRIEF COMMUNICATION

On 16 March 2012, the carcass of a 43-yr-old male southern white rhinoceros (*Ceratotherium simum simum*) was delivered to the Department of Ecology and Pathology of Game Animals in Wroclaw, Poland. The animal was born in Umfolozi National Park (South Africa). At 2 yr of age, it was moved to two successive European zoologic parks. The rhino developed mobility problems 2 yr prior to death, and nutraceuticals to support joint health were prescribed. Various treatments were attempted to improve the rhino's comfort and mobility, but these therapies provided only temporary benefit. The rhinoceros developed pressure sores secondary to spending more time lying down. For 2 wk prior to death, the rhinoceros was in a recumbent position or assumed a dog-sitting posture. Dyspnea began 4 days prior to death.

Necropsy was performed, and representative samples from the affected organs were collected for histopathologic examination. Paraffin sections,

4 μ m thick, were stained with hematoxylin and eosin. During necropsy several different size cutaneous pressure sores of the calcanea, stifle, and on the right front limb were found. The lungs were congested with a large amount of hemosiderin granules and pink edematous fluid in the alveoli. Vesicular emphysema appeared in many areas. In the bronchial wall, mild inflammatory infiltrations were seen. The pulmonary pleura was clumped in some areas by tiny inflammatory adhesions. The pericardial sac contained a small amount of serous fluid. The pericardium and epicardium were smooth and clear. Both ventricles contained semiclotting blood. Rough nodules were present on the mitral valve and a portion of the left ventricular endocardium (Fig. 1). On histologic examination, granulation tissue was present, at different stages of fibrosis and/or myxomatous metaplasia, covered by fibrinonecrotic masses with numerous cocci-like bacteria (Fig. 2). In some areas, it manifested irregular, lamellar formations. The myocardium revealed no inflammatory reaction but scattered vacuolar degeneration and myocytolysis. In the arch of the aortic wall, slightly hollow, creamy white superficial calcifications with a diameter of 4 cm were evident. The esophageal part of the gastric mucosa was pale, smooth, and unchanged, while the gastric body mucosa was moderately congested and covered with excessive mucus. The small intestinal mucosa was just slightly congested; however, multifocal bleeding erosions were noted on the colonic mucosa. The liver exhibited

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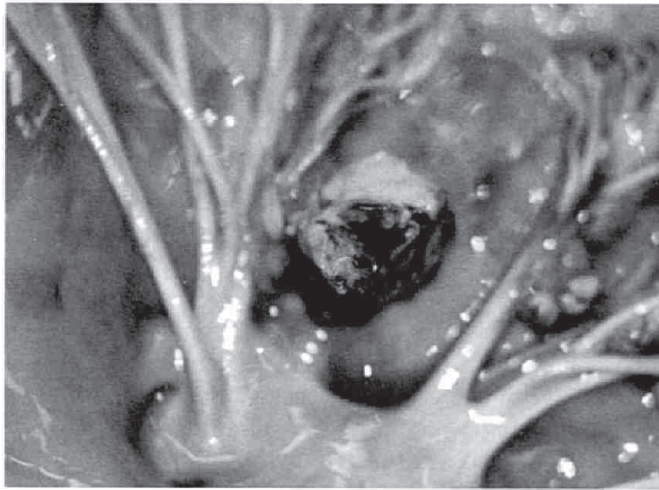


Figure 1. Vegetative endocarditis lesion on the mitral valve of a white rhinoceros.

moderate to severe fibrosis. Bile ducts proliferated, and some of them were dilated with cyst formation. Hepatocytes were swollen with fine hydropic vacuoles. The spleen was of normal size, with petechiae scattered under the capsule. Chronic interstitial nephritis of both kidneys was expressed by extensive fibrosis, hyalinization, and consecutive progressive atrophy of renal corpuscles and canaliculi. Some of them were dilated and formed cysts (1–10 mm in diameter), filled with amber-colored or brown liquid.

For bacterial culture, samples from the oral cavity, trachea, skin, lung, and heart lesions were plated on tryptic soy agar (bioMérieux Polska, 01–882 Warsaw, Poland), supplemented with 5% sterile sheep blood, and subsequently incubated at 37°C for 48 hr. The obtained colonies were examined biochemically using the API 20 STREP identification system (bioMérieux Polska) and further identified by means of partial sequencing of the 16S ribosomal RNA and *groEL* genes. Sequencing of the 16S rRNA was accomplished using universal eubacterial primers, 16S–27f (5'-AGAGTTTGATCMTGGCTCAG-3') and 16S–907r (5'-CCGTCAATTCMTTTRAGTTT-3').⁴ For sequencing of the *groEL* gene, streptogroELd and streptogroELr primers were used.² The sequences obtained were then compared with those available in the GenBank of the National Center for Biotechnology Information, using the Basic Local Alignment Search Tool algorithm.

Bacteriologic examination of the heart lesions yielded two phenotypically distinct types of streptococcal colonies. Colonies of the first type were small, 0.5 mm in diameter, translucent to gray and α -hemolytic. The other colonies were larger (0.5 to 1 mm in diameter), whitish gray and β -hemolytic. Because neither of the strains could

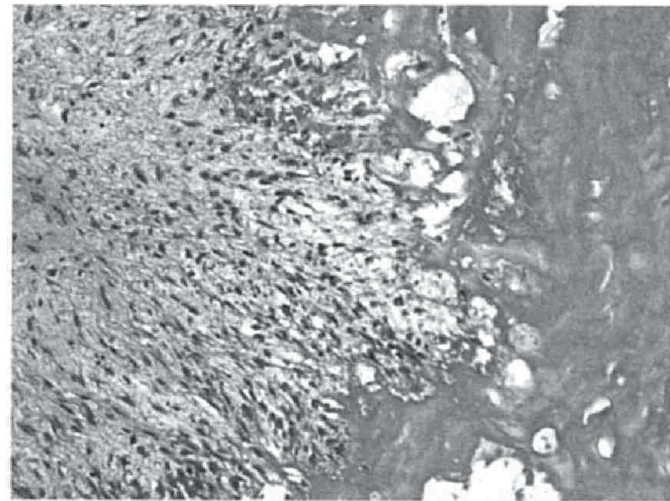


Figure 2. Valvular endocarditis. Granulation tissue covered by fibrinonecrotic mass with numerous cocci-like bacteria. Hematoxylin and eosin stain, $\times 100$.

be identified biochemically, genetic tests, including partial sequencing of the 16S rRNA and *groEL* genes were performed. Finally, the α -hemolytic isolate was assigned to *Streptococcus ovis* (sequence similarity reached 97.6 and 95.9%, respectively) and the β -hemolytic isolate to *Streptococcus dysgalactiae* subsp. *equisimilis* (with 97.5 and 88.8% similarity, respectively). The molecular identification of both strains from heart lesions gave consistent results for each isolate. Isolates corresponding to the two found in the heart (based on results of biochemical tests) were also recovered from other sites of the carcass: *S. ovis* was isolated from the oral cavity, and *S. dysgalactiae* subsp. *equisimilis* was detected in the oral cavity, trachea, skin, and lung.

The results of the performed examination suggest chronic streptococcal infection as a cause of the vegetative endocarditis. The primary source of streptococcal infection is not clear, but with the bacteremia and subsequent endocarditis, the skin lesions were the suspected source. The skin lesions were result of pressure sores from increased recumbency secondary to progressive weakness. Endocarditis likely led to valvular insufficiency and, ultimately, congestive heart failure and circulatory collapse. Previous reports show streptococci as bacteria commonly found in the skin and mucous membranes of rhinoceros.^{1,7} When animal are stressed or immunocompromised, these commensal bacteria can become opportunistic pathogens.

Localized streptococcal infections and streptococcal sepsis have been reported in rhinoceros.^{1,5,7,8} In a bacteriologic survey of black rhinoceros (*Diceros bicornis*), a β -hemolytic *Strep-*

Streptococcus was isolated from skin lesions and various wounds and caused septicemia and death in two animals.¹ In a different report, a β -hemolytic *Streptococcus* was implicated in a case of phalangeal osteomyelitis in a black rhinoceros.⁵ *Streptococcus* sp. has been isolated from adult white rhinoceros with pustular dermatitis.⁸ *Streptococcus equisimilis* was isolated from a variety of tissues, including blood, from several 2- to 3-yr-old white rhinoceros in a game farm in South Africa.⁷ Poor body condition, severe pharyngitis, and esophagitis were common features. These mortalities were postulated to be precipitated by postweaning maladaptation and stress.⁷

Reports of endocarditis in rhinoceros are rare and poorly described. A case of right ventricular endocarditis is mentioned in a 25-yr-old "African" rhinoceros that died after a year of progressive weight loss.⁸ Two additional cases of valvular endocarditis have been reported in southern white rhinoceros neonates: in a 6-wk-old calf secondary to *Escherichia coli* omphalophlebitis³ and 6-month-old calf likely secondary to hematogenous spread from extensive skin lesions.⁶ This case report represents the most extensive documentation of endocarditis in a rhinoceros to date.

In this present case, two species of streptococci were isolated. The opportunistic pathogen isolated from the skin lesions, oral cavity, trachea, lung, and heart lesions was *Streptococcus dysgalactiae* subsp. *equisimilis*. *Streptococcus ovis* is generally considered nonpathogenic; however, it was abundant in the heart lesion in this rhinoceros, so its role cannot be ignored.

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