Rocky Mountain Spotted Fever in Dogs, Brazil

Marcelo B. Labruna, Orson Kamakura, Jonas Moraes-Filho, Mauricio C. Horta, and Richard C. Pacheco

Clinical illness caused by *Rickettsia rickettsii* in dogs has been reported solely in the United States. We report 2 natural clinical cases of Rocky Mountain spotted fever in dogs in Brazil. Each case was confirmed by seroconversion and molecular analysis and resolved after doxycycline therapy.

*Rickettsia rickettsii*, the etiologic agent of Rocky Mountain spotted fever (RMSF), is the most pathogenic of the rickettsiae for humans and some animals. RMSF has been reported in North, Central, and South America, where different tick species serve as vectors (1). Although serologic studies among healthy dogs in Brazil have indicated past infection by *R. rickettsii* (2,3), clinical illness caused by *R. rickettsii* in dogs has been reported solely in the United States (4,5).

In Brazil, the most common vector-borne disease of dogs is canine monocytic ehrlichiosis (CME), caused by *Ehrlichia canis* (6). Clinical signs (fever, depression, petechial hemorrhages, thrombocytopenia) in dogs with overt RMSF infection or CME are often similar (5). Doxycycline is the treatment of choice for *R. rickettsii* infection in dogs (7) and the most commonly prescribed treatment for CME in Brazil. Thus, clinical cases of RMSF among dogs in Brazil could be being misdiagnosed as CME. We describe 2 natural cases of RMSF in dogs in Brazil.

The Cases

On August 23, 2007, a 4-year-old, female, Dogue de Bordeaux (dog 1) was brought to a veterinary clinic in São Paulo because of a high load of ticks noticed 5 days after she had been to a farm in the Itu Municipality (23°15′S, 47°17′W), state of São Paulo. The dog was treated with fipronil and sent home. Tick taxonomic identification was not performed. The next day, the dog had diarrhea and hematochezia and was taken back to the clinic, where laboratory test results were within reference ranges, except for a slight leukocytosis (18,000 cells/mm³) and elevated alkaline phosphatase level (278.6 U/L). Metronidazol was prescribed, and the dog was again sent home. Three days later, the dog was febrile (40.5°C), anorexic, and lethargic. Blood was sent to a private laboratory, where a battery of PCR tests failed to detect DNA of *Babesia* spp., *Borreliia* spp., *Mycoplasma* spp., or *Ehrlichia* spp. The dog was treated with subcutaneous imidocarb and oral doxycycline. The next day, the dog was still febrile (39.4°C) and anorexic, and neurologic signs (ataxia and vestibular syndrome with spontaneous nystagmus) had developed. The animal was hospitalized; doxycycline was switched to the subcutaneous route; and the next day oral prednisone was added. Blood values remained within reference range, except for a slight leukocytosis (17,600 cells/mm³). On August 30, neurologic improvement was noted, and the dog had no fever (38.5°C) and started to eat. Despite slight nystagmus, the dog was discharged the next day. On September 3, (8 days after doxycycline therapy began), the dog showed no clinical abnormality, and a new blood sample was collected for serologic testing. Another blood sample collected on September 10 showed hematologic parameters within reference range except for leukopenia (6,900 cells/mm³).

Serologic evaluation was performed by indirect immunofluorescence assay (IFA) by using antigens of 6 *Rickettsia* isolates from Brazil (8). Plasma from the sample collected on August 24 showed an IFA endpoint titer of 128 for *R. rickettsii* and no reactivity for the remaining rickettsial antigens at a 1:64 dilution. Plasma from the sample collected on September 3 showed the following endpoint titers for rickettsial antigens: *R. rickettsii* 2,048, *R. parkeri* 512, *R. amblyommi* 512, *R. felis* 512, *R. rhipicephali*, and 512; and *R. bellii* 256.

DNA was extracted from the blood samples collected on August 24 and September 3 (before and after antimicrobial drug therapy) by using the DNeasy Tissue Kit (QIAGEN, Chatsworth, CA, USA). Samples were tested by 2 PCR protocols: one targeting a 147-bp fragment of the rickettsial *gltA* gene (9), and the other, a heminested PCR, targeting a fragment of the rickettsial *ompA* gene (10). Extracted DNA from the first blood sample yielded expected products by both PCR protocols. No product was obtained from the second blood sample. Sequencing of the *ompA* product resulted in a 452-bp fragment 100% identical to the corresponding sequence of the Bitterroot strain of *R. rickettsii* from the United States (GenBank accession no. U43804). *Ehrlichia* spp. were not detected by PCR (6) in either sample.

The second case was noted on August 28, 2007, when a 10-month-old, female, miniature Schnauzer (dog 2) was examined at the same veterinary clinic for anorexia, lethargy, fever (40.2°C), vomiting, and tick infestation. This dog had visited the same farm at the same time as dog 1. No neurologic signs were observed. Dog 2 was treated with...
Conclusions

No other PCR product was obtained.

DNA was extracted from the samples collected on August 24 and September 3 (before and after antimicrobial drug therapy) and processed by the PCR protocols cited above. Extracted DNA from the drug therapy) and processed by the PCR protocols cited above. Extracted DNA from the first sample yielded expected product for the gltA-PCR, which was not sequenced. No other PCR product was obtained.

References


Address for correspondence: Marcelo B. Labruna, Departamento de Medicina Veterinária Preventiva e Saúde Animal, Faculdade de Medicina Veterinária e Zootecnia, Universidade de São Paulo, São Paulo, SP, Brazil 05508-270; email: labruna@usp.br

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