DELAY OF RETICULOCYTE MATURATION IN DOGS WITH CHRONIC ANEMIA CAUSED BY *Babesia gibsoni* INFECTION

Toshiyuki TAKAHASHI

Department of Veterinary Internal Medicine
Faculty of Veterinary Medicine
Hokkaido University, Sapporo 060, Japan

To elucidate the mechanism of chronic anemia induced by *Babesia gibsoni* infection in dogs, changes in characteristics of red cells and sera were investigated during the course of prednisolone treatment in infected dogs. The *B. gibsoni*-infected dogs showed no detectable haptoglobin in the serum, indicating a continuous blood loss by intravascular hemolysis. After successive administration of prednisolone to the infected dogs, serum haptoglobin increased to normal levels with elevated hematocrit values. The increase in haptoglobin suggested that the destruction of red cells decreased with administration of prednisolone. Electrophoretic analysis revealed that protein 4.1 in the red cell membrane from *B. gibsoni*-infected dogs consisted mainly of 4.1b, while the increase in 4.1a polypeptide, the most reliable indicator of red cell aging, occurred after prednisolone treatment. These observations indicated that the administration of prednisolone may decrease the intravascular hemolysis through some unknown mechanism, resulting in the prolongation of shortened red cell life span and a transient increase of hematocrit values in *B. gibsoni*-infected dogs. The present study also demonstrated that maturation of reticulocytes was delayed in *B. gibsoni*-infected dogs with chronic anemia, and that the sera of *B. gibsoni*-infected dogs also retarded maturation of reticulocytes obtained from dogs with some different types of anemia. However, the sera from those infected dogs after treatment with prednisolone had no effect on reticulocyle maturation, resulting in an apparent decrease of reticulocyte counts during the transient increase in hematocrit values in the infected dogs. Furthermore, it was demonstrated that hemin, a degradative product of hemoglobin, has an inhibitory effect on the maturation of reticulocytes *in vitro*, but that neither the serum from a dog with acute hemolytic anemia, nor the hemolysates had the same effect on reticulocytes. From these results, it is supposed that the delay of reticulocyte maturation observed in anemic dogs infected with *B. gibsoni* is due to an increase of a degradative product(s) of hemoglobin in the blood of the infected dogs.