MECHANISM OF THROMBOCYTOPENIA IN DOGS INFECTED WITH *BABESIA GIBSONI*

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The mechanism of thrombocytopenia in canine babesiosis was studied. The results were as follows:

1. In dogs infected with *Babesia gibsoni*, both the platelet count and hematocrit (Ht) value began to decrease at 3–4 days prior to the appearance of babesia parasites in the peripheral blood. The decrease of platelet count was more marked than that of Ht value, and it fell to the lowest level, $5\times10^4/\mu l$, when parasitemia reached its peak. On the other hand, the lowest Ht value was obtained after the peak of parasitemia.

2. Intravenous infusion of noradrenalin ($2\mu g$/min/kg) into infected dogs at the beginning of the decrease of the platelet count in their peripheral blood resulted in a transient increase of the platelet count in the peripheral blood. In contrast, administration of noradrenalin did not induce any increase of platelet count in these dogs when they were given it at the lowest platelet count level.

3. The spleen of one dog infected with *B. gibsoni* was examined by electron microscopy. The red pulp of the spleen contained many platelets and erythrocytes in the cords and sinuses. Most of the platelets showed degranulation, vacuolation, and pseudopod formation, and adhered to gaps in the sinus walls or among the cytoplasmic processes of reticular cells in the cords.

4. Anti-platelet auto-antibody was detected in the sera of infected dogs.

5. Microscopical observation of the bone marrow of infected dogs showed no change of the number and the morphology of megakaryocytes.

These results indicated that platelets were rapidly concentrated into the spleen through some unknown mechanisms by *B. gibsoni* infection. This may induce sequestration of erythrocytes in the spleen, resulting in anemia in the infected dogs.