Feline Haemobartonellosis
– A Case Report

by T. Balazs,* J. Robinson,* D. Grey,† and H. C. Grice* 

Feline haemobartonellosis was first described by Flint and Moss, (1) in 1953 in the United States. Haemobartonellae are Gram negative, pleomorphic bacteria and are parasites of the red blood cells. Species within the genus Haemobartonella are classified principally on the basis of host specificity, which is a marked characteristic of these organisms.

The disease caused by these organisms is usually latent until some predisposing factor such as splenectomy, infectious disease, or experimental stress causes an exacerbation of the symptoms.

Subject and History

A 3 year old neutered male cat, was presented for examination in September 1960. The cat lived at Columbus, Ohio, for a one year period (September 1959 — August 1960) and had a history of treatment with sulfa drugs for a pneumonitis (December, 1959). Since this time intermittent anorexia, and depression were observed by the owner.

Physical examination revealed a rectal temperature of 102°F., markedly pale conjunctiva and mucous membranes. A fast and weak pulse was observed on digital palpation. Alveolar sound and distinct heart sounds were detected on auscultation. No pain was elicited on abdominal palpation. The feces was formed and no parasites were seen at fecal examination.

Hematological examination revealed a macrocytic, hyperchromic anemia. The hemogram showed: Erythrocytes 1.6 million/mm³, Hemoglobin 5.5 g/100 ml., Hematocrit 12, Reticulocytes 6%, (MCV. 75 um³, MCH. 34.3 uug., MCHC. 45.8%), Leucocytes 6500/mm³ with 49% lymphocytes. Smears stained by Wright's method showed anisocytosis, polychromasia, normoblasts and Howell Jolly bodies. Coccolid, violet stained bodies morphologically consistent with Haemobartonella felis were found in the majority of the red blood cells (Fig. 1).

Trace amounts of albumin were found in the urine. The urobilinogen level was not above normal (afternoon sample). Blood for bacteriologic examination was taken aseptically from the marginal ear vein. Heparin was used as an anticoagulant.

On the basis of the hematological findings haemobartonellosis was diagnosed and treatment with antibiotics was initiated. Oxytetracycline (200 mg. orally in divided doses) was administered daily for 3 weeks and chlorotetracycline (50 mg. intramuscularly) intermittently. Apart from an initial improvement indicated by hematological examination the condition failed to respond to treatment. Blood transfusions, electrolytes and injections of vitamin B complex were administered as supportive therapy. They were without effect on the course of the disease. After one month 2 injections of oxyphenylarsen (10 mg. I.V.) were given one week apart with further supportive therapy. In the meantime anorexia and weight loss continued and the cat became emaciated. Three months after the first examination the cat was presented in a moribund state. Euthanasia was performed and blood was taken for experimental inoculation.

Post Mortem Examination

Post mortem examination showed dilation of the heart with hypertrophy. The other thoracic viscera appeared normal. The kidneys were khaki coloured and their capsules stripped easily. The spleen was moderately enlarged with the edges being slightly rounded. The liver was pale and

contained multiple small 2-3 mm. nodules that were raised slightly above the liver surface. These were grey white in colour and were seen to extend into the liver parenchyma. The cut surface of the liver presented a mottled appearance. No other gross lesions were observed.

**Histopathology**

The liver histology was quite remarkable in that approximately one-half the liver parenchyma was replaced by hemopoietic cells, (Fig. 2). The cells in this extra-medullary hemopoietic system were similar in type and degree of maturation to those of the bone marrow (compare with Fig. 3). They were not uniformly disposed throughout the liver, as some aggregates were observed around the portal trinity and others in the vicinity of the central veins. The ectopic myeloid cells were supported by a thin, lace-like reticular network, the cells of which were similar to supporting cells of the bone marrow except that their nuclei tended to be somewhat larger than those of the marrow. The liver parenchymal cells were poorly aligned and showed loss of cohesiveness. Necrotic liver cells were dispersed throughout the parenchyma without focalization. In the cells that had apparently been functional before death there was a loss of cytoplasmic basophilia with many cells showing early degenerative change. It was obvious from these histologic lesions of degeneration and necrosis and from the impingement on liver parenchyma by the hematopoietic system that during life this liver had been a severely taxed organ.

Ectopic myelopoiesis in the spleen was most apparent in the vicinity of the trabeculae. The white pulp was noticeably reduced in amount and it is probable that the increased size of the spleen observed in the gross was due principally to the myeloid metaplasia. Fatty change was observed in the heart muscle. This was generalized but not extensive. Cloudy swelling and fatty change was observed in the kidney tubules. These degenerative processes were observed in all but the collecting tubules.

Examination of bone marrow smear stained with Giemsa revealed a normal cellularity with suppression of normoblastic and myeloid development. Approximately one-half of the marrow cells were large with a light blue cytoplasm, large nucleus and distinct nucleoli. These resembled blast cells. A very few normoblast and polymorphonuclear leucocytes were seen whereas lymphocytes were numerous (Fig. 3).

**Bacteriology**

Blood collected aseptically into heparin was inoculated respectively onto the surface of an agar slant and into the liquid medium described by Gieman (2). Both media were dispensed in screw cap tubes and after inoculation were incubated at 28°C. (3) for four days. A thin film of growth developed on the surface of the agar culture and a very fine granular subsurface growth in appearance suggestive of ground glass, was evident along the wall of the tube containing the liquid medium. Smears made from the visible growth contained small Gram negative rods and cocci both of which appeared singly, in pairs or in short chains. In older cultures, larger coccus forms developed. Transfers made from the initial culture to freshly prepared media consistently failed to develop.

The morphological appearance of the cells present in the initial culture and its pattern of development very closely resembled that described by Ford and Murray (3) and Gieman (2) for *Haemobartonella muris*. The bacterium isolated from the cat differed from that isolated from the rat to the extent that the former failed to develop on subculture in the medium described by Gieman while the latter developed readily in such a medium.

**Animal Inoculation**

Two cats were inoculated intraperitoneally, one receiving 1 ml. of heparinized blood and the second 2 ml. of the culture grown on liquid media.

Ten days later both cats developed a slight degree of anemia. Blood smears of the cat injected with the culture showed small, ovoid bodies in the erythrocytes which resembled the *Haemobartonella*. Microscopic examination of blood smears was negative a month later, and no clinical symptoms developed in either experimental animal.

**Discussion**

The case reported herein showed a striking similarity to the chronic form of haemobartonellosis as described by Lumb (4). A profound macrocytic hyperchromic anemia and a concurrent leucopenia with relative lymphocytosis is characteristic of this
type of bartonellosis in dogs. The development of the extramedullary hematopoiesis is characteristic of chronic bartonellosis in rats, and has been observed also in cats, (5). This was the most apparent lesion in our case and appeared histologically to have developed at the expense of the liver parenchyma. This would suggest that the liver was not able to furnish the factors necessary for red cell maturation, which was manifested in the maturation arrest of hematopoiesis. In our case the marrow was probably exhausted and blood cell production was not equal to the demands imposed on it by massive blood cell destruction. However the rate of erythrocyte destruction in this chronic case was not accelerated to the extent that jaundice manifested itself.

The failure of our animal to respond to therapy is in accord with the findings of Donovan and Loeb (6) and with Flint et al (7). These workers reported that antibiotic therapy against haemobartonellosis in dogs and cats, respectively, did not prevent relapses nor accomplish complete destruction of the bacteria.

**Summary**

A case of feline haemobartonellosis observed in a 3 year old male cat is described. The condition was chronic in nature and failed to respond to treatment.

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