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A Case of Aplastic Anemia in the Canine

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On March 8, 1966, a three-year old Great Dane was admitted to the Stange Memorial Clinic. For the last week the dog had been lethargic and losing weight. Physical examination revealed an emaciated, anemic animal with a conjunctivitis, tonsillitis and a dry cracked muzzle.

Clinical pathology:

Fecal floatation: negative for parasite ova

Urinalysis:	specific gravity	1.046
	albumin	plus one (100 mg).
	sugar	negative
	acetone	negative
	occult blood	negative
	Ictotest	plus one
	sediment	many leukocytes and epithelial cells
Blood:	Hemoglobin	7.87 gm%
	P.C.V.	30%
	RBC	4,240,000/cu. mm.
	WBC	11,100/cu. mm.
	Differential:	
	segs	41%
	lymphs	53%
	eosin	2%
	bands	2%
	monos	2%

Therapy consisted of oral Visorbin®, Bio-Delta® I.M., and chloramphenicol ointment topically in the eyes and on the muzzle. After four days the only improvement was the clearing of the conjunctivitis. At this time the dog was given 500 cc of whole blood, vitamin B_{12}, Bio-Delta, penicillin-streptomycin, and Livitamin® with iron. The penicillin-streptomycin and Livitamin therapy was continued for two more days. At this time the hemoglobin measured 9.22 gm% and the P.C.V. was 36%. The dog was alert and active and was sent home.

On March 19 this Great Dane was readmitted. Blood analysis was made.

Hemoglobin	7.83 gm%
P.C.V.	31%
WBC	14,500/cu.mm.
Differential:	
segs	53%
lymphs	26%
eosin	2%
baso	1%
bands	15%
monos	3%

Over the next several months this dog returned frequently to the clinic with anemia, depression, and dermatitis. On October 15, 1966, he was once again admitted to the clinic. At this time he was extremely weak and anemic.

Clinical pathology:

Hemoglobin	7.49
P.C.V.	24%
RBC	3,000,000/cu. mm.
WBC	8,300/cu. mm.
Differential:	
segs	50%
lymphs	34%
bands	4%
eosin	12%

A bone marrow biopsy was made at this time and the results were interpreted as indicative of aplastic anemia. The dog's

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condition progressively worsened and the owner's permission for euthanasia was obtained on October 20, 1966.

Necropsy findings:
- pale mucous membranes
- pumpkin-shaped heart
- pale myocardium
- enlarged, endurated, brownish lymph nodes throughout the body
- contracted spleen
- dark, copper-colored kidneys
- small adrenals with very thin cortex

Histopathology:
- Liver: centrolobular cirrhosis, vests of hemosiderotic Kupffer cells, lack of hematopoietic centers
- Kidney: blood pigments in the epithelium of the convoluted tubules
- Lymph nodes: hemosiderosis, a few neutrophils, many plasma cells, apparently abortive erythropoiesis
- Spleen: hemosidersosis, apparent lack of hematopoiesis
- Lung: edema and "heart failure" cells
- Thymus: mild hemosiderosis
- Adrenal: atrophy of the zona fasciculata and the zona reticularis of the cortex; being replaced by a fibrotic zone
- Bone: fatty marrow with moderate cellularity, primarily granulocytic
- Rib: lack of erythropoietic elements

The final diagnosis was aplastic anemia of unknown etiology.

Discussion and Conclusions:

Aplastic anemia is usually classified as idiopathic since in most cases the etiology cannot be determined. Known causes of aplastic anemia are: chemical agents, radiant energy, endogenous toxins associated with bacterial infections, and possibly uremia or neoplastic diseases. In some cases, erythropoiesis is selectively inhibited, while in others there is almost selective leucopenia or thrombocytopenia. In suppurative infections, erythropoiesis is principally depressed. Clostridial and most viral infections cause primarily a leucopenia. Drugs such as chloramphenicol, sulfonamides, oxytetracycline, and chlortetracycline have been reported to produce aplastic anemia in man. Chemicals such as arsenic, bismuth, and gold compounds can cause aplastic anemia. Primary aplastic anemia is rare in animals, although there are reported cases.

The cause of the aplastic anemia in this case has not been determined. However, there are two likely possibilities. The dog had a history of a chronic *Staphlococcus aureus* infection which took several forms (suppurative dermitis about the feet and legs, furunculosis along the lower jaw, blepharitis, otitis externa, posthitis, and conjunctivitis). *Staphlococcus aureus* has been shown to produce a variety of toxins. In the furunculosis, the necrotic lesions in the tissue are caused by a dermonecrotizing toxin. Other toxins produced are leukocidin, acute killing fraction, nephrotoxin, coagulase, fibrinolysin, and most important, hemolysin. These toxins could have been liberated continuously for the year and seven months that the dog was affected with this chronic staphlococcosis. It seems highly likely that one or all of the hematopoietic centers could have been depressed.

The second possibility is a drug induced aplastic anemia. As expected the dog was treated extensively for the staphlococcal infection. Many drugs were used in the course of the infection. Documented cases of drug-caused blood dyscrasias in animals are rare, but it seems entirely plausible that the intense and protracted use of medicinals in this dog would be enough to tilt the delicate biological balance.

Summary:

A diagnosis of aplastic anemia was made because of the progressive untreatable anemia, and the gross and microscopic changes found on postmortem examination. The cause was not determined, but it may have been due to a chronic staphlococcal infection or due to the extensive application of drug therapy.

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