

1960

Hypoglycemia in a Dog

Frederick D. Branco
Iowa State University

Follow this and additional works at: https://lib.dr.iastate.edu/iowastate_veterinarian



Part of the [Small or Companion Animal Medicine Commons](#), and the [Veterinary Pathology and Pathobiology Commons](#)

Recommended Citation

Branco, Frederick D. (1960) "Hypoglycemia in a Dog," *Iowa State University Veterinarian*: Vol. 22 : Iss. 2 , Article 11.
Available at: https://lib.dr.iastate.edu/iowastate_veterinarian/vol22/iss2/11

This Article is brought to you for free and open access by the Journals at Iowa State University Digital Repository. It has been accepted for inclusion in Iowa State University Veterinarian by an authorized editor of Iowa State University Digital Repository. For more information, please contact digirep@iastate.edu.

Hypoglycemia in a Dog. On December 15, 1958, an eight and one half year old male German Shepherd entered the I.S.U. Veterinary Clinic for examination with a history of incoordination and convulsions. Since about September 15, 1958, the dog had several convulsions, the last of which was rather violent and accompanied by a temperature of 104° F. He had appeared normal and in good condition on November 3, when examined by another veterinarian except for a peculiar crossing over gait and an occasional collapse of the rear quarters. At times the front legs seemed to be involved. The dog had been kept on K/D (Hill Packing Co.) because of a mild chronic nephritis. But on December 15, the Shepherd had deteriorated markedly and had lost a lot of condition although the appetite was reported to be good. The motor control of the hind legs was very poor. Motion being extremely difficult, he fell over or went down behind quite often.

The laboratory tests performed at the clinic revealed a low blood sugar of 30 mg. The normal ranges from 80 to 100 mg. per 100 cc of blood. Symptoms of hypoglycemia occur when the blood sugar level goes below approximately 60 mg. per 100 cc. Also, x-ray pictures revealed an ankylosing spondylitis and spinal arthritis with the greatest involvement at the first and second lumbar vertebrae. There also seemed to be some spinal arthritis at the atlanto-occipital articulation and the supraspinous process of the axis and third cervical vertebra.

A tentative diagnosis of tumor of the pancreas was made. It was suggested that surgery be performed to locate and remove such possible tumorous etiology, but surgery was declined by the owner.

The dog was put on cortisone, Delta Cortef (Upjohn), and later Decadron (Merck) for the arthritis and its gluconeogenic effect. He was fed four to five times daily to combat the hypoglycemia. He responded very well in a few weeks. At present he is on Decadron and is fed four to five times daily. The only symptoms shown are occasional weakness of the rear quarters and depression. He has gained

15 to 20 pounds since entering the clinic and is in apparent good physical condition. Symptoms reoccur when cortisone therapy is stopped. When fasting this dog for a blood sugar test he becomes very lethargic.

Blood sugar levels that have been run on him since entering the clinic are as follows:

December 31, 1958	Blood sugar — 22 mg.	%
January 1, 1959	Blood sugar — 31 mg.	%
February 9, 1959	Blood sugar — 40.5 mg.	%
March 23, 1959	Blood sugar — 13 mg.	%

Discussion and Conclusions

Hypoglycemia is actually due to a hyperinsulinism which is usually caused by a functioning islet-cell adenoma. Less common causes in the dog of such a spontaneous hyperinsulinism may be hypertrophy or hyperplasia of the Islets of Langerhans, or an islet-cell carcinoma, severe liver disease, hypocorticoidism and hypothyroidism.

This condition is not commonly seen in dogs, but does occur occasionally. The symptoms are identical with those seen in dogs given excessive doses of insulin. The attacks may be mild or severe, depending on the degree of hypoglycemia. They may be of long or short duration, and occur when the blood sugar level drops below approximately 60 mg. per cent. Symptoms range from nervousness, weakness, and irritability to symptoms of mental confusion, incoordinated stumbling, and convulsions.

It should not be forgotten that transient hypoglycemia may normally be seen in certain functional states such as pregnancy, lactation, and exercise. In human literature a spontaneous functional hyperinsulinism is described as being caused by nervousness and anxiety. These symptoms occur especially after meals. Such a hyperinsulinism from nervousness could feasibly occur in the canine, considering some of the extremely nervous high strung individuals that exist.

Treatment should be directed toward correction of the cause. As islet-cell adenomas are the most common cause in dogs, surgery should be advised and the

tumor located and removed. It is very possible that in some cases the adenoma may be microscopic, and therefore impossible to locate and remove. Acute attacks are overcome immediately by oral or parenteral administration of glucose, or a subcutaneous injection of epinephrine. In case surgery proves unsuccessful or is declined, the patient should be fed several times daily on a diet low in carbohydrate, high in protein, and high in fat constituents. A high carbohydrate diet is not recommended as it stimulates the islet cells to secrete more insulin which would aggravate the already hyperinsulin condition. Glucocorticoids may be helpful because of their gluconeogenic properties and because hypoadrenalism may be a cause for hypoglycemia.

Frederick D. Branco, '60

REFERENCES

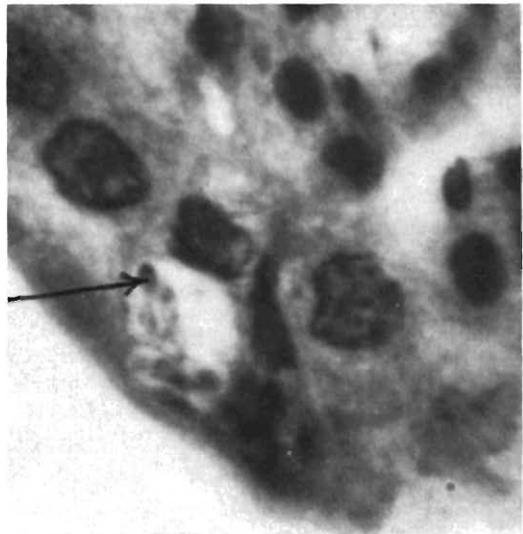
- Dr. R. F. Pahle, Canine Medicine, Am. Vet. Publications, Inc., pp. 398-399.
Principles of Internal Medicine, Harriso, McGraw-Hill: 1958, pp. 628-630.

5

A Case of Toxoplasmosis in a Domestic Feline. On November 13, 1959, a one year old male, domestic feline was admitted to Stange Memorial Clinic with a rather vague history of being weak and having a nasal discharge. Rales could be heard in the lungs upon examination along with signs of dyspnea and abdominal pain when palpated. The cat died on the examination table with no specific diagnosis.

Permission was granted by the owner and a necropsy was performed. The gross lesions found in their order of significance were petechial hemorrhages on the surface of both kidneys and throughout the cortex, emphysema and congestion of the lungs, and a marked brownish discoloration presumably due to congestion. All the mucous membranes and muscles were icteric and anemia was present. A gelatinous, bile stained pericardial sac was found. The spleen was enlarged by fifty to seventy-five per cent. The mesenteric lymph nodes were edematous and the trachea contained a yellowish fluid. There was generalized emaciation and dehydration.

Tissues were saved for histological examination. The lungs presented a large number of the protozoon, *Toxoplasma gondii*, when stained and examined microscopically with oil emersion. The organisms were in close proximity to areas of necrosis which were filled with a yellow



Toxoplasma gondii (H. E. Stain)

staining material. (This was a hematoxylin-eosin stained slide). The diagnosis could then be made of acute Toxoplasmosis.

This organism has been found in almost all warm blooded mammals, in domestic fowl, in 45 species of wild birds, and in some reptiles. The incidence of infection in man and the dog is closely related when they are living in close contact with each other. It is believed this applies to man and cat also. The eating of raw or poorly cooked meat is believed to be one method by which the organism is spread, since this produces higher antibody titers.

Significant titers in the United States have been found in five per cent of the children under four years of age. An increase to 65 to 70 per cent in the 50 year age group show titers. It is concluded that due to the wide distribution of the organism, the usual host-parasite relationship is a symbiosis with only exceptional cases becoming clinically diseased. Clinically in humans it is primarily seen in children and rarely in adults.

D. D. Kerns, '60