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Hypoglycemia in the Dog

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Case History

On August 20, 1969, a 9½ year old spayed female German Shepherd cross-bred dog was presented.** The patient had the following anamnesis: The animal had been noticed to be lame in the hind quarters, particularly in the morning. The owner stated that the dog seemed to "warm out" of the lameness as the day went on. The dog had been sleeping more than usual the previous few days and acted very weak at times. There had been polydypsia, polyuria, and polyphagia for the previous few weeks.

Physical examination revealed nothing abnormal except immature cataracts in both eyes, and a rectal temperature of 103.6° F. The dog was hospitalized to complete a more thorough clinical work-up.

Blood was withdrawn for a complete blood count and urine was obtained via a catheter for urinalysis (tables 1 & 2). After withdrawing the blood sample, the dog started having "chewing-gum fits" and subsequently went into a tono-clonic convulsion. For sedation 3.5 ml. sodium pentobarbital was given intravenously with 1.5 ml. acepromazine maleate† intramuscularly. The convulsion was controlled, but the dog still showed restlessness and anxiety. At this time a blood sugar determination was completed on the previously drawn blood sample (Table 3). A value of less than 40 mg/100 ml. was obtained.‡ Administration of 25 ml. of 50% dextrose intravenously plus 300 ml. of 5% dextrose and half strength saline subcutaneously relieved the restlessness and anxiety. The dog then became comatose within a matter of minutes. Twelve hours following the dextrose administration, the patient began to have "chewing-gum fits". Administration of 20 ml. of 50% dextrose intravenously plus 100 ml. of a concentrated sucrose solution per os relieved the convulsions and put the patient into a state of semiconsciousness.

On day 3 the blood glucose level again was 40 mg./100 ml. The dog was in a comatose state and unresponsive to both sight and sound. Administration of 20 ml. of 50% dextrose intravenously, 350 ml. of 5% dextrose and half strength saline with 0.5 ml. flumethasone§ intravenously 60 drops per minute did not relieve the comatose condition. The patient remained comatose throughout day 3 and was euthanatized on day 4 at the owner's request.

Post-mortem Findings

The mucous membranes were of normal color, and no external gross lesions were observed. Internally, no gross lesions were observed except pulmonary hypos-

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** Hickman Animal Hospital, Aurora, Illinois.
† Aryest Labs.
‡ "Dextrostix" Ames Co., Elkhart, Indiana.
§ Flucort Injectable. Syntex Labs.

tatic congestion, a hard nodule 3 cm. in diameter protruding into the lumen of the pylorus, and a firm nodule, 1 cm. in diameter, in the left lobe of the pancreas. Histopathology* revealed the nodule in the stomach to be a fibroma-like lesion with some calcific deposits. The nodule in the pancreas was diagnosed an islet-cell carcinoma.

Discussion

The above case offers a good chance to review the thought processes used in securing an accurate diagnosis. The clinical signs and clinical pathology very clearly indicate hypoglycemia; the problem is to pin-point the cause antemortem.

In this case the owner's history by itself gave no striking reason to suspect hypoglycemia. In fact, with the polydypsia, polyuria, and polyphagia in conjunction with the cataracts, diabetes mellitus was considered. Other conditions considered, based upon the history obtained, were hyperadrenalcorticalism, and diabetis insipidus. The owner gave no indication of previous convulsions or loss of consciousness, but this is often the case with certain types of hypoglycemia.[2] The weakness in the hind quarters noted by the owner probably was due to the hypoglycemia, but the age of the animal made the diagnosis of some musculo-skeletal disorder a possibility.

On day 2 the dog went into convulsions (following a 24 hour fast), and thus new differential diagnoses had to be considered. These include:[7] Congenital conditions such as epilepsy and hydrocephalus—conditions usually occurring in young animals; infectious conditions such as canine distemper often cause convulsions, but these also occur in young dogs; traumatic causes are a possibility, but the history is not indicative of this; toxic causes are also a possibility, but again the history is not indicative of this; neoplastic conditions of the central nervous system can be responsible for convulsions, and usually occur in old animals. The low blood glucose, however, distinguishes all of the above from hypoglycemia.

The possible causes of low blood sugar must now be considered.[4] Basically, decreases in blood sugar values may result from: 1) normal hepatic glucose output with an accelerated uptake peripherally; 2) a decrease in hepatic gluconeogenesis with a normal peripheral utilization of glucose; or 3) a combination of the two mechanisms. Conditions causing these mechanisms to occur include:[7]

1) Severe hepatic disease. This can be excluded in this case because the low blood sugar usually will not be measured until the liver damage is extensive enough to cause other signs of hepatic damage to be present.

2) Adrenal cortical insufficiency. Hypoglycemia is not as commonly associated with this condition in the dog as it is in man. In addition, adrenal cortical insufficiency is usually associated with very marked gastro-intestinal disturbances.

3) Chronic starvation or malabsorption. The history plus the clinical signs do not justify this diagnosis.

4) Glycogen storage diseases. These metabolic syndromes usually occur in puppies and younger dogs. This patient had no previous history to indicate this syndrome.

5) Excess insulin—either exogenous or endogenous. In this case exogenous sources can be ruled out. Endogenous excess insulin due to beta cell hyperplasia or neoplasia can be diagnosed by satisfying Whipple's triad—originally postulated for human medicine:[10]

A) Attacks occur during the fasting period or at times of effort.
B) Blood sugar levels below 50 mg.% during an attack or after a fast.
C) A return to a more normal state after administration of a glucose solution.

This case satisfied Whipple's triad. Two additional requirements in the form of metabolic tests have been added to Whipple's triad:

A) A glucose tolerance test which reveals:
   a) subnormal fasting blood sugar.
   b) a rise following glucose admin-

* Histopathology done by the Department of Pathology at the University of Illinois, Urbana, Illinois.

istration to a level which seldom exceeds 120 mg./100 ml.

c) restoration of subnormal values within 2 hours with a maintenance of these levels for 6 hours.

B) Continuous intravenous administration of glucose in which there is evidence of a great increase in the ability to remove glucose from the blood, i.e. failure of hyperglycemia and glycosuria to appear after administration of glucose at a rate of 1 gram glucose/Kg. body weight/hour for 2 hours.[5]

These two tests were not done on this dog, but would have aided in confirming the diagnosis.

With the history and clinical findings in this case an antemortem diagnosis of insuloma, an insidious disease in which the neoplastic islet cells of the pancreas produce an excess of insulin, was made. Because the patient could not be brought out of the comatose condition, treatment in this case was precluded. In cases detected earlier the only known successful treatment is excision of the tumor or total pancreatectomy. However, the tumor very frequently metastasizes and this usually causes death of the animal eventaully.[1] If surgery is not possible, the condition may be temporarily aided by medical management. This includes the frequent feedings of a noninsulin stimulative diet (i.e. high protein and low carbohydrate) plus ACTH or prednisolone daily.[3] The steroid causes an increase in liver glycogen stores by stimulating an increased rate of gluconeogenesis and by inhibiting insulin peripherally. This tends to overcome the deficit of available glucose. A review of the literature, however, indicates that neither surgical excision nor medical management are particularly successful.

Conclusion

Carcinoma of the islets of Langerhans (canine insuloma) is not a common syndrome, but the possibility should be considered when the clinical signs are present and if there is a subnormal blood glucose level. Other causes of similar clinical signs and of hypoglycemia must be considered before the diagnosis can be made. If canine insuloma is diagnosed, a poor prognosis must be given.

TABLE 1

Complete Blood Count	
Hemoglobin	16 gms./100 ml.
PCV	48%
Total WBC	10,600/cubic mm.
Differential WBC	
segmented neutrophils	74%
juvenile neutrophils	3%
lymphocytes	20%
monocytes	3%
eosinophils	0%
Blood Urea Nitrogen	10 mg./100 ml.

TABLE 2

Urinalysis	
Color	straw
Transparency	cloudy
Sp. Gr.	1.042
Reaction	6.0
Albumin	30 mg./100 ml.
Protein	30 mg./100 ml.
Blood	neg.
Sugar	neg.
Acetone	neg.
Sediment	neg.
Microscopic	few yeasts, few bacteria, 1–2 epithelial cells per field

TABLE 3

Blood Glucose	
Day 2	< 40 mg./100 ml.
Day 3	< 40 mg./100 ml.

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