1944

Canine Leptospirosis

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Recommended Citation
DeValois, D. J. (1944) "Canine Leptospirosis," Iowa State University Veterinarian: Vol. 7 : Iss. 1 , Article 8.
Available at: https://lib.dr.iastate.edu/iowastate_veterinarian/vol7/iss1/8

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Canine Leptospirosis. It has been noticed at the Stange Memorial Clinic that the incidence of canine leptospirosis has been steadily increasing. Definite diagnosis of this condition has not been established in all suspected cases, but symptoms and post-mortem lesions commonly seen in this clinic often point to one or more of the various syndromes of the disease. The attention of the general practitioner especially is called to the role which this condition may be playing in the gastrointestinal disturbances of the canine. Many times gastrointestinal symptoms are the most spectacular ones shown, and unless the practitioner is aware of the possibility of leptospirosis, he may very easily make a mistaken diagnosis.

Differential Diagnosis

The majority of cases of leptospirosis, black-tongue and enteritis of other causes all greatly resemble one another and considerable difficulty may be experienced in establishing a diagnosis. Mercury, arsenic, phosphorus and other heavy metal poisonings also produce the severe gastrointestinal symptoms, but usually history will serve to differentiate these poisonings. Laboratory diagnostic tests are necessary to definitely differentiate all these conditions which can be given only a tentative diagnosis clinically. Briefly, one may state that in black-tongue, extensive necrotic lesions are found in the oral cavity, whereas in leptospirosis the mouth lesions are not marked. Also a history of poor feeding would indicate black-tongue. Distemper is found in young dogs as a rule; leptospirosis is usually found in older dogs. One cannot rely upon this differentiating characteristic when not supported by other information, however.

It is also well to bear in mind that as this disease becomes increasingly prevalent, it may become a serious public health problem as *Leptospira icterohaemorrhagica*, one of the causative agents, is pathogenic to rats and humans in addition to dogs. In humans it causes a serious condition characterized by fever, icterus and extensive petechiation of the parenchymatous organs called Weil’s disease.

A recent case admitted to the Stange Memorial Clinic serves to illustrate this brief discussion. The patient was a 9-year-old Pointer which entered the clinic on May 15, 1944. The anamnesis presented with the case was that the patient had been vomiting for about a week, had a poor appetite and had been markedly depressed. Upon examination the patient was found to be icteric; hence leptospirosis was immediately suspected. Treatment was initiated at once with 40 cc. of anti-canine leptospirosis serum ad-
ministered subcutaneously and 700 cc. of an isotonic dextrose and saline solution given intravenously by the drip method. A urine sample was collected and taken to the laboratory for microscopic examination under dark field illumination. The presence of spirochetes typical of the genus Leptospira was demonstrated.

Necropsy

The patient expired a few hours later and a post-mortem study on the following day revealed lesions typical of the disease. Marked icterus was noted on all mucous membranes, in the fat, and in the subcutaneous connective tissues. A severe hemorrhagic gastritis, duodenitis and jejunitis were seen together with acute toxic hepatitis and hemorrhage of the lungs. The typical absence of extensive oral lesions was also seen.

The case which has been described here is typical of condition caused by the spirochete. It is true that variations are seen from this pattern of symptoms and lesions but the condition as manifested in this area resembles this case closely.

REFERENCES


Canine Gastroenteritis. Severe untreated cases of gastroenteritis terminate fatally in 2 to 7 days. In less severe cases death may occur from exhaustion in 1 to 2 weeks. Recovery in any instance is slow and convalescence is prolonged. The most unfavorable symptoms are great acceleration of pulse, profuse and hemorrhagic diarrhea, obstructe constipation, dehydration, anuria, intoxication and injected hemorrhagic mucous membranes followed by rapid emaciation and finally exhaustion.

On June 11, 1944, a 4-year-old Boston Terrier bitch was brought to the Stange Memorial Clinic with a history of having been off feed since June 5, and vomiting since June 7. Examination revealed the animal weak and depressed. The mucous membranes were injected and a sweetish odor was detected on the breath. Extreme dehydration was also noted. A diagnosis of gastroenteritis was made and symptomatic treatment begun.

An intravenous injection of 600 cc. of normal saline containing 3.5 percent dextrose was given on the first and again on the second day after admission in an effort to allay the dehydration and intoxication. The dog was observed to be taking water on the third day so intravenous injections were suspended for 2 days. The saline and dextrose administration was repeated on the fifth day.

The sixth day after entrance the saline injections were again suspended and thereafter the animal's own water intake was sufficient to overcome dehydration. The patient was allowed to exercise as her condition improved. It was noted that the depression was reduced greatly when out of doors. Feces were passed for the first time during treatment. These were passed with difficulty and showed evidence of blood. A fecal examination was made and found negative for parasite ova. Two No. 11 kaolin capsules were given on the seventh and eighth days. The frequent administration of peptone was continued. The dog began to eat solid food on the ninth day, at which time 2 No. 11 capsules of kaolin were given as the feces still showed evidence of blood. Exercise and the kaolin protective capsules were continued until the dog was released 5 days later. During this time the dog showed a steady improvement.

When the case was dismissed from the hospital the owner was advised to give a No. 11 capsule of kaolin as often as the dog's condition indicated.

Lymphocytoma in a Holstein Bull.
On May 27, a 7-year-old Holstein bull was brought to the Stange Memorial

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