CASE REPORT

VITAMIN E AND SELENIUM RESPONSIVE MYOCARDIAL DEGENERATION IN DOGS

P. D. GREEN AND J. W. H. LEMCKERT*

Introduction

There have been few cases reported of myopathy in dogs that were attributable to vitamin E deficiency (2, 4). However, recent experimental work has shown that vitamin E/selenium deficient diets can produce degeneration of the skeletal musculature and myocardium of growing dogs (5). This report deals with a recurring incidence of multiple acute deaths in a kennel due to acute myocardial degeneration. No further losses occurred after treatment with vitamin E and selenium preparation was instituted.

History

The kennel involved housed approximately 15 purebred Dachshunds when the first deaths were noted in January 1974. Over the period of one week, four mature dogs which were normal the night before were found dead the next morning. No changes in management had been made during recent months, and conditions of housing were acceptable. No potentially toxic compounds were identified on examination of the premises. Therapy prescribed at the time (see treatment) appeared to stop mortality.

The syndrome recurred in February of 1975 and January of 1976, with a similar history and the loss of three dogs both times. Mortality again ceased with treatment. Enquiries into the diet of these animals revealed that the owner was in the habit of purchasing dry food[1] in 500 pound lots, and that at the time of year when deaths occurred, the dogs were consuming feed that had been delivered in July or August of the previous year.

Pathological Findings

Gross examination of carcasses from the first deaths revealed no obvious abnormalities. Formalin fixed pieces of liver and heart as well as an intact carcass for necropsy were forwarded to the Provincial Veterinary Laboratory, Fredericton, New Brunswick. Gross lesions in the carcass were limited to petechial hemorrhages on the surface of the pancreas.

Histological examination of specimens from these submissions, and of formalized specimens from successive animals showed that lesions were limited to the liver, lung and myocardium. Hepatic changes consisted of centrolobular congestion, with slight swelling of the hepatocytes and increased granularity of the cytoplasm. Involvement was general throughout the liver, with prominence in the central vein area. Moderate pulmonary congestion and edema was noted, with a few macrophages being present in the alveoli.

Sections of the myocardium stained with H & E showed acute degeneration of the myocardial fibres, characterized by loss of striations and a granular appearance of the cytoplasm. The nuclei of the muscle cells were enlarged and pale, and vacuolated in appearance; however, the sarcolemmal sheaths were intact. The muscular layer of the coronary artery showed similar changes, with swelling of fibres and their nuclei, and with increased granularity of the cytoplasm. Application of Hematoxylin-Basic Fuchsin-Picric Acid stain (3) showed that the areas of myocardial degeneration were generally around the large arterioles throughout the myocardium, particularly in the ventricular musculature and the papillary muscle regions.

Treatment

After negative gross examination of the first carcasses, non-specific treatment was instituted, consisting of cortico steroids[2] and vitamins.[3] When a diagnosis of myocardial dystrophy was made, oral vitamin E/selenium (U.I.D. for three days)[4] and further vitamin E[5]

*Veterinary Laboratory, Agricultural Regional Building, Postal Bag 1, Airdrie, Alberta T0M 0B0 (Green) and Avenue Animal Hospital, 507 Rothesay Avenue, Saint John, New Brunswick E2J 2C6 (Lemckert).

[1]Gaines Meal, General Foods, Toronto, Ontario.

[2]Flucort tablets, Syntex, Montreal, Quebec.

[3]Vi-Sorbit, Norden, Lincoln, Nebraska.

[4]Seltoc Capsules, (Sodium selenite 2.19 mg and vitamin E 68 I.U.), Burns Biotec Laboratories, Oakland, California.

[5]Vitamin E capsules, (200 I.U.), Webber Pharmaceuticals, Rexdale, Ontario.

supplementation (U.I.D. for ten days) was prescribed. No further deaths occurred after that time, and when the syndrome recurred in subsequent years, mortality ceased when oral vitamin E[5] was instituted.

Discussion

Recurring multiple acute deaths in housed dogs is an unusual occurrence. Outbreaks of infectious disease will generally result in clinical signs of disease before death, and exposure to toxic compounds can generally be related to their use. In this case the pattern of occurrence in relation to the feed purchased, together with the findings of myocardial dystrophy, suggest that the condition was nutritional in origin. The immediate cessation of deaths when vitamin E/selenium supplementation was provided would indicate that these compounds were related to the manifestation of the syndrome.

Nutritional muscular degeneration has been described in many species, and has received much attention in domestic animals, however few reports of this occurrence in dogs can be found. One, from New Zealand, describes a severe acute polymyopathy in an adult sheepdog fed on mutton from selenium deficient sheep, and also mentions the occurrence of congenital myopathy in sheepdog pups resembling congenital white muscle disease of sheep (4). Recent work on experimentally induced deficiency in dogs showed that diets lacking the vitamin E and selenium produced a syndrome of muscular weakness which was progressive over a period of ten to 15 days, however acute deaths did not occur (5).

A decrease in the concentration of the fat soluble vitamins is known to occur in feeds subjected to long storage, and especially in prepared feeds the presence of unsaturated fats is known to affect levels of vitamin E (1). In this case, storage was indeed prolonged by normal standards, and it may be postulated that vitamin E levels were low. The conditions of purchase described in this case are not unusual through manufacturers' special bulk purchases offers to dog breeders, and as consumption by the smaller breeds is limited, it is possible that this situation may occur in other kennels where feed is stored for long periods. It would be desirable that, when feed is sold in bulk quantities, the date of manufacture appear on the package, or that some indication of an expiration date be included on the label.

Summary

Multiple acute deaths due to acute myocardial degeneration occurred in a commercial kennel. Further losses were prevented when vitamin E/selenium supplementation was instituted. Prolonged storage of dry feed may have been responsible for the deficiency state.

Résumé

Plusieurs morts soudaines, attribuables à une dégénérescence aiguë du myocarde, se produisirent dans un chenil commercial. L'addition de vitamine E et de sélénium à la nourriture arrêta les mortalités. L'entreposage prolongé de cette nourriture commerciale sèche pourrait être responsable de la condition qui sévissait dans ce chenil.

References

1. Blood, D. C. and J. A. Henderson. Veterinary Medicine. 3rd Ed. pp. 740–756. Baltimore: Williams and Wilkins. 1968.
2. Kaspar, L. V. and L. S. Lombard. Nutritional myodegeneration in a litter of Beagles. J. Am. vet. med. Ass. 143: 284–288. 1963.
3. Lie, J. T., K. E. Hollie, W. R. Kampa and J. L. Titus. New histochemical method for morphologic diagnosis of early stages of myocardial ischaemia. Mayo Clinics Proc. 46: 319–327. 1971.
4. Manktelow, B. W. Myopathy of dogs resembling white muscle disease of sheep. N.Z. vet. J. 11: 52–55. 1963.
5. VanVleet, J. F. Experimentally induced vitamin E/selenium deficiency in the growing dog. J. Am. vet. med. Ass. 166: 769–774. 1975.