

FOOD-ASSOCIATED MEGAESOPHAGUS / POLYNEUROPATHY OUTBREAK IN LATVIAN DOGS: SUMMARY OF GROSS AND MICROSCOPIC LESIONS

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Introduction

In Latvia during 2014-2016 there was sudden, approximately 10x, increase in number of dogs diagnosed with megaesophagus. Upon further investigation changes in nerve impulse conduction, atrophy of muscles, and generalized weakness were recognized in these dogs therefore name megaesophagus/polyneuropathy (ME/PNP) was coined with this outbreak. Epidemiological investigation indicated that main risk factor for developing ME/PNP was exposure to brand A dry dog food. According to surveys, 20-25% of affected dogs have died.

Objectives

To characterize gross and microscopic lesions in dogs affected in food-associated ME/PNP outbreak in order to elucidate cause and pathogenesis of the disease.

Methods

All dogs that had x-ray confirmed ME and/or clinical signs suggestive of ME and had died were subjected to thorough necropsies if owners consented to necropsy. There were 2 groups of dogs – group 1 (n=13) – dogs necropsied in 2015 and group 2 (n=19) – dogs necropsied in 2016. For group 1, general, thorough necropsy was done without focus on particular system. For group 2 more extensive sampling of nerves, muscles, ganglia and brain was done. Tissue samples were stained with H&E. One dog in group 1 was severely autolytic therefore samples for histological examination from this dog were not collected.

Results

All necropsied dogs had ME. Most frequent gross and microscopic lesions in group 1 were PNP (83%), skeletal muscle atrophy (58%), enteritis (54%), aspiration pneumonia (46%) and tumors (25%). In the second group all dogs had lesions consistent with PNP and muscle atrophy at least in one location perhaps reflecting more extensive sampling. Additionally, dogs of the second group had frequent collapse of tracheal muscles (68%) and enteritis (58%). Liver degeneration was seen in 26% and marked dilation of urinary bladder in 21% cases. Tumors in the second group were seen in 2 dogs. Lesions in the esophagus consisted of muscle fiber degeneration, atrophy and occasional necrosis. There also appeared to be loss of ganglion cells; however controls are needed for comparison. Nerve lesions consisted of subperineurial edema and formation of Renault bodies. Examination of ultrathin sections from select dogs showed changes in axons and myelin sheaths ranging from subtle to massive and demonstrating clear proximodistal advancement. Not all nerves were affected and lesions varied widely in severity, more commonly affecting small diameter motor nerves of distal limbs and affecting hind limbs more consistently than front limbs. Involvement of *n. vagus* and its branches shows that parasympathetic branch of nervous system is affected as well as sympathetic. Histological findings support and are in agreement with clinical and neurological findings in affected dogs. Analysis of mild changes in ganglia, brain and few other tissues is ongoing since comparison with control dogs is needed.

Conclusions

Food-associated ME/PNP in Latvian dogs is distally enhanced intermediate axonopathy more severely affecting longest nerves in the body (vagal and sciatic nerves). Due to nerve damage secondary changes occur in skeletal muscle. Lesions are indicative of toxic polyneuropathy. The mechanism is unclear but may be similar to that induced by orally dosed acrylamide or organophosphates.