Letters

GENETIC DISEASE

Polymyositis and DNA collection in the Hungarian vizsla dog

WE are writing to raise awareness of a breed specific polymyositis that has recently been recognised in the Hungarian vizsla dog (Haley and others 2011) and to make a request for DNA samples from affected dogs.

Affected individuals are most commonly presented with difficulty in eating and drinking with hypersalivation. The condition is characterised clinically by dysphagia and masticatory muscle atrophy. Generalised muscle atrophy may also be seen. Other common clinical signs include regurgitation, exercise intolerance and pain or difficulty in opening the jaw (Foale and others 2006).

Diagnostic tests usually reveals elevated creatinine kinase in the acute phase (>1000 U/l) .Thoracic radiographs may show evidence of megaoesophagus. Electromyography may reveal spontaneous activity within affected muscle, particularly the tongue and pharynx. Affected individuals have negative antibody titres for 2M muscle fibres and negative antibody titres for acetylcholine receptors. Definitive diagnosis is by muscle biopsy. Histopathology of affected individuals may show multifocal inflammatory changes (lymphocytic, lymphohistiocytic or plasmacytic myositis with or without fibrosis). Inflammatory changes may be absent in end stage disease or if non-representative sites are biopsied.

The disease appears to be immune-mediated and some dogs have responded well to appropriate immunosupressive therapy, although the prognosis remains guarded. The dogs showing the best response to therapy are generally individuals where diagnosis and treatment are initiated early in the course of the disease.

We believe that there is a genetic basis to this disease and are currently collecting DNA from affected individuals that fit our phenotypic criteria. Where possible DNA is also being collected from direct relatives of affected individuals (siblings, sires, dams).

Phenotypic criteria for inclusion are vizsla with at least one of the following in addition to dysphagia and have negative 2M and acetylcholine receptor antibody titres:

- creatinine kinase over 1000 U/l;
- exercise intolerance;
- megaoesophagus identified on thoracic radiographs;
- electrophysiological changes consistent with muscle disease; or
- histopathological changes consistent with myositis.

If blood has been collected from an affected dog for other clinical purposes please send the residual EDTA sample (fresh) to the CIGMR (Centre for Integrated Genomic Medical Research, University of Manchester) in accordance with the protocols detailed in Form 1 of the DNA collection pack that can be accessed on www.veterinary-neurologist.co.uk/vizsla.htm

Alternatively, we are able to provide Oragene collection kits for DNA collection (via saliva). To obtain a kit please contact Diane Addicott at the address below, telephone 01576 202 258, e-mail: diane@murrayfield.wanadoo.co.uk

We would also be interested to hear from colleagues who have seen Hungarian vizsla dogs with either steroid responsive meningitis-arteritis or immune-mediated polyarthritis.

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References

FOALE, R. D., WHITING, M. & WRAY, J. D. (2006) Myositis and pharyngeal dysphagia in Hungarian vizslas. Proceedings of 50th BSAVA Congress. Birmingham, April 3 to 6, 2008. Clinical research abstract

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