Degenerative myelopathy

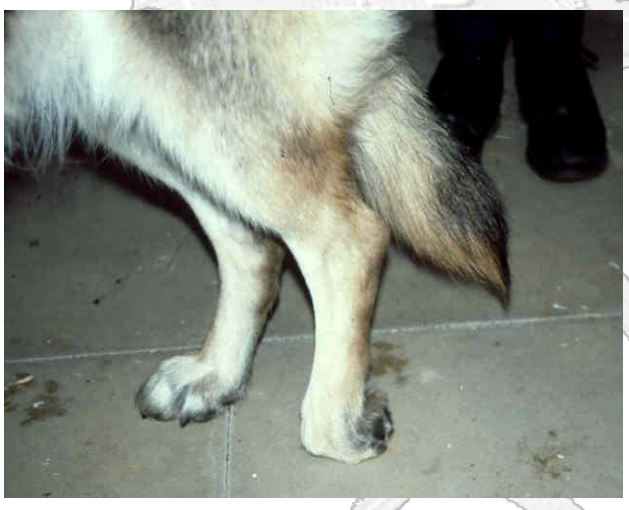
Canine degenerative myelopathy (DM or chronic degenerative radiculomyelopathy / CDRM), is characterised by slow progressive lost of hindlimb function. Over months to years the paralysis eventually ascends to involve the thoracic limbs. The German Shepherd dog (GSD or Alsatian) isthe breed most commonly affected however it is also seen in many other breeds.

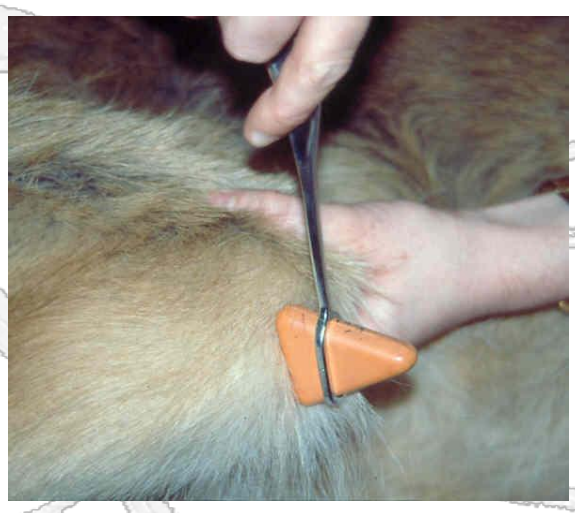
Cause of degeneration myelopathy

Dr Joan Coates and her team at the University of Missouri. has pioneered work into understanding this disease. Genome studies have revealed a mutation within the canine SOD1 gene in several dog breeds including the German Shepherd, Pembroke Welsh corgi, Boxer, Rhodesian ridgeback and Chesapeake Bay retriever. Affected dogs require 2 copies of the mutation for disease to develop -i.e. it is an autosomal recessive inheritance. Mutation in superoxide dismutase-1 (SOD1)results in overproduction of damaging reactive oxygen species (ROS) i.e. oxidative or free radical injury to the nervous tissue. Microscopic examination of spinal cords from affected dogs has revealed myelin and axon loss affecting the lateral white matter and neuronal cytoplasmic inclusions that bind anti-superoxide dismutase 1 antibodies. Canine DM is a spontaneously occurring animal model for amyotrophic lateral sclerosis (ALS) (Awano and others 2009).

Clinical signs of degenerative myelopathy

Affected dogs are generally 8 years of age or older. Typically owners will notice a subtle weakness of one pelvic limb which progresses to a tendency for the limb to be dragged and the toenails worn. Within weeks to months the other hind limb becomes involved. The gait is often described as uncoordinated or drunken with a tendency to fall when cornering. However despite the disability the dog is often keen to exercise and does not have spinal pain. Neurological examination at this time generally reveals a strong knee jerk reflex (hyperactive patella spinal reflex), consistent with disease of the spinal cord between the third thoracic vertebrae and the third lumbar vertebrae. As the disease progresses, the dog becomes less able to walk and/or support their own weight. At this time the knee jerk reflex can be lost. Later in the disease course the dog can develop faecal incontinence and if euthanasia is delayed, the clinical signs will ascend, causing flaccid tetraparesis. The presentation of the disease can vary between different dog breeds with some of them (e.g. Corgi and Rhodesian Ridgeback) having more obvious disease of the(lower) motor neuron rather than the spinal cord (Coates 2009).





Above Left *Proprioceptive deficit in a German Shepherd with degenerative myelopathy*

Above Right *In degenerative myelopathy in the German Shepherd the knee jerk reflex is brisk initially and then lost as the disease progresses*

Diagnosis of degenerative myelopathy

A DNA test for the mutation causing the disease in German Shepherds and other breeds isavailable through the University of Missouri. Determining that a dog has the mutation does not prove absolutely that he/ she has the disease i.e. the mutation increases the risk rather than meaning an animal is predestined to develop the disease. Other causes of spinal disease should be ruled out e.g. with diagnostic tests such as MRI (normal) and cerebrospinal fluid analysis (cell counts normal; protein may be raised). In some cases the dog

may have more than one potential or actual cause of the spinal cord disease e.g. disc disease and degenerative myelopathy.

Management of degenerative myelopathy

Mutation in superoxide dismutase-1 (SOD1) results in overproduction of damaging reactive oxygen species (ROS) i.e. oxidative or free radical injury. There is unfortunately no treatment however since the nerve is susceptible to free radical injury then there is a rational for supplementing the diet with free radical scavengers such as such as Vitamin E, Omega-3-fattyacids, Gamma linoleic acid and, L-Carnitine. However our experience is that this only helps in the early stages of the disease

Keeping the dog active has been shown to slow deterioration i.e. maintain walking and other exercise. It is worth arranging a session with a chartered physiotherapist (e.g. contact ACPAT,www.acpat.org, acpat@calra.net) with the aim of learning techniques to maximise remaining ability and neurological function. If possible hydrotherapy is also recommended, preferably also under the guidance of a chartered physiotherapist (e.g. contact Canine Hydrotherapy Association www.k9hydrotherapy.co.uk). If the dog has degenerative joint disease (i.e. arthritis) or other spinal disease then this should also be managed so that pain does not limit activity.

'Toe up Siatic slings' can improve the walking ability of some dogs. For more information see

www.orthopets.co.uk/Sciatic_Sling.html here The disease course of degenerative myelopathy can wax and wane. There appears to be an immune mediated component and some acutely deteriorating cases of degenerative myelopathy can benefit from short courses of corticosteroids at anti-inflammatories doses (not with concurrent non steroidal anti-inflammatory drugs).

Treatment in humans for the equivalent disease (Amyotrophic lateral sclerosis or ALS) is also mainly supportive and palliative. Riluzole is the only drug that has been shown to extend survival. To the author's knowledge this drug has not been tried in dogs (it is very expensive).

Degenerative myelopathy and carts

Many people elect to euthanize their pet when the dog is unable to support themselves to stand /walk. However some people feel that a quality of life can be maintained and by using a cart the dog can yet again enjoy the pleasures of "going for a walk". However a limiting factor can be the size of the dog (and therefore cart). If the forelimbs are weak then a cart is unlikely to be suitable option. Incontinence can also be a management problem.

Freddy lost the use of his hindlimbs though degenerative myelopathy. However this didn't stop him enjoying a full life and he lived many happy years after the diagnosis was made.

References

Awano T, Johnson GS Wade C M Katz M L Johnson GC, Taylor J F, Perloski M Biagi TBaranowska I Long S March PA Olby NJ Shelton G.D, Khan S O'Brien D P Lindblad-Toh KCoates JR Genome-wide association analysis reveals a SOD1 mutation in canine degenerative myelopathy that resembles amyotrophic lateral sclerosis PNAS, 2009, vol. 106, no. 8, 2794–2799.Coates JR Translation of Canine Degenerative Myelopathy To Human ALS 2009 ACVIM Forum/ Canadian VMA Montreal Convention p 295-7