FAQ’s About Dogs With Degenerative Myelopathy

By Eddie’s Wheels

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What is Degenerative Myelopathy

DM is an autoimmune disease similar to ALS in humans. The DM gene has been identified in over 100 breeds of dogs, but is most common in German Shepherds, boxers, and corgis. We have seen it in pugs, Rhodesian Ridgebacks, Bernese Mt. dogs, airedales, wheaten terriers, wire haired fox terriers, even a coyote.

What are the symptoms of DM?

The first sign is the sound of your dog’s toenails scraping the sidewalk, and then an increased lameness, usually on one side, with no signs of pain. Growing ataxia, wobbliness and instability of gait with rear legs scissoring, and the toes knuckling over within six months. As DM progresses, the rear legs become unusable, the bark changes and becomes increasingly hoarse. Some dogs become incontinent. Eventually, the front legs grow weaker and the core muscles grow lax. Even as dogs become increasingly weak, they remain engaged with their families.
What is the treatment for DM?

Unfortunately, there is no medical treatment to halt this disease. However, massage and regular exercise using assistive devices, such as a manual harness and a dog wheelchair can provide these dogs with a reasonably good quality of life.

How is DM diagnosed?

There is a simple genetic test available through the University of Missouri Vet School, www.caninegeneticdiseases.net - visit this website for the latest up-to-date information about the current research on DM.

When is it time to get my dog a wheelchair?

Most dogs will not accept assistance as long as they can get themselves around on their own, but once they start dragging their legs, it’s time. Most of the dogs we see here will continue to uses their rear legs for awhile when they first get their carts, as they still have a sense of weight-bearing and can move their legs from the hip, even if they are unable to properly place their feet. We encourage people to allow their dogs to use the cart as a walker for as long as possible. Once the rear legs no longer can aid in propulsion, it’s time to use the stirrups prevent foot dragging and wounds.
What kind of cart should I get for dog with DM?

We recommend our variable axle cart for dogs with DM. This axle allows caregivers the ability to change the balance of the cart to compensate for increasing weakness as the disease progresses forward. All the owner has to do is move the wheels forward on the axle assembly and adjust the support strap. Any standard cart can be upgraded to a variable axle cart. When ordering a cart, always ask us to check our stock of used carts to see if we have one that will fit your dog.

Notice the position of the wheels on the axle assembly. As Elliott’s front legs weaken, additional weight can be taken off his front end by moving the wheels forward on the axle assembly.

By Eddie's Wheels

Like all great businesses, Eddie's Wheels was created by Ed and Leslie Grinnell when they recognized that their need was shared by other pet owners. They developed a rough and ready wheelchair that can re-mobilize pets while they regain natural mobility.

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Mobility is the Gift of Life

Changing Lives

Mending Spirits

Restoring Independence

Eddie’s Wheels for Pets

Custom built in the USA
Engineered to match your dog’s disability
Warranted for the life of your dog

Durable and Affordable

888-211-2700 www.eddieswheels.com
While the cause of DM, is still unknown, the most recent breakthrough is in our ability to make a near-certain diagnosis of the disease ante mortem. Although there is still no effective treatment for DM, the magnitude of the recent advance in diagnosis cannot be over-emphasized. Without an understanding of the cause of a disease, proposed treatments are at best an educated guess.

Understanding the Cause:

Why was the cause of DM such a mystery for many years? Some of that answer relates to science and some to logistics. Pathologically, DM is characterized by symmetrical loss of both myelin and axons in the thoracolumbar spinal cord without any signs of inflammation. Coupled with the knowledge that the disease is slowly progressive, nutritional, toxic or genetic etiologies would be most likely. Equine DM is in part a nutritional disease, but nutrition in dogs with DM didn't seem to be any different than dogs without DM. Toxin seemed plausible, but none could ever be identified.

Some postulated the disease was immune-mediated despite the lack of inflammation in path samples, but the lack of response to prednisone and other immunomodulatory medications made this unlikely. That left a genetic etiology on the table. In some ways, this
theory made sense – the disease was seen most characteristically in certain breeds of dogs – German shepherds, collies, and boxers, Chesapeake Bay retrievers. The onset of disease in middle aged to older dogs was an argument against a genetic cause.

Logistically, DM was a hard disease to study as well. The diagnosis relied upon histopathology of the thoracolumbar spinal cord. Removing the TL spinal cord from a dog post-mortem is a very time-consuming process. It also did not help that dogs typically would not be euthanized until months to even years after the diagnosis was made. Maybe if the dog was diagnosed at a university teaching hospital and died in hospital, pet owners would have been happy to allow a necropsy and the removal of the cord for study. Months later, when the dog’s time had finally come, most owners did not enthusiastically volunteer to bundle their non-ambulatory 90 pound dog into the car for a three-hour drive to the university for euthanasia and necropsy.

DM sat was a problem waiting for an answer, when slowly things started to change in the world of veterinary neurology. More neurologists became available in private practices, which meant many dogs with DM were now located physically closer to their neurologist, making it harder for them to be lost to follow-up. When the time came to euthanize the dog, not only was it more likely the dog would be near its neurologist, but also that the neurologist was equipped to actually remove the spinal cord post-mortem. At the same time, on a scientific level, molecular genetics was advancing by leaps and bounds. The University of Missouri College of Veterinary Medicine decided to make a big push to study genetic diseases of animals.

SOD2: Shortly thereafter, a young veterinary neurologist, Joan Coates, joined Missouri’s faculty and she decided to tackle the DM question. She assembled a team of researchers in clinical neurology, neuropathology and genetics, invited all veterinary neurologists to participate in her project, and spent years communicating with breed organizations, breeders and individual pet owners. Eventually, she had enough DNA samples from dogs with histopathologically confirmed DM to start her genetic studies. She discovered that dogs with DM have a mutation in a gene known
as SOD2.

Would a mutation in the SOD1 gene make sense for dogs with DM? Yes, absolutely. "SOD1" stands for superoxide dismutase 1. This is a gene responsible for repairing oxidative damage to cells. What types of cells are most likely to be affected by oxidative damage? Any cell containing a high amount of lipid. Myelin has high lipid content. Myelin-rich white matter in the nervous system would be expected to be one of the most sensitive tissues in the body to oxidative damage — the exact pathology seen in dogs with DM. It would even make sense that DM doesn’t manifest until later in life, as it would take many years to accumulate enough oxidative damage to become symptomatic. It explains why dogs with DM don’t respond to treatment — by the time they show symptoms, the damage is already done.

Finally, Dr. Coates checked to see if there were any known diseases in other species caused by mutation of the SOD1 gene. It turned out that there is a human disease caused by SOD1 mutation — a rare spinal cord variant of Lou Gehrig’s disease — and has similar symptoms and clinical course to canine DM. Everything started to make sense — DM is the result of inheriting two bad copies of the SOD1 gene. Since this discovery in 2010, many dogs that tested positive for DM have since passed away and dozens of these have been necropsied. Only one has turned out to not have DM, supporting the validity of the test.

**Degenerative Myelopathy Testing:**

Genetic testing for DM is now available through the Animal Molecular Genetics Laboratory at the University of Missouri. Either blood samples or cheek swabs may be submitted. The cheek swabs are a little cheaper, but the blood samples contain much more DNA. DNA is banked by the lab when blood samples are submitted but not with cheek swabs. Therefore blood samples are preferred whenever possible, as they contribute to the overall study of the disease. A sample submission form and instructions are attached. Interpreting a DM test is straightforward if you understand what the test tells you. Two bad copies of the gene means the dog will likely develop the disease during its natural lifespan.
Other causes of a TL myelopathy could be present, so ideally spinal MRI +/- spinal tap are necessary to exclude other diseases. On practical grounds, most pet owners would be reluctant to perform spinal surgery on a DM positive dog, so the gene test alone may make sense for a lot of patients.

**Finding a Cure:**

The next step in DM research is to look for a cure. The best hope will be to start treatment before a dog is symptomatic, so eventually early testing of dogs of affected breeds may be recommended. Dr. Coates is collaborating on clinical trials to treat the human form of DM and hopefully some of this work will spill over into veterinary medicine soon.

**Prognosis:**

Current management recommendations for DM center around maintaining a sufficient degree of physical fitness to allow dogs to better compensate for their neurologic impairment. We typically recommend a formal physical rehabilitation program with a veterinarian whose practice is dedicated to rehabilitation. This currently seems to be the biggest factor in keeping these dogs functional for as long as possible, and most rehab vets are experts at managing and recommending various assistive devices such as slings, booties and carts as needed. It is also important to micromanage the dog's overall health and stress levels, as it seems periods of physiological or psychological stress are accompanied by decline (often transient) in neurologic function in dogs with DM. It seems to be very easy to upset the apple cart in DM dogs. Antioxidant therapy and good nutritional support may be helpful in these patients, but may actually be the least important factor in affected patients.

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**By Stacey A. Sullivan, DVM**

Dr. Stacey Sullivan received her Doctor of Veterinary Medicine degree from North Carolina State University in 1993. She moved to Auburn University for her internship, followed by a residency in neurology and neurosurgery at the University of Georgia.  

Historically, in veterinary medicine, the clinical diagnosis of canine degenerative myelopathy was a diagnosis of exclusion – if the symptoms were consistent with Degenerative Myelopathy and spinal imaging, and CSF were normal, DM was considered the most likely diagnosis. There was, however, no confirmatory test.

In the past three years we have fortunately seen real breakthroughs in our understanding of this all-too-common disease. At long last, we are able to make a near-certain diagnosis of the disease ante mortem. Although there is still no effective treatment for DM, the magnitude of the recent advance in diagnosis cannot be over-emphasized. Without an understanding of the cause of a disease, proposed treatments are at best an educated guess.
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<table>
<thead>
<tr>
<th>Antioxidants Used in the Management of DM</th>
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<tbody>
<tr>
<td><strong>Vitamin E:</strong> 1000-2000 IU per day</td>
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<tr>
<td><strong>Vitamin C:</strong> 1000-2000 mg per day</td>
</tr>
<tr>
<td><strong>B-complex:</strong> High potency 2 capsules per day or stress formulation 1 capsule per day</td>
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<tr>
<td><strong>Selenium:</strong> 100 (small dog) to 200 (large dog) ug/day. Excessive doses are toxic.</td>
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<tr>
<td><strong>Aminocaproic Acid:</strong> 250 mg/mL suspension, mix 2 ml with 1 ml chicken broth and give q8h</td>
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- See more at: [http://www.asgvets.com/canine-degenerative-myelopathy/#sthash.T9t2WLz0.dpuf](http://www.asgvets.com/canine-degenerative-myelopathy/#sthash.T9t2WLz0.dpuf)