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Degenerative Myelopathy in the German Shepherd

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The treatments just listed are initial treatments only. They should be followed with one of several methods of long term therapy. The method utilized depends on the owner's ability to prevent the dog's exposure to sunlight and how effective it is. Some temporary protection may be provided by the frequent use of a black-tipped marking pen. For a more permanent replacement of the lost pigment, the affected area can be tattooed with black ink. This is usually accomplished by using a Nicholson tattoo vibrator. This procedure must be done under general anesthesia, and it is usually necessary to repeat the treatment at least three times at 30 to 60 day intervals. When this procedure is completed, the tattooed skin is a slate gray color. Annual "touch-up" tattooing is usually necessary. Tattooing is most beneficial in mild cases that are treated early in the course of the disease. The most important thing to remember prior to tattooing is to be sure the inflammation is well under control. Otherwise, the tattooing will only aggravate the inflammation.

Sun-screening agents have also been used such as quinacrine hydrochloride, which is given orally at a dose of 50 mg., 2 to 3 times a day for one month, after which the dose is reduced to 50 mg. once or twice weekly. Para-aminobenzoic acid has also been used successfully as a sun-screening drug at a dosage of 2 gm., 4 times daily. The disadvantage of these drugs is the number of times they must be given.2

**Summary**

The most important factor in treating "collie nose" appears to rest on the importance of getting treatment started early and keeping it up at regular intervals. Another thing to remember is that since this is an inherited abnormality it is not advisable to use dogs with this problem as breeding stock. However, with proper management and therapy, this is a disease which we, for all practical purposes, can keep fairly well under control.

**Bibliography**


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**Degenerative Myelopathy in the German Shepherd**

by Kathy Mayberry* and Robert W. Carithers D.V.M., M.S., Ph.D.†

Older large breed dogs, primarily of the German Shepherd type, may be presented with progressive ataxic spastic paresis. There seems to be no sex predominance. The lesion found on necropsy is diffuse degeneration of spinal cord myelin and axons in all fiber tracts, most extensive in the mid-thoracic region, but not associated with intervertebral disk herniation.

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spondylosis or osseous metaplasia of the dura. The condition is thought to be primary spinal cord degeneration of unknown cause.

Introduction

The practicing veterinarian is occasionally presented with a middle-aged or older dog, frequently but not always a German Shepherd, exhibiting progressive, asymmetrical motor weakness of the pelvic limbs of several months duration. It is not an uncommon occurrence, but the pathogenesis remains unexplained. Previously the progressive paresis has been ascribed to dural ossification (formerly known as ossifying pachymeningitis) or multiple small intervertebral disk protrusions. A list of other diseases that would also evidence posterior paresis must include spinal cord and vertebral body tumors, toxoplasmosis, distemper myelopathy, trauma, luxations or subluxations, vascular disease (infarction, thrombosis or hemorrhage), necrotic myelitis, septic meningitis and epidural abscess, non-septic myelitis, and hypoglycemia. The evidence gathered on necropsy and histologic exam may not correlate well with any of these disorders. There seems to be a primary spinal cord degeneration affecting aging German Shepherds, and a few other large breeds.

Case #57421

On June 24, 1974, an eight-year-old male German Shepherd was presented to the clinic with bilateral asymmetrical pelvic limb weakness of six months duration. The dog was alert, and all cranial nerve and thoracic limb reflexes were intact. Good muscle tone was noted in the rear legs, but tactile placing (knuckling) was poor, and the patellar knee-jerk was strong especially in the right leg.

Blood work was normal and radiographs of the lumbar area revealed nothing significant. Hip dysplasia was not visible on radiographs submitted by the referring veterinarian. The tentative diagnosis was degenerative myelopathy.

On post mortem, the animal exhibited plaques of ossification on the ventral surface of the cord at L₂, L₃, and L₄, and also high in the cervical region. Histologically there were foci of calcification scattered along the spinal nerves.

Case #62306

On September 14, 1974, the clinic was presented with a nine-year-old female German Shepherd with bilateral, asymmetrical pelvic limb weakness of nine months duration. The dog had extreme difficulty rising without assistance, and was very incoordinated behind, knuckling with both feet and often crossing the rear legs. Incontinence of the bowels and occasionally of the bladder were noted by the owner. Moderate atrophy of the lumbar region and pelvic limbs was exhibited, and the animal was unable to raise it's tail or to wag it.

The cranial nerve and thoracic limb reflexes were intact. The right rear leg was weaker than the left, but both exhibited pronounced knuckling. The right patella was areflexic, the left was hyporeflexic. The animal seemed to be suffering more from mental confusion than from pain. The blood work was normal, and radiographs of the hips and lumbar region were insignificant.

On post mortem, plaques of ossification were found on the ventral surface of the cord at L₃ and L₄. The diagnosis was degenerative myelopathy, with the lower motor neuron signs due to dural ossification at the origin of the femoral nerve.

Upon histopathologic examination of the spinal cords of both of these animals, loss of axons and myelin sheaths was noted, especially in the thoracic region. In cross-sections, the staining density of the white tracts was reduced due to fragmentation of axons and the formation of myelin balls. On longitudinal section this was shown by oval or linear “voids” in the fiber tracts.

Discussion

Dural ossification occurs in many dogs of many breeds without neurologic signs.
It is unlikely that this alone would cause signs of this severity. No areas of myelitis were found associated with the plaques.

In a study of twenty-two dogs affected with degenerative myelopathy by Dr. D. R. Averill, Jr., every dog had cervical, thoracic and lumbar destruction of ascending and descending tracts. Lesions were most extensive in the middle to lower middle thoracic region and become less severe cranial or caudal to that point. Sites of intervertebral prominences, spondylosis or dural ossification were not associated with the lesions. In this study, cross-sections of the cords revealed a loss of myelin sheaths and axons, primarily axonal degeneration, and reduction of myelin to neutral fat. The astrocyte cells were frequently arranged in rows, suggesting that they had replaced previously degenerated fibers.

Because of this, the most common clinical signs will include asymmetry of strength, presence of crossed-extensor reflexes, hyperreflexic patellar knee-jerk, worn toe nails from dragging the rear foot or feet. The front leg reflexes will be intact, the animal will show a "dropped" tail, absence of sensory loss, pronounced rear leg knuckling, atrophy of the rear leg muscles, difficulty in rising and usually a lack of pain. A careful physical and neurological exam is required to distinguish between German Shepherd paresis and coxofemoral hip dysplasia. Some dogs may have both.

Diagnosis should be based on a history of slow onset and several months duration, neurological exam indicating upper motor neuron disease and radiographs to rule out other causes of paresis. Myelography will help rule out extra-axial lesions. The spinal fluid is usually normal in dogs exhibiting German Shepherd myelopathy.

Very little response to medication is seen. Steroids, phenylbutazone, salsalates, muscle relaxants and vitamin and mineral supplements are of no use. Medical or surgical treatment is very discouraging.

The progressive paresis results from a progressive loss of long-tract fibers in the spinal cord. The exact cause is unknown. Focal injury can be excluded because of the loss of fibers in all tracts at many levels. Neither were the lesions anatomically related to the sites of osseous proliferation or to spondylosis. Vascular insufficiency would have caused a primary lesion in the central region of the spinal cord gray. A similar B12 deficiency and several familial degenerations of nerve and muscle fibers exist in man. But the exact pathogenesis of German Shepherd degenerative myelopathy remains in question. At present, myelopathy in aging German Shepherds seems to be a primary neuronal atrophy with selective vulnerability of the thoracic spinal cord.

References