Distal Symmetrical Polyneuropathy in a Dog

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Abstract. A 1.3-year-old Great Dane dog had a chronic progressive neurologic disease clinically expressed as a distal symmetrical polyneuropathy characterized by weakness and bilateral atrophy of bulbar and distal appendicular musculature. Qualitative and quantitative studies showed neurogenic atrophy of muscles below the elbow and stifle. There was Wallerian-type degeneration, Schwann cell proliferation and cell bands of Büngner, and marked depletion of medium (5 to 8 μm) to large (9 to 15 μm) diameter myelinated fibers in the distal parts of appendicular and laryngeal nerves. Sensory (saphenous and superficial radial) and autonomic (sympathetic and dorsal vagal trunk) nerves were affected to a lesser degree. A distribution of distal axonal degeneration suggested a dying back process. The disease differed from classical dying back disorders by absence of axonal degeneration in selected pathways of the central nervous system.

Distal symmetrical polyneuropathies have been recognized in hereditary, toxic, nutritional, and metabolic disorders in man [5]. The disease process may involve primarily axons, Schwann cells or neuronal perikarya. The distal distribution may result from multiple small lesions scattered diffusely throughout the nerves producing proportionately more damage to the longest fibers; or from distal axons dying back from their peripheral connections [8]. Recently, a giant axonal neuropathy characterized by a distal axonal degeneration was reported in the dog [13]. We present morphologic and morphometric data from a dog with a form of spontaneously occurring distal symmetrical polyneuropathy different from that report.

Case Report

A 1.3-year-old male Great Dane dog was presented to the Auburn University Small Animal Clinic with a 4-week history of progressive hind limb weakness. The dog had an unusual prancing/skipping hind limb gait and muscle atrophy in the hind limbs. He had received routine rabies and canine distemper vaccines and had no history of trauma. A littermate was normal. The owner had another dog and a cat that were clinically normal. The owner sprayed house plants with a number of
The dog was bright and alert on neurologic examination [31] and walked with a degree of hypermetria in both hind limbs. There was no flexion of the hocks during movement. Cranial nerve function was normal. Postural reactions were depressed in both fore and hind limbs. Static and dynamic muscle tonus was normal in all limbs, as were segmental spinal reflexes. Panniculus reaction was normal over the lumbar and thoracic spine, and pain perception was within normal limits over the entire body. Cervical mobility was normal in horizontal and vertical planes and no pain or muscle spasms were evident on cervical spinal palpation. Muscle atrophy was bilateral in muscles distal to the stifle joints. Palpation of hind limb musculature caused some discomfort to the dog. There was no evidence of joint swelling or pain on joint manipulation in any limb. Sphincter function was normal.

The clinical condition of the dog deteriorated progressively during the 4-week period of hospitalization. Muscle atrophy, bilaterally symmetrical in development, became more pronounced in distal forelimb areas (below the elbow) and there was atrophy in the temporalis, masseter and digastricus muscles. Distal tendon reflexes (gastrocnemius) were depressed. The dog was killed and necropsied.

Materials and Methods

Ancillary procedures included routine hematologic and blood chemical profiles, spinal radiography, myelography, electromyography, nerve conduction studies, cerebrospinal fluid examination (for protein and cytology), and thyroid function tests. The proximal (biceps femoris) and distal (gastrocnemius—lateral head, cranial tibial) hind limb muscles were biopsied and the common peroneal nerve at the level of the stifle joint was examined by fascicular biopsy [7]. Muscle specimens were frozen in isopentane, pre-cooled in liquid nitrogen. Serial sections were cut in a transverse plane at 8 µm with a cryostat microtome maintained at −20°C and stained according to the following histologic and histochemical techniques: modified Gomori's trichrome [17], reduced nicotinamide adenine dinucleotide-tetrazolium reductase (NADH-TR), routine (pH 10.0) adenosine triphosphatase (ATPase) and modified ATPase (pH 4.3) [11]. Muscle fibers were subtyped histochemically as described [6]. The fascicular nerve sample was suspended in 3% glutaraldehyde fixative for 3 hours, divided in half, and each half fixed in 1% osmium tetroxide for 1 hour. One half was dehydrated in graded ethanol and embedded in Epon 812. The other half was processed through glycerol, and single fibers were teased as described [16]. Semi-thin sections (1 to 2 µm) were cut, stained with paraphenylenediame [19] and examined with a light microscope. Silver to grey ultra-thin sections were cut from suitable areas, stained with uranyl acetate and lead citrate and examined with the electron microscope.

At autopsy, the brain, spinal cord, and representative samples of other tissues were immersed in 10% buffered formal solution. Samples taken from the following skeletal muscles were processed for histochemical staining: distal part of biceps femoris, vastus lateralis, triceps brachii (long and medial heads) and proximal parts of gastrocnemius—lateral head, cranial tibial, forelimb superficial and deep flexors and deltoideus. Samples from the following nerves were immersion-fixed in glutaraldehyde and processed for teased fiber preparation and epon embedding: (a) mid to low thigh or brachium level: common peroneal, tibial, ulnar, radial, musculocutaneous nerves; (b) metatarsal or metacarpal level: common peroneal, tibial and radial nerves; (c) phrenic nerve (immediately prediaphragmatic); (d) left recurrent laryngeal
nerve at the level of the aortic arch (proximal) and immediately before entering the laryngeal musculature (distal); (e) saphenous nerve on the medial surface of the thigh immediately proximal to the stifle; (f) superficial radial nerve at a level distal to the branching from the radial nerve in the distal brachium; (g) sympathetic trunk, at mid-thoracic level; (h) dorsal vagal trunk at prediaphragmatic level.

Morphometric studies were done on common peroneal, ulnar and laryngeal nerves. The images of semi-thin transverse sections of each nerve were projected onto a sensor surface (Ladd Graphic Data, Analyzing System, Ladd Research Industries, Burlington, Vt.) interfaced with a programmable calculator. The mean fiber diameter of 400 to 600 fibers per section was recorded from random sampling and the frequency distribution of the myelinated fiber diameter was tabulated for each nerve. The frequency distribution of common peroneal and ulnar nerve fiber diameters was compared with that of five and four age-matched controls, respectively, using standard sampling sites [7].

Results

Clinical findings

Results of hematology, blood chemical profiles, spinal radiography, myelography, thyroid function testing, and cerebrospinal fluid analysis were normal. Electromyographic studies showed bilateral changes of fibrillation potentials and occasional positive sharp waves in all muscles distal to the elbow and stifle. All proximal limb muscles were normal. Evoked muscle action potentials were absent in distal muscles in all limbs.

Light microscopic findings

Biopsy and necropsy findings were identical and will be described together. Pathologic changes were restricted to skeletal muscles and peripheral nerves. The most severe muscle changes were found in distal forelimb muscles (superficial and deep digital flexors) and hind limb muscles (gastrocnemius—lateral head and cranial tibial). There was marked fiber size variation because of atrophic and hypertrophic fibers in most fascicles. The atrophic fibers were angular and formed small and large groups in a disseminated random distribution (fig. 1a). Both type I and type II fibers were atrophic, especially the latter (fig. 1b), whereas hypertrophic fibers were mainly type I. In some areas there was a relative increase in perimysial and sometimes endomysial connective tissue. Many basophilic fibers containing vesicular nuclei were seen (fig. 2a). Subsarcolemmal nuclei were prominent. Internal nuclei often were seen together with many pyknotic clumps. Atrophic fibers usually stained intensely with NADH-TR and many fibers had a central area of increased staining intensity with this oxidative stain (fig. 2b). Myelinated fibers were conspicuously absent in the intramuscular nerves (fig. 3) compared to those in proximal muscles. Muscle spindles were normal. Proximal limb muscles (biceps femoris, vastus lateralis, medial and long head of the triceps brachii and deltoideus) were normal except for occasional focal, small fiber group atrophy.

Peripheral nerve lesions were found in every nerve examined except the phrenic nerve. The changes were most pronounced in the distal parts of the nerves and were
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Fig. 1: a. Cranial tibial muscle. Fiber size variation associated with large group atrophy. HE. b. Atrophic fibers, mainly type II. Routine ATPase, pH 10.00.

categorized by a multifocal or diffuse depletion of myelinated nerve fibers throughout most fascicles (fig. 4a,b). Large diameter fibers appeared to be affected especially. Endoneurial connective tissue was increased in these areas, as was Schwann cell proliferation. Similar but less severe changes were seen in sensory (superficial radial, saphenous) and autonomic (sympathetic and dorsal vagal trunk) nerves. There was no sign of axonal cluster formation, and no abnormalities were seen in any spinal segmental nerve roots, ascending/descending long fiber tracts or neuronal cell bodies within the spinal cord or brain stem.

Electron microscopic findings

Electron microscopy supported light microscopic findings of myelinated fiber depletion. There was obvious endoneurial fibrosis, evidenced by increased numbers of endoneurial fibroblasts, endoneurial processes alternating with layers of collagen, and many collagen bundles (fig. 5). There were increased numbers of Schwann cells, many with reactive changes characterized by collagen pockets invaginated by...
Schwann cell processes [2, 20] (fig. 6). Many Schwann cells were unassociated with axons and were grouped together, forming cell bands of Büngner, some of which were associated with redundant loops of basement membrane (fig. 7). Organelles within axoplasm and Schwann cell cytoplasm looked normal. Degenerative changes were minimal although dense membranous bodies sometimes were seen in Schwann cell cytoplasm. No onion bulb formation or thinly myelinated axons were seen.

**Single fiber teasing**

The most conspicuous change was diffuse nerve fiber degeneration, characterized by multisegmental formation of linear myelin ovoids and balls (fig. 8). Nodal varicosities and paranodal demyelination were seen occasionally. No intercalated nodes were evident.

**Morphometric findings**

There was a reduction of large diameter (9 to 15 μm) myelinated fibers in common peroneal and ulnar nerves (sampled at mid-stifle and mid-elbow sites, respectively)
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Fig. 3: Cranial tibial muscle. Absence of myelinated fibers in intramuscular nerves. Gomori’s trichrome.

compared to control values (Table I, II; fig. 9, 10). In contrast, fewer fibers 5 to 8 μm in diameter were present in the distal recurrent laryngeal nerve compared to more proximal segments (Table III; fig. 11). A relative increase in the number of small diameter fibers was found in the common peroneal, ulnar and distal recurrent laryngeal nerves.

Discussion

The clinical signs of distal symmetrical muscular atrophy involving all limbs makes this disease entity distinct from previously reported canine polyneuropathies. It differs from polyradiculoneuritis [9, 10] by its progressive course and absence of lower motor neuron signs; from hereditary progressive neurogenic muscular atrophy [26] by the distal limb distribution of muscle atrophy; and from giant axonal neuropathy [13] by preservation of both tendon reflexes (patellar, bicipital and tricipital) and cutaneous sensation, and by the forelimb involvement. With the exception of the distal bilateral muscle atrophy, the clinical signs and slowly progressive course of this disorder, as well as the age and breed of the dog, are similar to those seen with cervical vertebral instability [24].

The electromyographic findings of fibrillation potentials and positive sharp waves and absence of evoked muscle action potentials in distal limb muscles were indicative of a diffuse denervation atrophy [23].
Fig. 4: a. Tibial nerve, distal part. b. Recurrent laryngeal nerve, distal part. Diffuse depletion of myelinated nerve fibers. Para-phenylene diamine.

Fig. 5: Common peroneal nerve. Normal, large diameter myelinated fiber surrounded by endoneurial processes (arrows). Many collagen bundles (C).

Fig. 6: Collagen bundles (arrows), invaginated by Schwann cell processes.
The myopathic changes of type I and II atrophic angular fibers distributed in small and large groups, intense oxidative staining of atrophic fibers, and presence of a central target or core of increased staining intensity with NADH-TR in many fibers are all commensurate with denervation [1, 11, 21]. The more apparent type II fiber atrophy has been reported in denervated muscles from several species [18, 22, 28]. The predominant type I fiber hypertrophy may reflect the chronicity of the neuropathy [11], and probably is caused by these fibers compensating for the decreased function of the atrophic fibers [1].

The most conspicuous finding in all peripheral nerves examined, with the exception of the phrenic nerve, was the varied degree of myelinated fiber depletion. This was most severe in the distal parts of the appendicular nerves and in the distal laryngeal nerve. Similar but less severe depletion was seen in the sensory and autonomic nerves. Teased nerve studies indicated that many fibers were undergoing Wallerian
Table I. Myelinated nerve fiber percentage and mean fiber diameter in ulnar nerve (elbow level) from affected dog and controls

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<td>0</td>
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¹ Figures in parentheses refer to total number of myelinated fibers measured.

Table II. Myelinated nerve fiber percentage and mean fiber diameter in common peroneal nerve (stifle level) from affected dog and controls

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<tr>
<th>Fiber Diameter (μm)</th>
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<td>4.81 ± 2.66 (616)³</td>
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¹ Figures in parentheses refer to total number of myelinated fibers measured.

Table III. Myelinated nerve fiber percentage and mean fiber diameter in proximal and distal parts of the left recurrent laryngeal nerve

<table>
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<tr>
<th>Fiber Diameter (μm)</th>
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<td>0.2</td>
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<td>Distal</td>
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<td>2.9</td>
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<td>0</td>
<td>3.81 ± 2.17 (453)³</td>
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</tbody>
</table>

¹ Figures in parentheses refer to total number of myelinated fibers measured.
type degeneration characterized by multisegmental linear rows of myelin ovoids and balls. While the mechanism of the paranodal segmental demyelination we saw is poorly understood, it has been suggested that it probably is secondary to axonal degeneration [15]. The teased fiber preparations showed degenerative changes in nerve fibers of all sizes but especially in the large diameter fibers. Ultrastructural examination of peripheral nerve showed Schwann cell proliferation, denervated Schwann cells, and endoneurial fibrosis. Axoplasmic and Schwann cell cytoplasmic organelles looked normal.

Based on the degenerative changes in motor and sensory nerves, the condition in our dog can be designated pathologically as a distal symmetrical sensorimotor polyneuropathy; clinical recognition of deficits in sensory modalities other than pain, however, probably is objectively impossible in the dog [31]. There were no clinical signs attributable to autonomic nerve lesions. In retrospect, the severe fiber depletion in the distal recurrent laryngeal nerve may have accounted for the dog’s not barking during the period of hospitalization. There was no history of dysphonia.
Fig. 11: Left recurrent laryngeal nerve. Apparent depletion of medium sized fibers in distal part compared to those in proximal nerve.

The cause of the disease was not determined. There was no evidence of nutritional disorders, uremia, or diabetes, which have been associated with distal symmetrical polyneuropathy in man [5]. An organophosphate compound was included in the plant spray mixture used by the owner; however, the small quantity used, absence of signs of acetylcholine toxicity [29], and clinical normalcy of other housed animals make toxic neuropathy a remote possibility.

The distal pattern of axonal degeneration in our dog, with normal spinal roots and neuronal cell bodies throughout the spinal cord, is suggestive of a dying-back neuropathy. This is a disease characterized by axonal degeneration that selectively involves the distal parts of long and large diameter fibers with a slow proximal spread of nerve fiber breakdown with time [8]. The proximal parts of the axons and their perikarya remain intact [5, 16]. Theoretically, a similar pathologic picture may occur after loss of perikarya and axons of the longest fibers [5]. Quantitative evaluation of ventral horn cells and rootlets was not done in our dog. The dying-back pattern of axonal degeneration usually is expressed clinically as a distal symmetrical polyneu-
ropathy. Such a clinical distribution has been reported to occur in a giant axonal neuropathy in the dog [13], a condition characterized by presence of large neurofilamentous axonal swellings in both the peripheral and central nervous systems. Dying-back neuropathy has been studied extensively in experimental animals, in which the disease can be produced by a variety of toxic chemicals including organophosphate compounds and neurotoxic hexacarbons [3, 4, 8, 34, 35]. From these studies, a number of pathogenetic mechanisms have been suggested to explain the dying-back process. These include primary toxicity of the nerve cell body [32], Schwann cell abnormalities [27], primary axonal changes [33], and disrupted axoplasmic flow [30].

In addition to appendicular nerve lesions, recurrent laryngeal nerve degeneration has been reported in experimental [8, 25] and spontaneous [12, 13] dying-back diseases in animals. The depletion of large diameter myelinated fibers in common peroneal and ulnar nerves from our dog, as shown by morphometric studies, is consistent with the dying-back concept [8] and also with the suggestion that nerve fiber diameter is more important than axon length in determining differential vulnerability of peripheral nerve fiber degeneration in experimental dying-back diseases [35]. In the distal part of the recurrent laryngeal nerve, however, medium size fibers appeared to be affected selectively. This canine disease differs from the classical dying-back disease [8, 34] by absence of axonal degeneration in selected pathways of the central nervous system (spinocerebellar tracts and dorsal columns in upper cervical cord segments; rubrospinal and corticospinal tracts in lumbosacral cord segments). Dying-back disease restricted to the peripheral nervous system has been described, however, in equine laryngeal hemiplegia [12] and triorthocresyl phosphate toxicity [14].

Although seven littermates of our dog (five female, two male) were reported to be clinically normal, genetic factors cannot be discounted. There was a considerable degree of inbreeding in the lineage of this dog. Furthermore, this was the first breeding of the bitch; the sire had been used for stud on previous occasions. A repeat test-mating may determine whether or not the condition is inherited.

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