COMMENTARY

Acquired Equine Motor Neuron Disease


Equine motor neuron disease (EMND) is an acquired neuromuscular disease of horses that was first recognized in 1985 and reported in a preliminary report of ten horses in 1990. The disease is characterized pathologically by degeneration of motor neurons in the ventral horns of the spinal cord and in selected brain stem nuclei. Death of motor neurons results in secondary axonal degeneration and denervation atrophy of skeletal muscle. Clinical signs of diffuse neuromuscular disease are seen in EMND and include marked weight loss due to muscle atrophy despite a normal appetite, generalized weakness with a short-strided gait, a characteristic stance with low head carriage and feet positioned well under the body, frequent shifting of weight from limb to limb, excessive recumbency, excessive sweating, especially following exercise, and muscle fasciculations. Involvement of neurons in the spinal ganglia is thought to be the basis of the hyperesthesia noted in some cases. Clinicopathologic features include mild to moderately increased serum levels of creatine kinase and/or aspartate aminotransferase and frequent increase in cerebrospinal fluid protein concentrations. Denervation potentials, primarily positive sharp waves, can be detected with concentric needle electromyography. The clinical and pathologic features of the equine disorder closely resemble those of the motor neuron diseases of human beings, of which the most important is amyotrophic lateral sclerosis (ALS; “Lou Gehrig’s disease”). This name is applied to a group of fatal, degenerative motor neuron disorders that are characterized by progressive degeneration of spinal and selected brain stem motor neurons, with only mild involvement of sensory neurons. The pathology of the human disorder differs from that of EMND, however, in that extensive degeneration of pyramidal tracts is seen in human beings with classical ALS. Only mild degeneration of pyramidal tracts is present in horses with EMND. However, the pyramidal tracts are poorly developed, and less extensive, in ungulates as compared to human beings. Alternatively, EMND may be more analogous to progressive muscular atrophy, a variant of ALS in which pyramidal tracts are spared. Histopathologic and ultrastructural lesions in neurons of horses with EMND closely resemble those seen in ALS. Immunocytochemical studies have shown that the swollen neurons in EMND contain dense aggregates of neurofilaments, a finding similar to those in ALS.

This equine disorder is distinctly different, clinically and pathologically, from previously described diseases of the equine neuraxis. Equine degenerative myelonecephalopathy and cervical stenotic myelopathy are diseases of spinal cord white matter and result in clinical evidence of spastic paresis and ataxia. Protozoal encephalomyelitis may result in focal or multifocal degeneration of motor neurons and degeneration of the white matter of the brain and spinal cord, but the resulting lower motor neuron weakness is localized and frequently asymmetric. Rabies virus polioencephalomyelitis may initially produce signs suggestive of lower motor neuron disease. However, the rapid, diffuse spread of this lesion will be accompanied by other neurologic signs. The motor neuron degeneration seen in EMND is pathologically similar to that seen in a presumed inherited motor neuron disease of zebras (Equus burchelli), but EMND is clearly a sporadic acquired disorder affecting horses of all ages and breeds. Quarter Horses are overrepresented in the various breeds affected, and Thoroughbreds are also at increased risk. The risk of EMND increases with age, with a peak at 16 years of age.

At the time of this writing, 67 cases of EMND have been confirmed by pathologic and/or clinical and clinicopathologic findings, and many more are suspected. Most of the 67 recognized cases of EMND have occurred in the northeastern USA, the area from which most cases seen at the New York State College of Veterinary Medicine are drawn. However, cases have been confirmed in Ohio (four), Indiana (two), West Virginia (one), Maryland (one), Kentucky (one), Alaska (two), California (one), Oregon (one), Nebraska (one), Tennessee (one), Florida (one), and Ontario, Canada (one). Confirmation has relied on recognition of typical clinical and clinicopathologic findings and on frozen section histochemistry applied to muscle samples mailed by overnight carrier to appropriate laboratories. Additional cases are suspected in Great Britain. At Cor-
Table 1. Number of horses with lesions in selected skeletal muscles out of a total of 23 horses with EMND.

<table>
<thead>
<tr>
<th>Lesions</th>
<th>Lateral Triceps (n = 21)</th>
<th>Extensor Carpi Radialis (n = 19)</th>
<th>Cranial Tibial (n = 17)</th>
<th>Intermediate Vastus Lateral (n = 20)</th>
<th>Medial (n = 11)</th>
<th>Biceps Femoris (n = 22)</th>
<th>Medial Triceps (n = 8)</th>
<th>Lateral Vastus (n = 6)</th>
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<td>Excessive fiber size variation</td>
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<td>Scattered angular atrophied fibers</td>
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Cases of EMND probably will continue to occur, and veterinary pathologists in many parts of the world will find themselves confronted with biopsy or necropsy material from a horse suspected of having EMND. This report reviews the diagnostic light microscopic lesions in the nervous system of 23 horses, including studies of autonomic nerves and ganglia, and details the diagnostic gross and light microscopic lesions in skeletal muscle (Table 1). These cases were recognized over a 7-year period beginning in 1985. Age of affected horses ranged from 15 months to 15 years. Breeds involved were Quarter Horse (nine), Thoroughbred (ten), Standardbred (two), Morgan (one), and grade (one). There were 11 castrated males, three intact males, and nine intact females. Duration of clinical disease ranged from 2 weeks to 2 years. All 23 were from the northeastern USA.

The brain, spinal cord, and representative samples of peripheral nerves, including nerve roots, were removed and fixed in 10% buffered neutral formalin. Samples of the brain, spinal cord, and peripheral nerves, including nerve roots, were taken at multiple levels in all cases except for one horse in which histologic sections of the brain were not examined. Spinal cord segments examined included cranial cervical, cervical intumescence, thoracic, cranial lumbar, lumbosacral intumescence, and sacral. The number of levels sampled ranged from three to 17, with a mean of eight levels sampled per horse. Spinal roots were selected from the cervical region, thoracic region, lumbosacral region, and cauda equina. In addition, spinal root sections from various levels accompanied sections of spinal cord. Peripheral nerve samples were examined from all horses. Nerves examined were cervical, supraspinal, subcapsular, recurrent laryngeal, phrenic, axillary, musculocutaneous, radial, median, ulnar, obturator, femoral, sciatric, peroneal, and tibial. Samples of spinal ganglia were also examined and included ganglia from the cranial cervical spinal cord, cervical intumescence, thoracic spinal cord, lumbosacral intumescence, and caudal spinal cord. Cranial nerves and ganglia were examined and included the trigeminal nerve and ganglion, glossopharyngeal, oculomotor, abducent, facial, and spinal accessory. Autonomic nerves (major splanchnic, sympathetic trunk) and sympathetic ganglia were also examined. Formalin-fixed samples were embedded in paraffin and processed routinely for light microscopy. Tissue sections were stained with hematoxylin and eosin (HE), Luxol fast blue-cresyl violet, Holmes' silver, and Masson's trichrome.

Unfixed, unclamped samples of skeletal muscle from multiple sites of all horses were collected and were snap-frozen in isopentane cooled in liquid nitrogen. Muscles sampled varied from horse to horse but included an epaxial muscle, diaphragm, supraspinatus, deltoideus, lateral head and medial head of the triceps brachii, extensor carpi radialis, gluteus, biceps femoris, lateral vastus, rectus femoris, medial and lateral portion of the intermediate vastus, semitendinosus, gastrocnemius, and cranial tibial muscles. Most samples were collected at postmortem examination, with a small number collected as biopsy samples prior to eutha-
Transverse sections of these samples were cut at 8-10 μm on a Reichert cryostat at −20 C and serial sections were stained with HE, modified Gomori’s trichrome, reduced nicotinamide adenine dinucleotide tetrazolium, periodic acid-Schiff, and myofibrillar ATPase at pH 10.0 and 4.35. Selected cases were stained with alizarin red S.

Gross necropsy findings

All horses had moderate to severe diffuse muscle atrophy of cervical, trunk, and limb musculature. Severe atrophy of internal and subcutaneous fat was noted in two horses. Fat stores were normal or slightly decreased in 21 horses. One horse had bilateral shortened flexor tendons in the pelvic limbs. Grossly apparent discoloration and softening were noted in muscles of 20 horses. The color of these muscles ranged from pale red to yellow-red. The most consistent gross muscle lesions were found in the medial and lateral portions of the intermediate vastus muscles as they arise from the shaft of the femur and in the medial head of the triceps brachii, lying under the lateral head of the triceps and arising from the caudomedial surface of the humerus. These muscles were pale, soft, and almost gelatinous and were often yellow-red. These muscles contrasted with the adjacent, more normal appearing muscle. In those cases in which both medial and lateral portions of the intermediate vastus muscles were examined, the medial portion was more severely affected. In chronic cases, i.e., horses with clinical signs for 1.5 to 2 years, gross lesions were less obvious in the medial head of the triceps brachii and in the lateral portion of the intermediate vastus but were still obvious in the medial portion of the intermediate vastus. Peripheral nerves, spinal roots, brain, and spinal cord were grossly normal in all 23 horses.

Histopathologic findings

Brain. Moderate neuronal degeneration was consistently present in the facial nucleus and the motor nucleus of the trigeminal nerve. Neuronal degeneration was also common in the hypoglossal nuclei and in the nucleus ambiguus. Rarely, neuronal cell body degeneration was seen in the vestibular nuclei, reticular formation, and mesencephalic nucleus of the trigeminal nerve. In hematoxylin and eosin (HE)-stained sections, affected neurons were most frequently swollen, pale, and eosinophilic, with dispersed chromat. Nuclei, when visible, were central or slightly eccentric, and some contained clumped heterochromatin or were undergoing karyorrhexis. There were single or multiple brightly eosinophilic inclusions ranging from 1 to 8 μm in size within the cytoplasm of some neurons. Occasional neurons contained cytoplasmic vacuoles. With Luxol fast blue-cresyl violet stain, degenerating neurons were pale because of depletion of Nissl substance (“ghost cells”). Lipofuscin content varied, but all horses had neurons with visible cytoplasmic lipofuscin. With Holmes’ silver stain, swollen chromatolytic neurons stained more densely than unaffected neurons. Occasional spheroids, glial scars, and shrunken neu-
neurons undergoing neuronophagia were seen. Glial scars characteristically contained one or more lipofuscin-laden macrophages and glial cells surrounding a pale core of astrocyte processes. In chronic cases, only glial scars were present. Neuronal degeneration was correlated with axonal degeneration in the intramedullary projections of the associated cranial nerves. Axonal degeneration was also evident in a few fibers in the spinal tract of the trigeminal nerve. Mild degeneration of pyramidal tracts of the medulla was present in a few horses.

**Spinal cord.** Neuronal degeneration similar to that seen in brain stem nuclei was present in the ventral horn neurons at all levels of the spinal cord (Fig. 1). Neuronal lesions were most easily detected in longitudinal sections through the ventral horns. The ease with which neuronal degenerative changes could be detected varied among horses. In general, active neuronal degeneration was more readily detected in horses in which clinical signs had been present for 2 to 3 months or less. After several months of clinical disease, degenerate neurons were less frequent, although the ventral horns often appeared depleted of neurons, and characteristic glial scars were more apparent (Fig. 2). Small numbers of spheroids were also present within the ventral horns. Examination of intramedullary tracts of the ventral roots revealed frequent axonal degeneration (Fig. 3). This change was considered a useful marker of motor neuron death, particularly in those cases in which overt neuronal degeneration was less obvious. Lesions were not detected in the dorsal horns or intramedullary fibers of the dorsal roots.

A few degenerate fibers were present in white matter tracts at multiple levels in most horses. White matter lesions were more severe in a 15-year-old mare that had clinical evidence of ataxia as well as weakness and muscle atrophy.

**Nerve roots and spinal ganglia.** Moderate to severe degeneration was present in the ventral spinal roots at all levels examined. Lesions varied from active fiber degeneration, characterized by axonal fragmentation, myelin ellipsoids, and the formation of chains of "digestion chambers" containing macrophages and cell debris, to severe depletion, characterized by loss of myelinated fibers with replacement by columns of proliferated Schwann cells (Büngner’s bands) and increased endoneurial collagen. Intradural ventral roots only showed axonal degeneration. Only mild axonal degeneration was seen in dorsal roots. Occasional swollen, degenerating neurons were present in spinal and trigeminal ganglia. In some of these ganglia, Nageotte bodies were evident where satellite cell proliferation replaced lost cell bodies.

**Spinal and cranial nerves and cranial nerve ganglia.** Mild to severe, active to chronic axonal degeneration was present in all spinal nerve branches that were studied (Fig. 4). Lesions were most readily detected in longitudinal sections of nerves and were present at all levels (proximal and distal). Lesions often appeared fascicular in nature, with severely affected fascicles adjacent to mildly affected or apparently unaffected fas-
Active lesions were characterized by loss of axons and formation of myelin ellipsoids and macrophage-containing “digestion chambers,” either singly or in chains along the length of the fibers. Chronic lesions were characterized by less obvious active fiber degeneration, with loss of myelinated fibers, increased endoneurial collagen, and proliferated Schwann cells (Büngner’s bands), resulting in an overall pale appearance of chronically affected fascicles when stained with HE. Luxol fast blue and Masson’s trichrome stains highlighted the loss of myelinated fibers and scarring present in chronically affected nerves.

Degenerative lesions were consistently present in the spinal accessory, trigeminal motor, and facial nerves, and in some cases in the hypoglossal nerves. Oculomotor and abducent nerves were generally spared. Occasional degenerating neurons and/or glial scars were seen in the trigeminal ganglion.

Sympathetic nerves and ganglia. The majority of horses studied did not have involvement of the sympathetic nervous system. Rarely, very mild lesions were detected in sympathetic nerves and/or sympathetic ganglia.

Skeletal muscle. A summary of the lesions present within selected skeletal muscles is presented in Table 1. The severity of lesions varied from muscle to muscle, but similar lesions were present in all cases. The majority of muscles contained angular atrophied fibers that occurred either as scattered individual fibers or as small groups of fibers (small group atrophy; Fig. 5).

When angular atrophy occurred as scattered individual fibers, the atrophy involved either type 1 and type 2 fibers (Fig. 6) or predominantly type 1 fibers (Fig. 7).

In muscles with small group atrophy, both type 1 and

![Fig. 5. Light micrograph, frozen section. Intermediate vastus muscle, lateral portion; 11-year-old Thoroughbred stallion with signs of EMND for 1.5 years. Note small and large group atrophy with adjacent fiber hypertrophy. Modified Gomori’s trichrome. Bar = 50 μm.](image)

![Fig. 6. Light micrograph, frozen section. Intermediate vastus muscle, lateral portion; 12-year-old Quarter Horse gelding with signs of EMND for 2 months. Note angular atrophy of type 1 (lightly stained) and type 2 (darkly stained) fibers. This muscle is composed primarily of type 1 fibers. Myofibrillar ATPase, pH 10.0. Bar = 50 μm.](image)

![Fig. 7. Light micrograph, frozen section. Biceps femoris muscle; 9-year-old Quarter Horse mare with signs of EMND for 1 month. Note angular atrophy of predominantly type 1 (lightly stained) fibers. Myofibrillar ATPase, pH 10.0. Bar = 25 μm.](image)
type 2 fibers were atrophic, either in relatively equal numbers or with a predominance of type 1 atrophy. In muscles with severe fiber atrophy, adjacent fibers were frequently hypertrophied. Severe atrophy of entire fascicles, in which all fibers were small and rounded (large group atrophy), occurred in the medial head of the triceps brachii, the medial portion of the vastus intermedius, and occasionally the lateral portion of the intermediate vastus (Figs. 5, 8). Small or large group atrophy was consistently present in those muscles in which discoloration and softening was noted at necropsy. These severely atrophied muscles were composed primarily of type 1 fibers, but both type 1 and type 2 fibers were atrophied. These muscles also frequently contained increased amounts of perimysial and endomysial fat. Endomysial fibrosis of these severely affected muscles was generally mild, involving only small scattered areas of the muscle. Fibrosis was more severe and more diffuse in chronic cases. The diaphragm, although it contains a high proportion of type 1 fibers, was spared. Muscles containing numerous angular atrophied fibers, or with large group atrophy, frequently demonstrated irregular staining of fibers with reduced nicotinamide adenine dinucleotide tetrazolium (NADH) stain. Dense peripheral rims, subsarcolemmal masses, central clearing, and fibers with multiple cytoplasmic clear areas ("mini-cores" and "motheaten" fibers) were visible with NADH stain. Atrophied fibers were frequently very pale in periodic acid–Schiff-stained sections. Excessive fiber size variation was a consistent finding in all muscles examined (Fig. 9). An increased number of fibers with internal nuclei was present in many muscles, often those with minimal fiber atrophy (Fig. 9). Internal nuclei were more common in horses with chronic disease. Ring fibers, fibers with a distinct peripheral ring of maloriented myofibrils, were numerous in the extensor carpi radialis muscle of one horse, and a single ring fiber was seen in the same muscle of a second horse. Scattered muscle fiber necrosis and regeneration were common findings and most frequently involved individual fibers. Necrotic fibers had pale cytoplasm with pyknotic nuclei, frequent macrophage infiltration, and loss of cytoplasmic detail. In some cases, only a small cluster of macrophages remained (Fig. 10). In cases with scattered fiber necrosis, alizarin red S staining revealed only rare calcium-positive fibers. Regenerating fibers had increased cytoplasmic basophilia in HE-stained sections and large, euchromatic nuclei. Massive, acute fiber necrosis was present in a severely affected horse that was recumbent and died spontaneously. Alizarin red S staining for calcium revealed numerous intensely
positive degenerating fibers in this horse. There was no evidence of reinnervation (fiber type grouping) in these 23 horses.

Intramuscular nerves. Many sections of skeletal muscle contained transverse or oblique sections of intramuscular nerves. The density of myelinated fibers, cellularity, and amount of endoneurial collagen were evaluated in HE- and modified Gomori's trichrome-stained sections. Some nerve branches were apparently normal, but many others had moderate to severe loss of myelinated fibers, with increased cellularity and increased endoneurial collagen. Occasional oblique sections contained focal areas of active axonal degeneration.

Muscle fiber atrophy of both type 1 and type 2 fibers is indicative of denervation atrophy. Small and large group atrophy of both type 1 and type 2 fibers, as seen in severely affected muscles in equine motor neuron disease (EMND), is pathognomonic of denervation atrophy. The finding of predominantly type 1 fiber atrophy as a manifestation of denervation atrophy in horses with EMND is unusual and is not a feature of denervating diseases in other species, nor has it been reported in amyotrophic lateral sclerosis (ALS). No correlation could be made between severely affected muscles and lesions in the peripheral nerves, ventral spinal roots, or motor neurons of the ventral horns.

Other skeletal muscle changes seen, such as excessive fiber size variation, internal nuclei, and cytoarchitectural alterations resulting in irregular staining, particularly with reduced nicotinamide adenine dinucleotide tetrazolium stain, are considered nonspecific myopathic changes. Scattered fiber degeneration appears to be a consistent feature of EMND, as judged by the increased serum levels of creatine kinase and/or aspartate aminotransferase found on clinicopathologic evaluation and by the frequent necrotic fibers seen on histopathologic sections. Although fiber degeneration can be seen in denervating diseases of human beings, it is not a consistent feature. If evidence of denervation atrophy is subtle, the presence of fiber degeneration in equine muscle samples could lead to a misdiagnosis of degenerative myopathy. Massive acute fiber degeneration, as seen in one horse in this study, is not a feature of denervating diseases in other species. In this horse, it most likely reflects ischemic damage secondary to prolonged recumbency due to severe weakness. The cause of the scattered necrotic fibers seen in less severely affected horses is not clear but could reflect either increased workload on remaining innervated fibers or very mild ischemic damage due to increased recumbency.

The purpose of this report is to document the range of diagnostic gross pathologic and light microscopic lesions of EMND in the nervous system and skeletal muscle. Examination of samples of brain stem and spinal cord, particularly cervical and lumbar intumescence, revealed the characteristic neuronal degeneration and/or neuronal loss that is the hallmark of EMND. Secondary axonal degeneration leading to a loss of myelinated fibers was present in the ventral spinal roots, spinal nerves, and certain cranial nerves (spinal accessory, trigeminal, facial, and hypoglossal). Neuronal degeneration could be detected at all levels of the spinal cord but was most obvious in the cervical and lumbar intumescence. Axonal degeneration within ventral spinal roots and peripheral motor nerves was widespread. If EMND is suspected, multiple sections should be prepared from samples taken from the spinal cord at the cervical and lumbosacral intumescences and from the brain stem at the levels of the cranial and caudal cerebellar peduncles and at the obex. Longitudinal sections through the ventral horns of the spinal cord are recommended, because large numbers of motor neurons are available for evaluation in these preparations. Brain stem sections should include the trigeminal, facial, and hypoglossal nerves. The results of this study suggest that careful examination of multiple sections will reveal degenerating neurons. In chronic cases, neuronal loss with resultant glial scarring may be the predominant lesion in the central nervous system. Luxol fast blue-cresyl violet stain may highlight degenerating neurons and glial scars. In the absence of overt neuronal degeneration, the presence of axonal degeneration and/or loss of myelinated fibers in the intramedullary and extramedullary spinal roots and in the spinal nerves and specific cranial nerves should alert the pa-
thologist to the possibility of EMND. Multiple samples of ventral spinal roots and peripheral motor nerves should be examined. Masson’s trichrome and Luxol fast blue-cresyl violet may be useful because they will highlight the presence of axonal degeneration and/or loss of myelinated fibers.

Careful examination of multiple muscles, particularly the deep proximal limb muscles, often revealed gross evidence of denervation atrophy. In these severely affected muscles, the massive large and small group fiber atrophy, fiber hypertrophy, and frequent fibrosis and fat infiltration would be obvious even on routinely processed formalin-fixed muscle. Masson’s trichrome stain is recommended to assess individual fiber diameters and presence of collagen and to evaluate intramuscular nerves. Lesions in the more superficial muscles, those that are accessible for biopsies, are often subtle. Detection of the increased fiber size variation and scattered angular atrophy and/or degenerate fibers requires special handling. Frozen section histochemical examination is ideal but is not always available. Properly handled unfixed muscle samples mailed by overnight courier can be processed for frozen section histochemistry, although periodic acid-Schiff staining and ATPase staining at acid preincubation may be lost due to fiber autolysis. For routine preparation, muscle samples should be clamped prior to formalin fixation. Transverse sections are more useful than longitudinal sections for evaluation of fiber diameter and to detect angular atrophied fibers.

The etiology of EMND is as yet unknown. The predilection for highly oxidative (type 1) skeletal muscle atrophy suggests the possibility that highly oxidative motor neurons may be selectively damaged. A deficiency of antioxidant activity in the central nervous system could result in oxidative injury to neurons. Preliminary studies of affected horses indicate that although whole blood selenium levels are within normal limits serum vitamin E is consistently low. Recent findings from studies of the familial form of ALS in human beings have shown that the genetic defect resulting in this form involves the gene coding for superoxide dismutase, an enzyme that is involved in free radical scavenging and therefore in prevention of oxidative tissue injury. Oxidative injury may be involved in various forms of motor neuron disease. Etiologic factors that predispose horses to EMND, including vitamin E deficiency, may be more common in the northeastern USA. However, as awareness of the disease and of the diagnostic lesions increases, additional cases may continue to be recognized in other geographic areas. Ongoing studies of this debilitating equine disease could benefit both the equine industry and the medical community.

References


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