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Suspected LMND in a Siberian Husky

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A four-year old, male Siberian husky was presented with an ambulatory tetraparesis of seven days duration. The neurological exam showed an ambulatory tetraparesis with normal proprioception and reduced muscle tone in the limbs and neck. Spinal reflexes were diminished and the cranial nerves were normal. The motor function of the tail was absent and the animal was not incontinent. The dog was alert. There was no spinal pain and severe paraspinal muscular atrophy was evident.

The work-up consisted on a complete blood and urine analysis, thoracic x-rays, abdominal ultrasound, titration against Neospora, antibodies agaisnt Achr, serum colinesterase concentration and electrodiagnostic studies (Key-Point; Medtronic). All this work-up was normal except the electromyograhy; fibrillation potentials and positive sharp waves were seen in every evaluated muscle. A neurography of the motor nerves was performed on the right ulnaris nerve and the left tibialis nerve. Both of them showed a marked reduction of the amplitudes (1,6-1,5-1,4 mV) and conduction velocities of 44.7 m/s in the ulnar nerve and 31.4 m/s (distal) and 43.5 m/s (proximal) in the tibialis nerve. Neither nerve examined showed an F-wave. The repetitive nerve stimulation at the distal tibial nerve of the left rear limb showed reduced amplitudes.

This animal eventually returned after 10 days with a non-ambulatory tetraparesis and a mixed dyspnoea, and died. The ventral horns of the spinal cord showed a marked decrease in the number of neuronal cell bodies and some of them showing degenerative changes (ballooning, pallor). The ventral nerve roots showed a marked decrease of the number of nerve fibres and the peripheral nerves showed besides Wallerian degeneration and Schwann cell proliferation. Marked accumulation of an orange and granular material in the cytoplasm of some neurons in the ventral horns of the spinal cord and the medulla oblongata, affecting vestibular nuclei, nucleus ambiguous and the trigeminal whit a bilateral and symmetrical distribution. The whole brainstem presented vasogenic oedema.

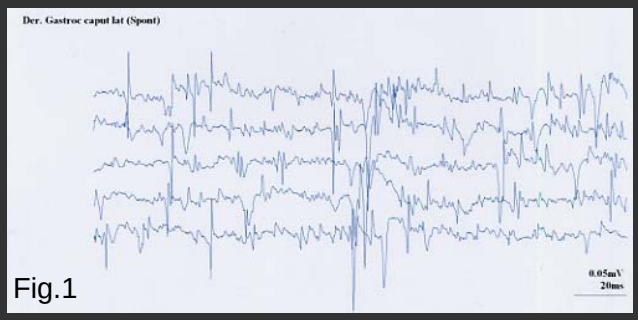

Fig.1. Spontaneous activity (fibs and PSW) in the right gastrocnemius muscle.

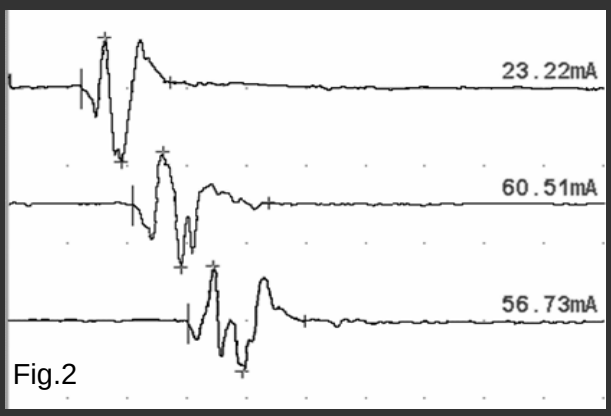

Fig.2. MNCV of the left tibialis nerve showing poliphasic waves and temporal dispersion indicating demyelination. The amplitudes were very low and it is indicative of axonal damage.

This case could be an atypical presentation of degeneration of the lower motor neurons. This type of disease usually has the onset at an earlier age and in particular breeds. This autor has not found evidence of this disease in huskies in the bibliography. In our case familial history is lacking. The cause could be the result of environmental agents rather than genetic disorders. In that case we have a disease affecting the GSE system, more accentuated at the intumescences. The Wallerian degeneration could have had its origin at the nerve roots, peripheral nerves or the LMN´s. A mild inflammatory infiltration was observed at involved motor nerve roots and although this is a point for a polyrradiculoneuritis, loss of motor neurons in the ventral horns of the spinal cord is not usually seen in this disease. In our case we had loss of motor neurons and some of them were degenerated. Altered axonal transport was detected by the abnormal accumulation of lipofucsin in the soma of the neurons. In certain cases is difficult differentiate between lesions in the nerve roots or its neuronal soma. In this case we have a degenerative process rather than a inflammatory one affecting the motor unit with involvement mainly at the LMN´s.

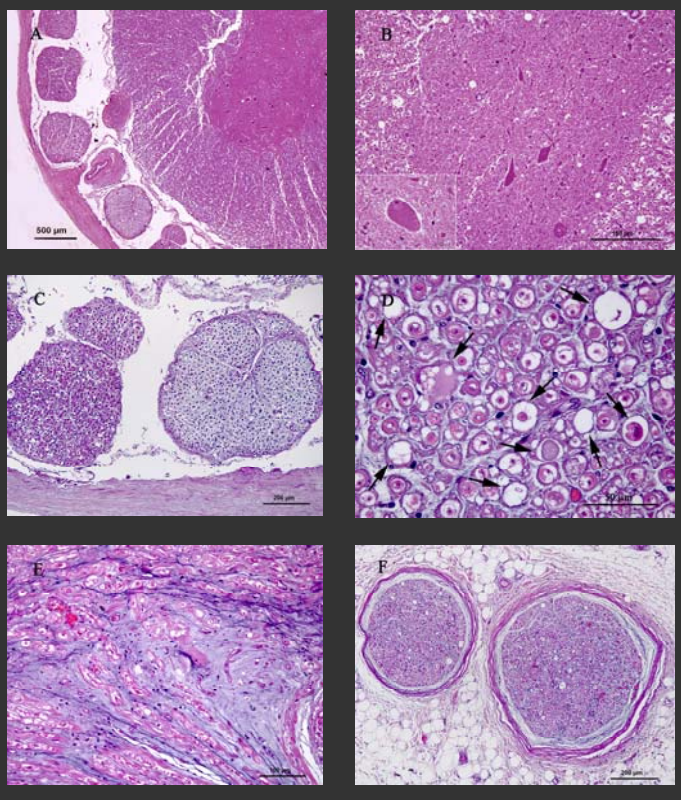

Fig.3. A,B) Transversal sections of C6-T2 spinal cord segments. Decreased number of motor neurons in ventral horns, spheroids and chromatolytic neurons are seen in the grey matter (B). C) Different degree of lesions in transversal section of spinal ventral roots showing deeper pathological changes in the right one. D) Wallerian degeneration in a zoomed transversal section of the spinal ventral root; spheroids and myelin sheaths dilatations are shown (arrows). E,F) Longitudinal and transversal sections of the sciatic nerve fascicles showing an abundant myxoid amorphous material deposition in the endoneurium (E) and increased perineurial thickness (F).