Immune-Mediated Polymyositis in a Dog
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Polymyositis is a noninfectious inflammatory disease of
the striated muscle and is classified as a systemic immune
mediated disease. Human polymyositis is diagnosed
through the following criteria: (1) the clinical signs like
symmetrical proximal muscle weakness, (2) biochemical
data (high concentrations of serum muscle enzymes), (3)
abnormal electromyographic (EMG) findings and (4)
histopathological diagnosis [2-4, 10]. However, there
have been only a few reports on immune-mediated
polymyositis in dogs [1, 6-8]. This paper reports a case of
definite polymyositis of a dog which fulfills these 4 criteria.

A 4-year-old female mongrel dog weighing 7 kg was
presented with histories of exercise intolerance and acute
hindlimb weakness (Fig. 1). Physical examination re-
vealed kyphosis and the generalized muscular atrophy.
The dog walked clumsily with a short stride. The weakness
of the hindlimbs was severer in the morning than in the
afternoon. The rectal temperature was continuously high
(approximately 40°C). There were no specific painful
areas by palpation, however spontaneous pain seemed to
be evident due to occasional painful yelping on postural
changes. The reflexes were normal.

A complete blood count, serum chemical analyses and
urinalyses were performed. Abnormally elevated values
of serum aspartate aminotransferase (121 U/L), lactate
dehydrogenase (691 U/L), creatine kinase (CK, 860 U/L)
and aldolase (15.9 U/L) were obtained. From isoenzyme
analysis of the CK, 86.7% was an MM-segment originated
from muscles. The total serum protein was slightly high
(8.2 g/dl) with high β- and γ-globulin fractions (2.1 g/dl
and 2.3 g/dl, respectively) and a low A/G ratio (0.61). The
result of direct Coombs test was negative, however, lupus
erhymatous (LE) cell preparation was positive and
antinuclear antibody (ANA) titer was high (1:256) with-
out clinical signs specific to systemic lupus erythematosus
(SLE) (Fig. 2).

From the above results, the patient was suspected to
have an immune-mediated myogenic disease, thus further
examinations were conducted. Tension test by an
intravenous injection of 0.5 mg of edrophonium chloride
showed negative, ruling out myasthenia gravis [9]. Needle
electromyography was performed on thoracic and lumbar
paraspinal muscles and on limb muscles. Low amplitudes
of motor unit potentials, fibrillation potentials, and
positive sharp waves were detected in all the investigated
muscles. A triad of EMG findings is usually present with
human polymyositis: (1) fibrillation and positive sharp
waves, (2) pseudomyotonia, and (3) a large number of
small motor unit potentials. Two of this triad were found
in over 90% of the cases of polymyositis [14]. Polyphasic
motor unit potentials and insertional fibrillation potentials
were also detected. Motor and sensory nerve conduction
velocities of the tibial nerve measured between the stifle
and tarsal joints were 60 m/sec and 72 m/sec, respectively
[5]. These values seemed to be within normal range [13,
16], which may rule out the peripheral neuropathy in this
case.

Histopathologic examinations of the muscles biopsied
from the left biceps femoris and the right quadriceps
femoris revealed muscle fiber degeneration, necrosis,
lymphoid and perivascular infiltrations of plasma cells
(Fig. 3). Furthermore, immunoglobulin G (IgG) was
demonstrated on the muscle fibers by direct immuno-
fluorescence, however C3 component was not
detected. IgG against the muscles was not detected in
the serum of the patient by indirect immunofluorescence.
Electron microscopic findings revealed partially disarray
of myofibrillar architecture [15] (Fig. 4).

Radiographic examination on the peripheral joint re-
vealed the small osteophyte formation in the stifle and
tarsal joints, but not destructive joint lesions. A synovial
fluid aspirated from the left stifle joint revealed a mild
increase in total cell count (2700/μl) with a marked
increase in neutrophils (70%). No bacteria was isolated
from the synovial fluid. This finding ruled out the
infectious polyarthritis in this case [11, 12].

From these findings, this case was diagnosed as im-

Fig. 1. The posture of the dog at the admission. The patient drags the hindlimbs like a dog with a spinal cord disorder.
mune-mediated polymyositis. Prednisolone (3 mg/kg, b. i. d., per os) was used as an initial treatment [1]. After a week of treatment, the muscle strength was enhanced and the dog was able to run, although the pyrexia remained. CK level returned to normal (54 U/L) and the β- and γ-globulins also decreased to normal levels (0.6 g/dl and 0.8 g/dl, respectively). The dose of prednisolone was gradually reduced and maintained at a dose of approximately 0.7 mg/kg given on alternate days for 10 months. At present, the dog is healthy without any treatments, though LE cell preparation is still positive and ANA titer is also high. Further observation is necessary whether clinical signs will recur or not.

REFERENCES