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for more than 4 months and died in the last 9 years were used for the pathological study. The main lesions were as follows: skin lesions in 18 dolphins, pneumonia in 15 dolphins, ruminal ulcers in 9 dolphins, and enteritis in 7 dolphins. Other lesions were proliferative cholangiohepatitis in 4 animals, and encephalitis and meningoencephalitis in 2 dolphins. Nine dolphins (36%) died of septicemia. The results proved that septicemia, skin lesions and pneumonia often occur in captive dolphins.

Aspiration pneumonia was found in 8 of 15 dolphins with pneumonia. Bacterial and fungal encephalitis was observed in 2 dolphins.

Aspiration pneumonia was composed of mild focal fibrinous bronchopneumonia to severe diffuse necrotizing pneumonia, and the severity of the lesion varied depending on the case. Three dolphins died of severe aspiration.

Foreign body accumulation in the rumen was observed in 6 of 8 dolphins kept in an open pen. Parakeratosis and ulcers were found in their rumina. The mucosal lesions were severe in the rumen, containing sharp or firm foreign bodies. As severe foreign body accumulation in the upper

alimentary tract caused death by starvation in one dolphin, swallowing of foreign bodies seems to be life-threatening for some dolphins.

Calcium deposition in the bronchiolar mucosa was observed in 24 dolphins. The lesion was observed only in the bronchiolar mucosa, and no significant elevation of the serum calcium concentration was seen in those dolphins. Cellular debris occasionally accumulated on the bronchiolar mucosa and was incorporated in the mucosa. From these findings, it was assumed that the calcium deposition resulted from dystrophic calcification on the exudate, cellular debris and foreign bodies in the bronchiolar mucosa. However, the lesion was considered to be incidental and pathologically insignificant.

Fungal infection were often seen, particularly in mucosal lesions (rumen 7, esophagus 3, laryngopharynx 3, and nasal sinus 2). The fungal infection caused severe, systematic infection such as pneumonia, nephritis and encephalitis in 2 dolphins treated with steroids for a long time. Therefore, the findings suggested that long-term steroid therapy in dolphins can induce severe fungal infection.

Pathological Studies of GM₁ Gangliosidosis in a Shiba Dog

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GM₁ gangliosidosis is a lysosomal storage disease caused by deficient activity of lysosomal acid beta-galactosidase. This genetic disorder is inherited as an autosomal recessive trait.

It has been identified and studied in humans, cats, cattle, dogs, and sheep. In this study, we the described morphological manifestations of GM₁ gangliosidosis in a Shiba dog and compared this case and other cases with GM₁ gangliosi-

dosis.

A 6-month-old Shiba dog showed progressive clinical signs of a predominantly cerebellar ataxia and resting and intentional head tremors. Clinical signs, including ataxic abasia, hypermetria, tetraplegia, corneal clouding and visual deficits were also prominent at 10 months of age. Vacuolated lymphocytes suggesting a storage disorder were seen in the peripheral blood. At

autopsy, the liver was pale and moderate splenomegaly and mild kyphosis were found.

The brain parenchyma was generally yellowish in color and had mildly increased consistency.

Microscopic study revealed distension of neurons throughout the central nervous system. Intracytoplasmic storage vacuoles and granules were also found in visceral and skeletal organs but the submucosal and myenteric plexus, pancreatic islets, adrenal cortex, kidney, thyroid gland, and skeletal muscle were not affected. Neuronal storage material was negative to weakly positive for lipid staining, PAS-positive in frozen sections, positive for anti-asialo GM₁ antibody, and stained with lectins, including Con A, DBA, SBA, and RCA-1. The results of lectin staining were different from those of other two types of canine GM₁ gangliosidosis.

Accumulation of neurofilaments was found in cell bodies of neurons and axons of the cerebral cortical pyramidal cell layer, brain stem, cerebellum and spinal cord. Neurons and axons of the pyramidal and extrapyramidal tracts were positive for anti-Tau antibody. Swollen neuronal and microglial cells were positive for anti-beta-amyloid protein antibody.

The ratio of white matter to gray matter in the cerebellum of this case was reduced. Histologically, loss of oligodendrocytes and irregular thickness of the myelin sheath were observed with proliferation of microglia and many myelin bulb-like structures. Myelin showed metachromasia in toluidine-blue-stained frozen sections, suggesting aberrant myelin development.

There was severe diffuse microgliosis and astrocytosis with mild to moderate proliferation of Alzheimer type II astrocytes throughout the

gray matter of the central nervous system. However, fibrillary gliosis, which is generally observed in the late-stage of brain injury, was not prominent in these regions of this case.

A series of neuron systems consisting of the cerebellum, mesencephalon, pons, medulla oblongata, globus pallidus, and ventral lateral nucleus of the thalamus were selectively affected. These neuronal deficits were consistent with the clinical symptoms of this case.

Ultrastructurally, typical membranous cytoplasmic bodies (MCB) observed in GM₁ gangliosidosis and several variants of morphologically different MCBs were found in neurons. Storage materials in hepatocytes and pancreatic exocrine cells were low-electron-dense vacuoles that contained a different form of MCBs from those of neurons.

Biochemical analysis revealed 1% residual beta-galactosidase activity and excessive deposition of GM₁ ganglioside in the brain compared with normal dogs.

The present case was considered to be an infantile form based on biochemical and pathologic results. The excessive neurofilament accumulation, metabolic deposits, and beta-amyloid protein deposition in neurons appeared to be caused by a disturbance of cytoplasmic transport resulting from cytoplasmic accumulation of storage materials related to the GM₁ gangliosidosis. Astrocytic dysfunction was suspected from the aberrant astrocytic reaction to the neuronal loss and degeneration. These oligodendroglial changes with metachromatic substances may suggest dysmyelination in the white matter of this dog.