Polioencephalomalacia caused by Thiamine Deficiency

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Signalment: Two Chihuahua dogs from the same litter (9-year old) with head tilt, head turn, nystagmus, ataxia, rolling, and status epileticus were presented at Haemaru Animal Referral Hospital.

Results: In magnetic resonance images, the left cerebellar and the medullary regions showed hyperintensity in T2 and T1-wighted post contrast images in one dogs. In another dog, the left cerebral hemisphere, especially in the occipital lobe, showed hyperintensity in T2 weighted images, but no contrast enhancement. Differential diagnosis include meningioma, meningoencephalitis and cerebral infarction was listed. One dog which had shown more severe persistent seizure and chewing activity without drug respose than another dog was euthanatized at the owner's request. In necropsy and pathological findings, severe unilateral polioencephalomalacia with left cerebral hemisphere, cavitary change of cerebral cortex caused by severe malacia, neuronal and myeline degeneration, and multifocal infiltrate of inflammatory cells including glial cells, lymphocytes, and plasma cells over cerebral parenchyma and meninges and perivascular regions was diagnosed. In cerebellar region, Purkinje cell degeneration of focal folia and thinning of granular layer were observed. Polioencephalomalasia mainly elicited by thiamine deficiency, lead poisoning and ischemia. However, tubular necrosis which is commonly occur in the case of lead poisoning and thrombo embolic complications is not observed in our dog. Therefore, thiamine deficiency was strongly suspected, and we trial treatment was initiated with thiamine 5 g/kg twice a day, per orally. Neurological signs were decreased dramatically and disappeared eventually. Our dog has not been evoked any neurologic signs in follow-up examinations for 2 years.

Clinical relevance: Thiamine deficiency induced neurologic diseases is rare in dogs, but may elicit bilateral symmetric cerebral lesions. However, in this case unilateral polioencephalomalacia was observed and clinical signs were disappeared with thiamine supplement only. Though limitation of our study is that we did not analyze the dietary thiamine, thiamine deficiency may be included in the differential diagnosis even in case showing generalized seizure with unilateral cerebral lesion.

Key words: polioencephalomalacia, thiamine, MRI, seizure, histopatholic

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