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NOTE

Pathology

Spontaneous peripheral neuritis in two electric eels (*Electrophorus electricus*)

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ABSTRACT. This study represents cases with spontaneous neuritis of peripheral nerves in electric eels. Two electric eels were presented with abnormal swimming behavior and loss of appetite. Electric eels had extensive histopathologic lesions in the splenic and cardiac nerves. The lesions were characterized by swelling of neuronal cells, central chromatolysis and marked inflammatory cell infiltration consisting mainly of lymphocytes around the affected nerves. To the best of our knowledge, this is the first case report of spontaneous neuritis of peripheral nerves in electric eels.

KEY WORDS: electric eel, neuritis, peripheral nerve

Inflammation of the peripheral nervous system is rare in animals. Several infectious diseases cause inflammation in both peripheral nervous system (PNS) and central nervous system (CNS) in fishes, such as edwardsiellosis, nodavirus infection and viral hemorrhagic septicemia [2, 8, 14]. A few cases of peripheral neuritis have been reported in dogs; acute polyradiculoneuritis is an acute disease that is characterized by sudden onset of paralysis, paresis or tetraplegia [5]. This is the common form of acute polyneuritis in dogs [5]. Histopathologically, axonal swelling and pale myelin with variable mononuclear inflammatory cell were observed in peripheral nerves [6, 10]. This histopathological lesions have been likened to the acute idiopathic polyneuritis-Guillain–Barré syndrome (GBS) in humans [4, 12].

Although neuritis of CNS including brain and spinal in fishes have been reported [8, 14], but neuritis of PNS in fishes have not recorded. The electric eel has an elongated, cylindrical body, typically growing to about 2 m in length, and 20 kg in weight, making them the largest species of the Gymnotiformes [1]. Neuritis of PNS in electric eels have not been well-established. The aim of the present study is to describes histopathologial lesions of spontaneous neuritis of PNS in electric eels.

Electric eels were caught and maintained in Kobe Municipal Suma Aqualife Park, Japan. Postmortem samples of two electric eels were sent to the Laboratory of Veterinary Pathology at Osaka Prefecture University for pathological examination. The history of the two electric eels included abnormal swimming behavior and loss of appetite; and other informations are listed in Table 1. They had been single-housed in a box of aquarium with acidic environment (pH 6.0–6.8) and water change was conducted once per week. They were treated by antiparasitic drugs, which did not improve the conditions. No relationships of two cases as to onset time and breeding space were noted. Finally they died and a necropsy was performed. No gross abnormalities were observed in the viseral organs, CNS and PNS. Tissues were collected from the liver, spleen, heart, kidney, intestine, brain and skin; then the samples were fixed in neutral-buffered formalin. After fixation, samples were routinely embedded in paraffin wax, sectioned at 4 μm in thickness and stained with hematoxylin and eosin (H&E) for microscopic evaluation.

Two electric eels were shown histopathologic lesions of PNS. Extensive histopathologic lesions were observed in the splenic (Fig. 1) and cardiac (Fig. 2) nerves. The lesions were characterized by swelling of neurons and central chromatolysis. In addition, there was marked inflammatory cell infiltration consisting mainly of lymphocytes around the affected nerves (Figs. 1 and 2). Based on the histopathological findings, we diagnosed these cases as neuritis of PNS in multiple organs. Moreover, one eel had

Table 1. Case information

Case No.	Age	Sex	Body length (cm)	Body weight (kg)	Origin	Clinical sign	Survival time
1	Unknown	Female	109	4.50	Purchase	Loss of appetite, abnormal swimming	11 days after onset of clinical sign
2	Over 3 years	Unknown	138	3.59	Unknown	Loss of appetite, abnormal swimming	9 days after onset of clinical sign

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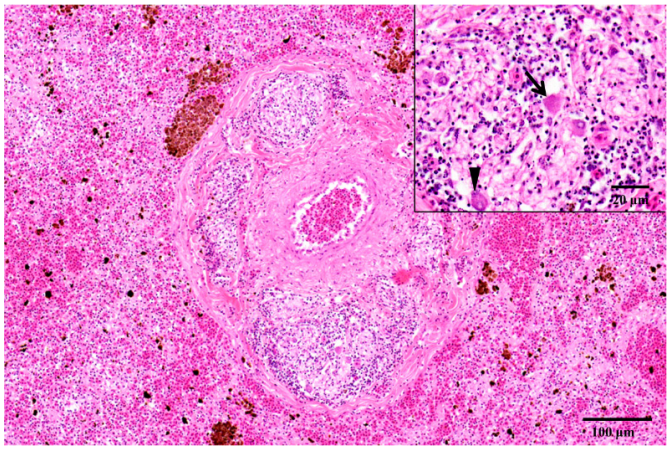

Fig. 1. Splenic nerve plexus; electric eel. Swelling of neurons (inset, arrow) and central chromatolysis (inset, arrowhead). Disseminated aggregations of inflammatory cells are found. H&E staining. Bar=100 μm, inset; Bar=20 μm.

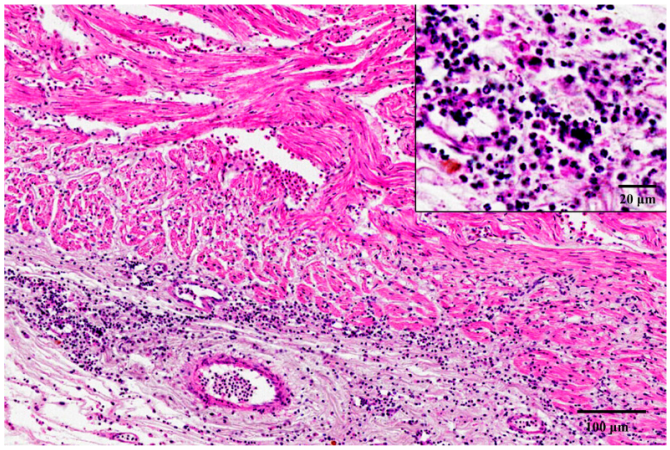

Fig. 2. Cardiac nerve; electric eel. Infiltration of lymphocytic inflammatory cell in the nerve fibers. H&E staining. Bar=100 μm, inset; Bar=20 μm.

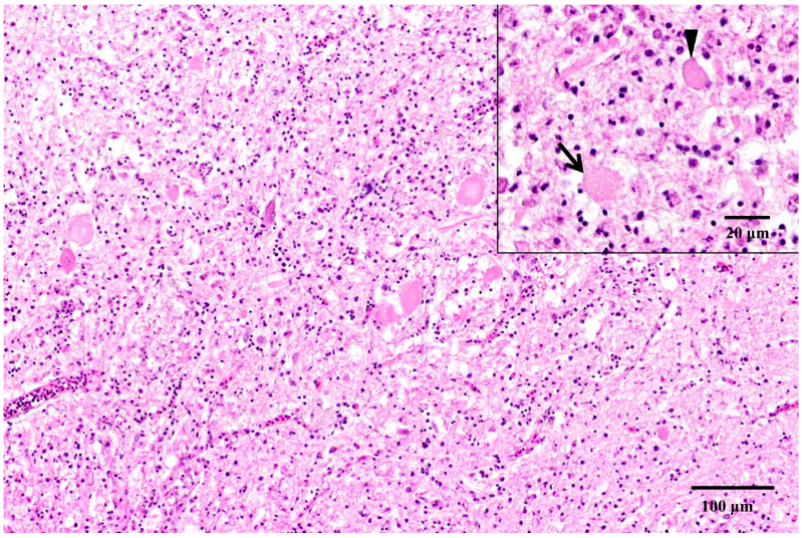

Fig. 3. Midbrain; electric eel. neuronal degenaretion (inset, arrow), marked infiltration of inflammatory cells with central chromatolysis (inset, arrowhead). H&E staining. Bar=100 μm, inset; Bar=20 μm.

histopathological lesions in the midbrain (Fig. 3), which characterized by neuronal degenaretion with central chromatolysis and marked infiltration of inflammatory cells.

Although an alternative diagnosis of noda virus infection and viral hemorrhagic septicemia should be considered which is charcterized by necrosis with vacualization of nerve cells in the CNS and necrotic nerve cells contain intracytoplasmic inclusion bodies [8]. In the current study, we did not find any cells with intracytoplasmic inclusion bodies in the CNS and PNS. Differential diagnosis also included edwardsiellosis caused by *Edwardsiella ictaluri* which is charcterized by infiltration of inflammatory cells in olfactory rosettes composed of macrophages, neutrophils, eosinophilic granular cells and fewer lymphocytes [14]. In the present study, histopathological lesion showed infiltration of inflammatory cells consisting lymphocytes, fewer plasma cells in the PNS.

Moreover, the classification of neuritis in humans and animals including fishes is incomplete; the pathogenesis and the relationship among neurotic syndromes are not well established [3]. Moreover, GBS has an immunologic basis in the pathogenesis, possibly secondary to postinfectious etiologies [11, 16]. According to human guidelines of GBS, histopathological patterns were characterized by perivenular leukocytic infiltration, degeneration of myelin sheaths, swelling and fragmentation nerve cells, and chromatolysis of ventral horn cells [6]. Cases of canine neuritis of peripheral nerves was reported with the most severe lesions in the region of the cauda equine and histologically presenting as mononuclear cell infiltration with swelling of neurons in the cauda equine [9, 15]. Trigeminal neuritis was also reported in dogs [13]. There are no information related to neuritis of peripheral nerves

in this specific species (electric eel).

In our case, the histologic lesions were restricted to the splenic and cardiac nerves and areas of necrosis were not found in any histopathological sections of the kidney, liver and others collecting tissues. We also did not find bacterial colonies and intracytoplasmic inclusion bodies within the affected areas of PNS. Moreover, GBS in human and peripheral neuritis in dog mainly affected in the somatic nervous system [7, 10], but peripheral nervous lesions in our present cases mainly involved in the autonomic nerves. A small focus of neurodegenarative and inflammatory lesion was observed in the midbrain of one eel; but relevance to peripheral nerve lesions is unknown. Peripheral nerve lesions were more prominent and extensive in both two eels. Thus we diagnosed these cases as peripheral neuritis. The cause of neuritis of PNS in electric eel remains uncertain and the present cases may be idiopathic.

Morphological evidence from light microscopical investigations performed in this study supports contention of neuritis of peripheral nerves in electric eels. To the author's knowledge, this case is the first case of neuritis in peripheral nerves with unknown cause in electric eels. This report also highlighted the need to consider for studying fish nervous system as counterparts of animal and human nervous system.

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